Shoulder Stiffness

Current Concepts and Concerns

Eiji Itoi · Guillermo Arce · Gregory I. Bain
Ronald L. Diercks · Dan Guttmann
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Editors

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Preface

The definition, etiology, and management of the stiff or “frozen shoulder” have been an enigma. This disabling condition occurs predominantly in the middle aged and spontaneously resolves. This is the reason why little attention has been paid to this very common but not well-understood condition. Knowledge of common shoulder conditions (e.g., rotator cuff tear and shoulder instability) has increased tremendously over the last decades; however, frozen shoulder still remains a challenge.

The members of the Upper Extremity Committee of ISAKOS met in Amsterdam in May 2014 with the aim to create a consensus statement on the definition, classification, pathophysiology, and treatment of the stiff shoulder. Of our 23 members, 19 members contributed. Each member provided presentations on assigned topics, which were followed by detailed discussions.

This book, *Shoulder Stiffness: Current Concepts and Concerns 2014*, is our consensus statement based on this meeting in Amsterdam. The consensus of the meeting was to make the following recommendations:

1. Definitions
   - “Stiff shoulder” – to describe the patient who presents with a restricted range of motion regardless of etiology, either primary or secondary causes
   - “Frozen shoulder” – to be used exclusively for idiopathic stiff shoulder
   - “Secondary stiff shoulder” – for a stiff shoulder with a known etiology
   - “Adhesive capsulitis” – not to be used because there is no adhesion
2. Classification
   - Intra-articular (cartilage and synovium)
   - Capsular
   - Extra-articular (rotator cuff muscles and tendons)
   - Neurological
   - Other remote causes (heterotopic ossification, burns contracture)
3. Treatment

There are many treatment options for the stiff shoulder, such as steroid injection/oral medication, rehabilitation, home exercise, joint distension, manipulation under anesthesia, and arthroscopic capsular release. However, we are unable to recommend any single treatment over others due to lack of evidence.

Based on these observations, we came to a conclusion that high-quality evidence needs to be established in the field of the pathogenesis and treatment of the frozen shoulder.

We thank the members of the committee for having provided their time and effort to attend the meeting and prepare the chapters for this book. We thank Professor Ronald Diercks for being the local host and the ISAKOS office staff for coordinating the logistics of the meeting.

All of the members of the committee have learnt considerably from each other during the presentations and discussions. We trust that this consensus document will assist the practicing clinician in the assessment and management of the stiff shoulder in the future.

Sendai, Japan Eiji Itoi, MD, PhD
Adelaide, SA, Australia Gregory I. Bain, PhD, MBBS, FA(Orth)A, FRACS

Upper Extremity Committee, ISAKOS 2013–2015

Members at the closed consensus meeting in Amsterdam, May 12–13, 2014

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Part I

Classification and Epidemiology

The Pathogenesis and Classification of Shoulder Stiffness

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Gregory I. Bain and Harry D.S. Clitherow

1.1 Introduction

The members of the Upper Extremity Committee of ISAKOS meet in Amsterdam in May 2014 with the aim to create a consensus statement on the definition, classification and treatment of the stiff shoulder. The academic chairman of the meeting was Dr Eiji Itoi, and deputy chairman Dr Gregory Bain. At this 2-day meeting, all members provided presentations on the selected topics. Following each presentation there was a lengthy discussion regarding the various concepts presented. At the completion of the meeting, the committee created consensus statements of the definitions, classification and treatment of the stiff shoulder.

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1.1.1 Purposes

The purposes of this chapter regarding the stiff shoulder are to:

1. Provide a review of the published definitions and present the committee's conscious definitions.
2. Describe the functional anatomy of the normal shoulder.
3. Describe the pathogenesis of shoulder stiffness.
4. Present a consensus classification of shoulder stiffness.

1.2 Definition of Shoulder Stiffness

In order to classify a condition, one must first define it. In its simplest form, stiffness means loss of normal movement. The word "contracture" means a shortening. "Frozen shoulder" was coined by Codman to describe "many conditions which cause spasm of the short rotators or adhesions about the joint or bursae" [1]. Other authors have attempted to subdivide this broad description into primary (idiopathic), secondary (known disorders) and tertiary (post surgery or post trauma) categories [2].

This approach has been criticised, as the pathological processes in idiopathic frozen shoulder, namely, capsular inflammation and fibrosis, are not present in other conditions. This implies that the term "frozen shoulder" is inappropriate for the other categories [1].

The term "adhesive capsulitis" has been used in place of idiopathic frozen shoulder in an attempt to highlight the primary capsular pathology present in this group. Other authors have suggested that the term "frozen shoulder" should be abandoned altogether and replaced with the term "contracture of the shoulder" in order to remove all confusion [3].

However, the term frozen shoulder persists, and the majority of published definitions of shoulder contractures have freely used the terms frozen shoulder and adhesive capsulitis. Zuckerman and Rokito [4] proposed a definition for frozen shoulder incorporating the term "functional restriction of both active and passive shoulder motion". The definition stipulated that a normal joint space must be present on plain radiographs and it included a separate subgroup for idiopathic/adhesive capsulitis. A survey of 190 members of the American Shoulder and Elbow Society found that 82 % of the respondents either agreed or strongly agreed with this definition [4].

The authors propose that the phrase "functional restriction of both active and passive shoulder motion" is better used as a definition for shoulder contracture due to any cause. A classification system can then be used to separate the different aetiologies and, more importantly, the different pathological processes present.

1.3 The ISAKOS Upper Extremity Committee Consensus Definition of Shoulder Stiffness

1.3.1 Stiff Shoulder

The term "stiff shoulder" should be used to describe the patient who presents with a restricted range of motion. The aetiology can be due to primary or secondary causes.

1.3.2 Frozen Shoulder

The term "frozen shoulder" should be used exclusively to describe the primary idiopathic stiff shoulder.

1.3.3 Secondary Stiff Shoulder

The term secondary stiff shoulder should be used to describe all other cases of shoulder stiffness with a known aetiology.

1.3.4 Adhesive Capsulitis

We do not recommend that this term be used as it does not reflect the pathological processes present.

1.4 Normal and Abnormal Shoulder Function

Abnormal joint motion is the result of pathological changes to normal anatomical structures. The effect of these pathological changes varies with time. Therefore anatomical, pathological and time factors must all be considered when developing a classification system (Fig. 1.1).

1.4.1 Anatomical Factors in Normal Joint Function

To produce active motion requires a series of normal physiological events to occur to the normal anatomical structures. This commences with the central nervous system, which initiates the intent to mobilise the joint, which then affects multiple anatomical structures to produce the active motion.

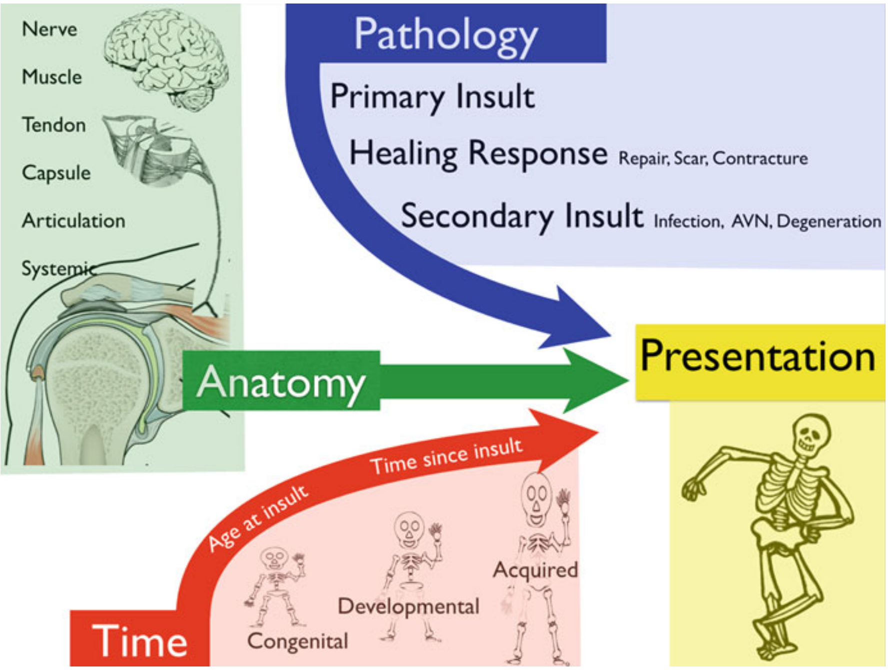

Fig. 1.1 There are 3 factors that make up the assessment and classification of the stiff shoulder: anatomy, pathology and time (Copyright Dr Gregory Bain)

1.4.2 The Shoulder "Machine"

The different anatomical components that must work together to produce functional shoulder movement can be simplified using the analogy of a crane (Fig. 1.2). An electric motor drives a lever arm that moves around a fulcrum. The anatomical components and their function can be divided up according to the different components: electric, motor, tension cable, articulation and lever. The shoulder articulation has several tendons that act across it, creating a series of individual motors that all act on the same fulcrum and lever arm. This creates a balanced system that can control motion and provide dynamic stability to the articulation (Fig. 1.3).

1.4.3 Electrical

The central nervous system (CNS) is the control room; it assesses the environment, identifies a need to perform an action and then initiates and coordinates the multiple muscles involved. Activity is modified according to sensory information relayed back from the joint and extra-articular sensory organs. The CNS activates the switch, which is conducted by the peripheral nerves to the muscle motor.

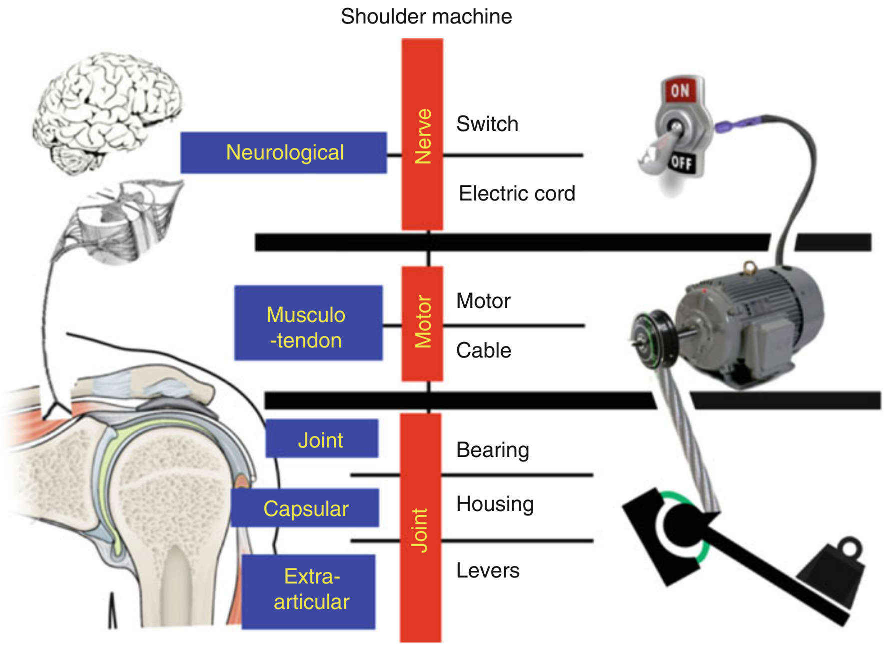

Fig. 1.2 The shoulder machine: with nerve (electrical switch, cord, coordination, sensory feedback), motor (motor and cable) and articular components. Articular components include the joint (bearings), capsular (housing) and osseous components (levers). Not shown are the other local extra-articular components such as the bursa, adventitia and skin (Copyright Dr Gregory Bain)

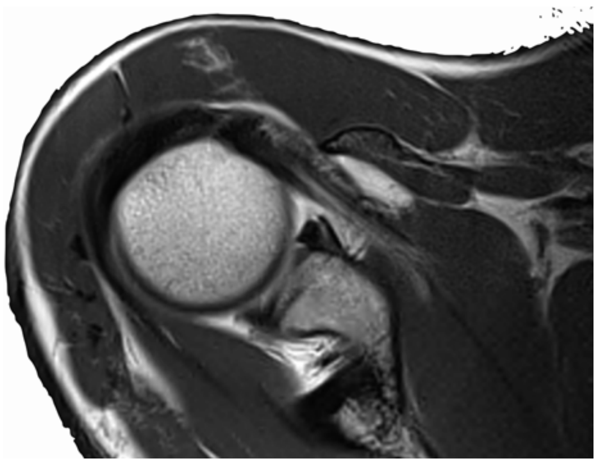

Fig. 1.3 The subscapularis (SSC) and infraspinatus (ISP) work in a coordinated fashion, in front and behind the articulation, to control the motion and provide dynamic stability for the joint. Note that the fascial attachments from the anterior and posterior are joined. This combined motion allows fine control, co-contraction for stability, eccentric contraction for control

1.4.4 Motor

The motor generates tension in the cable that attaches to the lever arm. Traction on the cable alters the position of the lever arm in space. The muscle is not like a typical motor, but is like a solenoid, which is an electric motor that can contract, but requires to be passively stretched, to regain the muscle length. Altering the tension in the tendon will alter the position of the bone.

1.4.5 Fulcrum

The glenohumeral articular surface is the fulcrum about which the lever (humerus) moves. It has several components that combine to allow efficient conversion of tensile force of the tendon into movement of the humeral lever. The articular cartilage is the bearing surface, with the synovial fluid the lubricant. The fulcrum is constrained by the articular congruency, the labral bumper and the ligaments. The load placed across the articular surface is supported by the subchondral and metaphyseal bone. The rotator cuff tendons reinforce the capsule and provide a dynamic restrain to the articulation, particularly with functional activities.

1.4.6 Lever Arm

The humerus is the lever arm in this system. The cable inserts into the humeral metaphysis and the force is transmitted to the diaphysis. Ultimately this positions the forearm and hand in space.

1.4.7 Other Structures

The surrounding extra-articular structures need to be compliant and not impede upon the motion of the entire lever mechanism. This includes the muscles, adventitia, bursa and skin.

1.5 Pathological Factors in Abnormal Joint Motion

1.5.1 Active vs. Passive Movement

Normal movement of the joint requires all components of the motor and lever system to be functional. If there is an isolated motor deficiency, then there will be weakness and possibly a lag, but there will be a full range of passive motion.

If there is an isolated fulcrum deficiency, then there will be a restriction of passive and active motion, but there will be normal strength.

1.5.2 Pathological Effect

The primary pathological effect will be different depending upon the anatomical site of the pathology.

CNS Poor control, initiation or coordination of motion, presenting as dyskinesia, tremor and spasticity.

Peripheral Nerves Signal does not reach the motor, leading to the lower motor neuron signs of flaccid paralysis, wasting and weakness. Failure to relay sensory input back to the CNS will impair the normal modification of activity in response to microtrauma or pain. This can result in a neuropathic arthropathy.

Muscle Reduced power produced presenting as weakness.

Tendon Muscular force does not reach the lever arm, presenting as weakness and loss of dynamic control of the articulation.

Articular Surface and Synovial Fluid The articulation loses its smooth motion, which may present as crepitus, synovitis, effusion and pain. The guarding response to this pain (conscious or unconscious) impairs the shoulder motor.

Capsule Contracture of the capsule will reduce the passive joint motion. If the capsulolabral complex is insufficient, the joint will be unstable.

Pathology affecting the subchondral bone plate and/or the other load-bearing areas of the scapula and humerus can compromise the ability of the bone to bear the joint reactive force. The fulcrum cannot support the lever arms.

The presence of marginal osteophytes may block the motion of a joint, similar to a wedge placed against the fulcrum, blocking the lever arm from moving.

Extra-articular Tissue Loss of tissue compliance increases the effort required to mobilise the joint and may impede motion. Examples include contractures of the skin (e.g. burns, trauma and surgery), contractures and heterotopic ossification of the intermuscular planes and thickened bursa.

1.5.3 Individual Pathology

The different elements of the normal shoulder anatomy form a balanced system.

When one element of the system becomes dysfunctional, this will affect the function of other elements. With time, secondary and tertiary pathologies may develop in these other elements. Failure to identify and address these additional pathologies will negatively affect the functional outcome of any treatment.

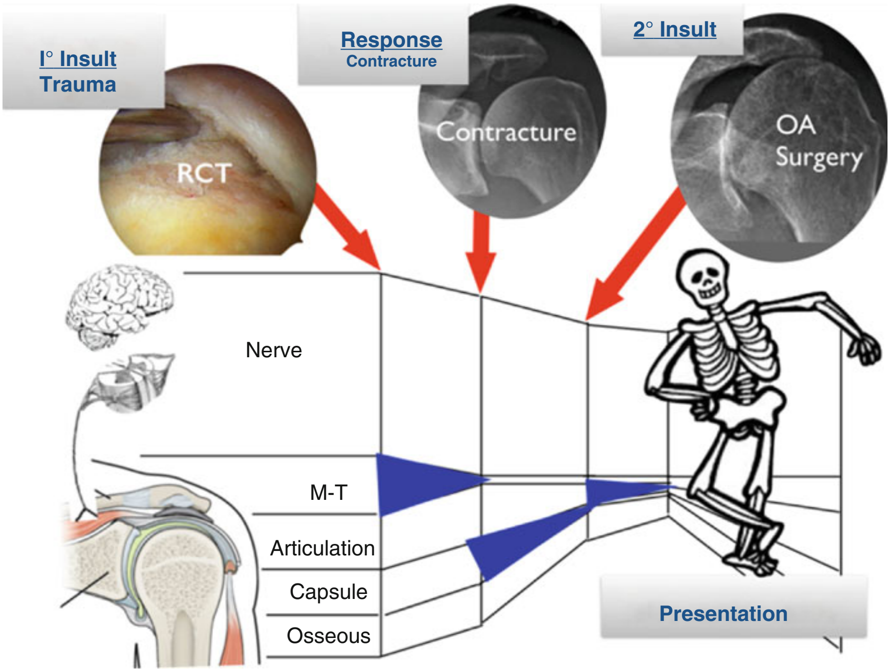

Fig. 1.4 Pathoanatomy of the stiff shoulder. The *initial insult* was a fall, which produced a tear of the rotator cuff. The *response* is a failed attempt at healing and development of a capsular contracture with superior migration of the humeral head. The *secondary insult* is degeneration of the articular surface, which is the final common pathway. Note how anatomy, pathology and time are linked. The presentation is due to three different pathologies, of three anatomical structures at three different times (Copyright Dr Gregory Bain)

The unique set of pathological processes present in each patient with shoulder contracture is a function of their premorbid state, the initial insult to the shoulder and their response to that insult over time (Fig. 1.4).

1.5.4 Premorbid Status

There are many aspects of the individual that may be important in the development and presentation of the patient:

Congenital or developmental abnormality (e.g. Sprengel shoulder, retroverted glenoid, Ehlers–Danlos syndrome)
Normal individual anatomical variability (e.g. glenoid version, ligamentous laxity)
Epidemiological factors, e.g. age, gender, ethnicity, genetic predisposition [5]
Mental state, e.g. psychological and psychiatric conditions, motivation to rehabilitate, secondary gain issues
Lifestyle factors, e.g. smoking, exercise and physical fitness
Systemic conditions, e.g. diabetes, Dupuytren's contracture, hypothyroidism and generalised arthritis

The Surgical sieve
Congenital
Degenerative
Infective
Inflammatory
Metabolic
Neurological
Neoplastic
Idiopathic
Immunological
Iatrogenic
Traumatic
Cardiovascular

Fig. 1.5 The "surgical sieve" of possible pathology causes of a clinical condition

1.5.5 Primary Insult

The insult is the event that causes the motor and wheel system to become deranged. This may be an acute event, such as a traumatic fracture or dislocation, or a more chronic process such as inflammatory disease or degenerative changes seen in idiopathic osteoarthritis. Usually there is a single cause of the initial event. The insult may also be due to a systemic condition, such as synovial inflammation due to autoimmune disease or joint haemorrhage due to bleeding disorders. There are many possible pathological disorders that can affect the shoulder motor. The surgical sieve is a commonly used method to ensure that uncommon causes are not overlooked (Fig. 1.5).

1.5.6 Response

The *physiological* response of the body to an insult is to attempt to repair and remodel the damaged structures and restore the motor and wheel system to the premorbid state.

A *pathological response* occurs if there is tissue ischemia and necrosis. It is common for the tissue to heal with a scar, and which may become a contracture.

Each individual will have a different response, which will depend upon their premorbid factors, the type of pathological insult and its severity.

1.5.7 Secondary Insult

Either during the healing phase or at some time later, a secondary insult may occur. This may include avascular necrosis, infection, nonunion or degenerative arthritis. Our treatment, including surgery, can also be a secondary insult. The final common pathway is usually to develop degenerative arthritis.

1.5.8 Examples

This model of pathoanatomical change can be illustrated with some common scenarios.

1.5.9 Joint Instability

A number of premorbid factors may predispose a patient to instability. These may be genetic (e.g. lax connective tissue) or environmental (e.g. participation in contact sports). The age of the patient is also important – the chance of recurrence is high if the patient is in their teens when the initial insult occurs. A joint that is dislocated or subluxated will have a restricted motion due to the loss of congruity between the two articular surfaces. Following an acute, traumatic dislocation, reduction of the joint will usually allow healing of the capsular tissues in their normal position. If the joint is immobilised, some adaptive shortening of the capsular structures will occur. However, if the capsule–labrum injury successfully heals, no further components of the system will be compromised. Mobilisation of the shoulder will address the capsular shortening, and normal or near-normal joint motion can be restored.

However, in a chronically unstable joint, the failure to restore the normal anatomic alignment of the joint surfaces causes the capsule to be held in a chronically lax position. Adaptive shortening of the capsular tissue occurs, creating a secondary contracture. The lack of normal joint motion and synovial fluid distribution affects cartilage nutrition. Persistent instability episodes directly injure the articular surfaces and subchondral bone. The cartilage surfaces thus develop secondary degenerative changes. The result is a joint that is both stiff and unstable. This is the worst situation. An open release (or salvage) procedure is required to reduce the joint, yet even if the joint can be restabilised, if the secondary changes have developed, this will prevent the joint from returning to its normal state.

1.5.10 Congenital Deformity

A congenital deformity of the musculoskeletal system can affect the shape, alignment or restraint of a joint. Conditions acquired during childhood, such as brachial plexus injury, polio or trauma, will also produce a significant effect on the primary anatomy.

Compared to an adult, the immature skeleton has an enhanced ability to remodel due to the presence of growth plates. The anatomical components develop in response to the stresses exerted on them. If the joint does not mobilise correctly, then over time the motor and wheel components will all develop in an abnormal manner.

In this instance time can be a positive factor. The longer the interval between the insult and the onset of skeletal maturity, the more chance there is for the skeleton to remodel. If normal forces can be placed across the joint, the normal anatomy can potentially be restored.

1.5.11 Spasticity

Spasticity is a velocity-dependent increase in muscle tone, resulting from an upper motor neuron lesion. The primary pathology causes an alteration in the signals sent to the muscle, resulting in a permanently contracting muscle. This can cause the joint to be held at the extremes of its normal motion, which can cause articular point loading. This may result in local degeneration, pain and joint instability.

With prolonged spasticity the sarcomeres and the muscle belly connective tissues undergo shortening. Prolonged immobilisation causes extrinsic adhesions to develop between the muscle–tendon unit and the surrounding soft tissues. Portions of the joint capsule are under increased tension due to the humeral head position, creating microtrauma and, frequently, pain. Other portions of the capsule are in a chronically lax position and undergo adaptive shortening, creating contracture and inhibiting restoration of normal joint alignment.

1.6 Clinical Assessment of Shoulder Stiffness

The clinical assessment needs to be aimed to understand the primary cause of the stiffness. The stiffness will be due to a pathological effect on an anatomical structure, which will vary with time. Therefore anatomical, pathological and time factors all need to be assessed. As there are many possible causes of shoulder stiffness, a detailed history and examination need to be performed.

1.6.1 History

This should include information regarding the initial onset and development of the stiffness and pain. This may be the best way to gain an insight into the primary pathology that underpins the entire presentation.

The history identifying any significant past medical history (e.g. diabetes and Dupuytren's contracture), previous injuries, surgery, weakness and paraesthesia must be sought.

1.6.2 Pain

Details of the pain history may identify a sudden onset of disabling pain (e.g. calcific tendonitis, brachial neuritis, gout, pathological fracture, nerve root compression).

Pain due to frozen shoulder can be very disabling and is usually slow in onset, but it may be more acute, often after a minor injury. The patient will often describe that after a simple manoeuvre, such as removing a jacket from the back seat of a car, they will have an aching and burning pain for 2 or 3 days. Night pain is well recognised to occur in subacromial impingement, frozen shoulder, degenerative arthritis and avascular necrosis. It may peak and then recede despite persisting shoulder stiffness (primary idiopathic frozen shoulder).

The patient with secondary gain may describe their pain in a dramatic way and report extreme disability after what would usually be a minor injury. The absence of pain is also important. In a grossly arthritic shoulder with minimal symptoms, the diagnosis of a neuropathic (Charcot) arthropathy must be considered.

1.6.3 Examination

Observation of the patient will reveal scars from prior injury or surgery. The resting posture of the shoulder and the pattern of any wasting that is present are also important examination findings. Passive and active range of motion must be assessed, as this will differentiate the patient with a primary motor problem from a patient with a primary articular problem.

Observing active motion allows assessment of the deltoid and peri-scapular muscle control. The presence of spasticity can be elicited during assessment of passive motion.

In idiopathic frozen shoulder the loss of passive motion tends to be global. In contracture secondary to other causes such as trauma or spasticity, the motion may be restricted in one plane yet relatively preserved in another. Documenting the unrestricted planes will help prevent inappropriate release of tissues at the time of any surgical intervention.

The strength of all rotator cuff muscles is assessed and the presence of any lag is noted [6].

1.6.4 Investigation

The requirement for any imaging is based on the history and examination. Plain radiographs are the mainstay of initial imaging, providing rapid assessment of osseous structures and articular alignment. Computed tomography (CT) will provide finer detail for assessing joint incongruity, joint degeneration and dysplasia.

Soft tissue pathology is generally best appreciated using magnetic resonance imaging (MRI). The addition of an arthrogram can enhance both CT and MRI studies of the shoulder.

Electromyography (EMG) will provide information on the integrity of the lower motor neurons (wires) and the muscle (motor).

Investigations may need to be made in areas remote to the shoulder if the stiff shoulder is secondary to pathology elsewhere such as a CNS lesion or a generalised medical condition (diabetes, Dupuytren's contracture, etc.).

1.7 Classification

The ideal classification system must achieve three features:

Reproducible – the system must accurately and reproducibly divide patients with the condition to be classified into separate groups.

Describe pathology – there must be a demonstrable difference in the pathoanatomy present between each group.

Dictate management – the pathoanatomy present in each group must translate to a difference in the prognosis and/or treatment for that group.

The consensus definition for frozen shoulder proposed by Zuckerman and Rokito [4] included a classification system based on both the aetiology and anatomic location of the pathology (intrinsic, extrinsic or systemic). Lundberg [7] classified frozen shoulder by aetiology. Primary frozen shoulder was reserved for cases of restricted range of motion with no cause identified on clinical history, examination or investigations. Reduced shoulder range of motion following traumatic lesions was defined as secondary frozen shoulder [7]. These systems are reproducible; however, the authors believe that they do not clearly discern the pathological processes that are present. Therefore it does not provide a clear guide for potential management of the condition.

Previous authors have divided joint stiffness into intrinsic (joint) and extrinsic (outside joint) [2, 4, 8]. However, the capsule is an important part of the entire scenario. It is different to the other intrinsic and extrinsic causes of contracture. Due to its unique position, the capsule is sensitive to the initial pathological insult and is the structure most at risk of developing a contracture as a response to the initial insult. The capsular pathology has a different aetiology, treatment and prognosis; thus it is considered as a separate category. The authors have used this same principle in the wrist and elbow [9, 10].

For any given cause of a stiff shoulder, the overall clinical picture will depend upon which anatomical structures are involved. We propose that a classification system should locate the pathological processes within three anatomical regions: intra-articular, capsular and extra-articular (Table 1.1). Initially only one area may be affected, but with time and the development of secondary and tertiary changes, a mixed pattern will occur.

By identifying the pathological processes present in each region, the surgeon can create a tailored management strategy that will be able to address each component of the patient's shoulder stiffness.

Table 1.1 Patho-anatomical classification of shoulder stiffness

Intra-articular (bearings)	Capsular (constraints)	Extra-articular (motor, cable, levers)	Neurological (control, electrics, sensors)
Articular surface Osteochondral defect Degeneration *Subchondral* Dysplasia, fracture AVN, degeneration *Synovium* Inflammatory Crystalopathy	*Labrum and ligaments* Deficient Tear *Capsule* Patulous capsule Capsulitis, contracture *Congruity* Subluxation Dislocation	*Muscles* Myopathy Fatty infiltration *Tendons and bursa* Tear, calcification degeneration, bursitis *Other (external to shoulder)* Fracture, malignancy, HO Skin contracture	*Central* Behavioural, dyskinesia, Dystonia *UMN and LMN* Spasticity Flaccid paralysis *Sensory and autonomic* Charcot joint Chronic regional pain

Note: The extra-articular causes are outside of the joint and include rotator cuff tendon and muscle. "Other causes" are completely separate from the shoulder. The systemic causes will affect a specific anatomical structure, e.g. diabetes causes capsulitis

1.8 Management Principles

The clinical effect of the disorder will depend upon the effect of the pathologies present in each anatomical structure. These may evolve with time. The prognosis depends upon the pathological process and how severely it has affected the anatomical structures (Fig. 1.6).

1.8.1 Intra-articular Pathology

The intra-articular structures can be managed with articular surgery. This includes arthroscopic synovectomy, removal of chrondral flaps and loose bodies. In late cases, debridement is unlikely to be successful, in which case a joint resurfacing arthroplasty may be required.

1.8.2 Capsular Pathology

The capsular contracture can be released or resected. This can be performed as an open or arthroscopic procedure. If this is an isolated pathology, then the prognosis is excellent. It is important to be sure to not perform an excessive capsular release, as this may compromise the stability of the joint. The primary ligamentous joint stabilisers should be maintained. If they are compromised, they require repair or reconstruction.

Fig. 1.6 Management of shoulder stiffness

Management of Shoulder Stiffness

Assessment

Anatomical Structures involved

Produces Disability

Pathological process 1°, response, 2°

Natural history / prognosis

Treatment

Address all affected anatomical structure
Respect all pathologies
Modify natural history / prognosis

1.8.3 Extra-articular Pathology

The extra-articular structures can be managed with open (or endoscopic) resection or release of the offending structures. This includes heterotopic bone resection and release or excision of soft tissue contractures. The prognosis depends upon the pathological process. Release or resection of the extra-articular tissues often regains most of the passive range of motion. However, if surgery is directed at the muscles, it may affect the strength of active motion.

1.8.4 Neurological

The neurological causes need treatment directed to the primary neurological disorder. In addition the secondary effects may also need treatment.

1.8.5 Systemic Disorders

There are systemic causes, but their pathology affects the individual anatomical structures. For example, rheumatoid arthritis affects the intra-articular components,

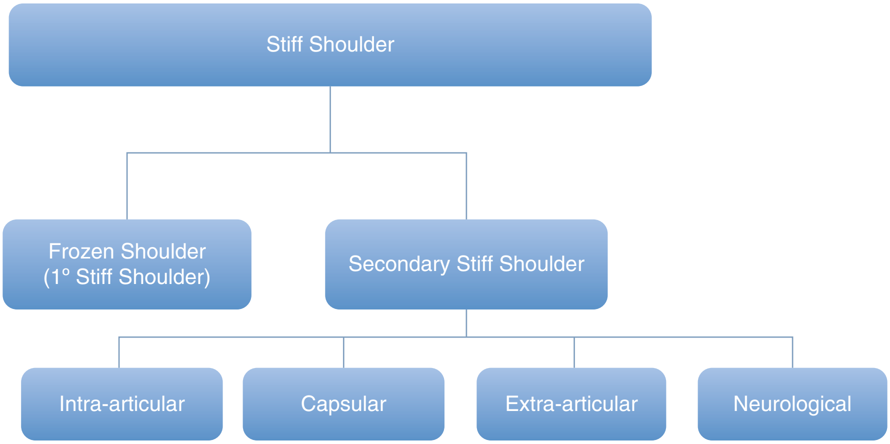

Fig. 1.7 Classification of shoulder stiffness

ISAKOS Upper Extremity Committee Definitions

Frozen Shoulder – The term "Frozen Shoulder" to be used exclusively to describe the primary idiopathic stiff shoulder. It develops without any trauma or specific shoulder disease period.

Stiff Shoulder – The term "Stiff Shoulder" to be used to describe the patient who presents with a restricted range of motion. The aetiology can be due to primary or secondary causes.

Secondary Stiff Shoulder – The term secondary stiff shoulder should be used to describe shoulder stiffness with a known aetiology, such as diabetes.

Adhesive capsulitis – This term is not recommended, as it does not reflect the pathological processes present.

Fig. 1.8 Definition of ISAKOS Upper Extremity Committee consensus meeting 2014

Dupuytren's contracture and diabetes affect the capsule and muscular dystrophy affects the muscles. The systemic factors should be considered and treated.

As can be seen the stiff shoulder has so many causes; it is not simply intrinsic or extrinsic. There are intra-articular, capsular, extra-articular and neurological causes of stiffness of the shoulder machine (Fig. 1.7). The definitions developed by the ISAKOS Upper Extremity committee, hopefully will assist clinicians in the future (Fig. 1.8).

There are local pathological causes such as trauma and infection. There are systemic causes such as diabetes and thyroid and renal disorders. All of these pathological aspects are part of the surgical sieve, which affects the anatomical components of the shoulder machine. The timing of the insult, the response of the patient and the secondary insult all affect the presentation of the patient with the stiff shoulder.

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Epidemiology of Frozen Shoulder

2

Carina Cohen and Benno Ejnisman

2.1 Introduction

As the etiology of frozen shoulder is not clearly understood, it is important to determine its epidemiology, so that specific risk factors, along with the interaction between them, and its prevalence can be identified. Strategies for intervention and prevention that take into account the underlying disease mechanisms should also be developed. The literature indicates that the incidence of idiopathic stiff shoulder or frozen shoulder is from 2 to 5 % in the general population [17]. Not only is it not uncommon, the associated annual health care and non–health care costs are estimated to be between $7,000 and $8,000 per episode [6, 40].

2.2 Epidemiology

Some authors have reported that the prevalence of frozen shoulder in women may reach 59–70 % [34, 38]. Other data have shown that it affects 3.38 women and 2.36 men per 1,000 person-years [43], or 2.4 people per 1,000 person-years for both sexes [41], mainly during middle age [15]. Our group performed a study in the Brazilian population, which is ethnically diverse, to identify the epidemiology. The

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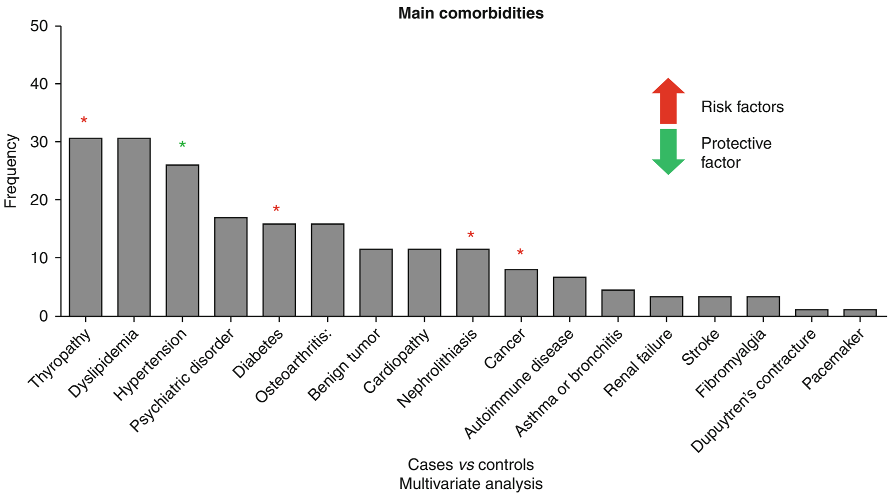

Fig. 2.1 The most common comorbidities found in patients with frozen shoulder. After a multivariate analysis, thyropathy, diabetes, nephrolithiasis, and cancer remained statistically significant, while hypertension was shown to be a protective factor

study included 88 frozen shoulder patients, of whom 52.3 % were female; the mean age at onset was 50.5 ± 8.4 years. There was a family history of the disease in 9.5 % of the patients, as determined by a heredogram.

Frozen shoulder often presents bilaterally, mainly in patients with diabetes, where its incidence varies between 10 and 41.7 % [4, 24], but it does not seem to affect the same shoulder twice [26]. In our study, we found bilateralism in 25.6 % of 88 patients, and 2 % of those represented a recurrence on one of the sides.

The most common comorbidities in our findings are shown in Fig. 2.1. They were present in 85 % of the patients with frozen shoulder, and 37.5 % had more than three comorbidities. After multivariate analysis, thyropathy, diabetes, nephrolithiasis, and cancer remained statistically significant, while, curiously, hypertension seemed to be a protective factor for frozen shoulder.

2.3 Thyropathy

In our data, we found individuals with thyropathy (hypothyroidism, nodules, Hashimoto's disease, Grave's disease, or thyroiditis) (Fig. 2.2) were 4 times more likely to develop frozen shoulder ($p < 0.001$) (Fig. 2.3); however, as the sample consisted of 88 frozen shoulder patients and 229 control patients, further studies should be performed to confirm this data.

Hypothyroidism is known to be risk factor for frozen shoulder. In one study of patients in an endocrinology clinic, frozen shoulder was present in 10.9 % of those with thyroid disorders, 8.8 % with Dupuytren's contracture, 9.5 % with carpal tunnel syndrome, 4.4 % with limited joint mobility, and 2.9 % with trigger

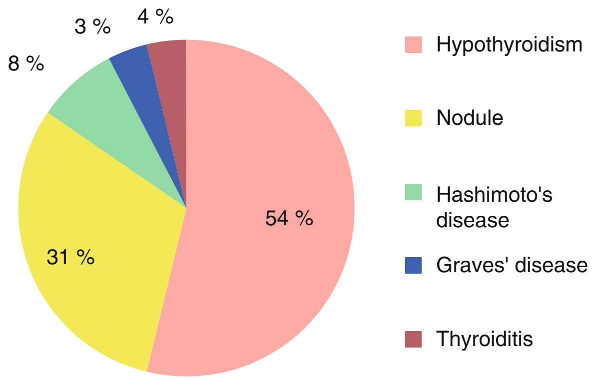

Fig. 2.2 Hypothyroidism represented 54 % of thyroid conditions associated with frozen shoulder

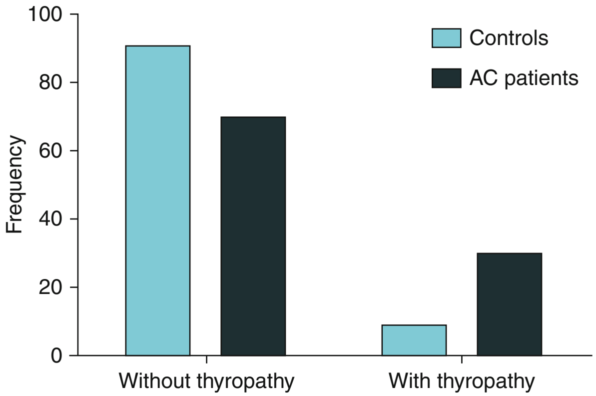

Fig. 2.3 Thyropathy shown as an independent risk factor [$p < 0.001$; ODDS = 4.37 (95 % CI: 2.01–9.52)] for developing frozen shoulder

finger [8]. Comparing the medical histories (diabetes, thyroid dysfunction, hypercholesterolemia, and hypertension), drug treatment, and blood tests of 126 frozen should patients with an age-matched control group of 98 patients from an orthopedic foot and ankle clinic and to the regional population disease prevalence registry in Jerusalem, Milgrom et al. [26] concluded that thyroid diseases may be not only be a risk factor for musculoskeletal disorders in general but a specific risk factor for frozen shoulder in women. Bilateral presentation of frozen shoulder was statistically significant ($p = 0.014$) in the Brazilian population, with the presence of thyroid nodules in comparison to unilateral involvement.

2.4 Diabetes

The diabetic population has a higher incidence of frozen shoulder, between 10 and 36 %, which is 2–4 times greater than in the general population [33]. Insulin-dependent patients are at higher risk [12], and the mean age of onset of disease is younger than that in the general population. There seems to be less pain in frozen shoulder patients with diabetes, but the time course is longer because these patients show less response to treatment [27].

In our study comparing 88 frozen shoulder patients with 229 age- and sex-matched controls, we found diabetes to be an independent significant risk factor ($p = 0.006$), showing almost 4 times the chance of developing the disease, which is in accordance with the study of Milgrom et al. that found 5 times the risk [5, 26].

Bridgman et al. studied 800 diabetic patients and 600 nondiabetic controls and found the incidence of frozen shoulder to be 10.8 and 2.3 %, respectively ($p < 0.05$). An association was also found with severity of diabetes: 36 % of frozen shoulder patients were insulin-dependent, whereas only 23 % of the 800 diabetic patients investigated were insulin-dependent (p < 0,001). Pal et al. [29] found the incidence of frozen shoulder to be 20.4 % of insulin-dependent patients, 18.3 % in non-insulin-dependent diabetic patients, and 5.3 % in the nondiabetic population.

In another study with 778 patients with diabetes [32], the incidence of frozen shoulder was 4.4 % in the diabetic patients, whereas the incidence in the control group was only 0.5 %. The low incidence in this study reflects the strict criteria used for frozen shoulder. The researchers found that 25.7 % of patients in the diabetes group complained of shoulder pain compared with only 5 % in the control group. A longer duration of diabetes was correlated with increased risk of frozen shoulder, although there was no relation with HbA1c levels. Insulin use was not found to be a risk factor.

2.5 Cancer

Cancer has also been pointed to as a risk factor for frozen shoulder. In our study, there was a history of tumor (malignant or benign) in 18.2 % of the frozen shoulder patients ($p = 0.017$), mainly represented by breast cancer and breast tumor, thyroid cancer, and thyroid nodules (Fig. 2.4).

As survival after breast cancer treatment increases, morbidity in the upper extremity is more frequently seen. Shoulder pain after breast cancer treatment varies between 9 and 68 %, and restricted motion is present in 1–67 % of survivors [16, 36]. Breast cancer treatment is followed by moderately to greatly decreased shoulder flexion and abduction [5, 22].

Primary chest wall tumors may mimic the symptoms of frozen shoulder and may lead to misdiagnosis of the tumor in young patients [9].

2.6 Renal Failure

In patients undergoing long-term hemodialysis, findings of frozen shoulder, as rotator interval obliteration, with magnetic resonance imaging (MRI) correlates with the range limitation of shoulder motion [19]. In our study, renal failure was statistically significant ($p = 0.002$) in patients with bilateral frozen shoulder in comparison to

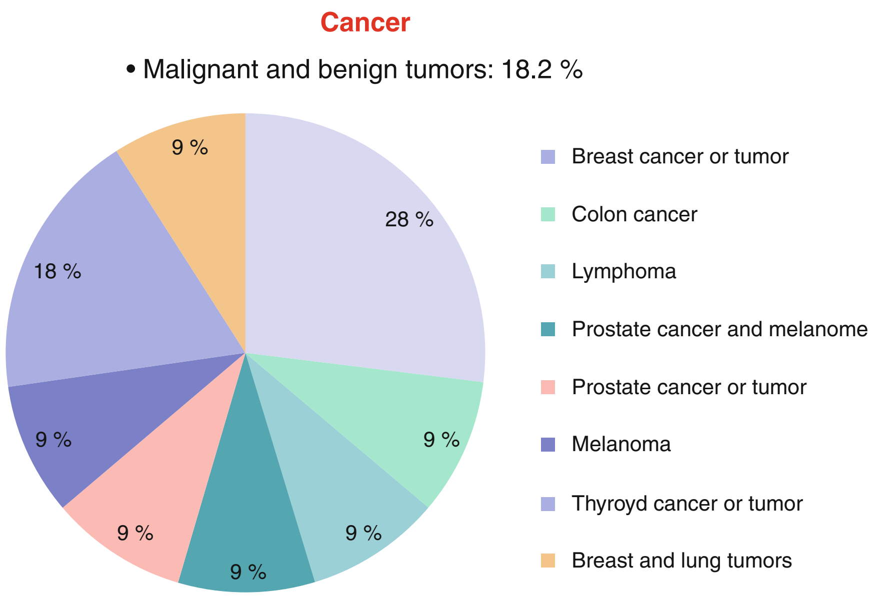

Fig. 2.4 Incidence of malignant and benign tumors in frozen shoulder patients in the Brazilian population, showing prevalence

unilateral presentation of the disease. This may correlate the severity of shoulder involvement with poor renal function, but whether there is an associated mechanism remains unknown.

2.7 Hypertension

A 2014 study showed that there is not a clear correlation between metabolic syndrome markers and frozen shoulder, but, for the first time, an association of hypertension with frozen shoulder resulting from a proinflammatory condition was described [2]. In contradiction, our data showed that hypertension was a protective factor for frozen shoulder ($p=0.02$; ODDS = 0.44), perhaps because of increased blood flow or interactions among risk factors. Further studies are needed.

2.8 Parkinson's Disease

Parkinson's disease may also correlate with frozen shoulder. It was initially thought that the higher incidence was the result of immobility in later phases of the disease. However, in a study of 150 patients with Parkinson's disease and 60 controls, Riley et al. [32] showed that shoulder complaints were present in 43 % of those with Parkinson's disease versus 23 % of the controls. Diagnoses of frozen shoulder were made in 12.7 % of patients with Parkinson's disease and 1.7 % of the control group. The frozen shoulder group had initial symptoms indicative of akinesia twice as frequently as tremor, whereas the ratio was reversed in the control group. Curiously, in 8 % of the frozen shoulder group, frozen shoulder was the first symptom noted, 0–2 years prior to onset of Parkinson, suggesting a further relation between the two diseases beyond immobility.

2.9 Dupuytren's Disease

Dupuytren's disease is believed to have a strong correlation with frozen shoulder. Bunker et al. [7] reported that up to 60 % of patients with idiopathic frozen shoulder have a history of Dupuytren's disease. In addition, histological findings from samples of shoulder capsule are comparable to samples of from patients with Dupuytren's disease, showing increased local collagen production, myofibroblasts, and fibroplasia [35].

2.10 Genetics

While strong evidence exists that Dupuytren's disease has an inheritable component and a racial predilection, the same has not yet been shown in frozen shoulder [25]. A study of 865 monozygotic and 963 dizygotic twins found the prevalence of frozen shoulder to be 11.6 % and the rate of heritability to be 42 % [14].

Future research is needed to confirm whether specific genetic components or environmental factors are responsible. We believe there may be a genetic predisposition to developing frozen shoulder.

2.11 Other Risk Factors

Other risk factors are thought to contribute to the development of frozen shoulder, including ischemic heart disease, use of a pacemaker, stroke, depression and other psychiatric diseases, immobility, neck surgery, cardiac surgery, neurosurgery, smoking, low body mass index, and some medications, such as highly active antiretroviral therapy (HAART) with protease inhibitors for HIV [10, 13, 21], anticonvulsant drugs, and others [1, 11, 28, 39, 42].

A summary of the literature on factors associated with frozen shoulder is provided in Table 2.1.

Table 2.1 Summary of literature on factors associated with frozen shoulder

Associated factor	Incidence of association	Incidence of frozen shoulder	Risk ratio
Diabetes mellitus	29.4 % (Milgrom et al. [26])	0.65 % (Yian et al. 2012) to 10.8 % (Bridgman [5])	3.08 (1.23–4.98) (Lo et al. [23]) 5.9 for males (4.1–8.4) (Milgrom et al. [26]) 5.0 for females (3.3–7.5)
Hyperlipidemia			2.67 (2.36–4.06) (Lo et al. [23]) 1.789 (1.366–2.343) for hyper-cholesterolemia 1.643 (1.190-–2.269) for measured hyper-low-density lipoproteinemia 1.645 (1.259–2.151) for hyper-non-high-density lipoprotein cholesterolemia (Sung et al. [37])
Thyroid disorders	13.5 % hypothyroidism	1.7 and 27 % in thyrotoxicosis (Baran [3]) 10.9 % in thyroid disorders group (Cakir et al. [8]) 13 % in hypothyroidism patients 10.8 % in subclinical thyrotoxic and thyrotoxic patients	7.3 (4.8–11.1) among females (Milgrom et al. [26]) 1.22 (1.03–1.45) (Huang et al. [18]) 5.33 (1.01–27.9) in hypothyroidism (Wang et al. [42]) 1.49 (1.25–1.77) in hyperthyroidism (Huang et al. [18])
BMI		Lower absolute body weight $p = 0.011$ Lower body mass index $p = 0.020$ (Wang et al. [42])	
Age			For each 5-year increment in age: Men: 1.14 (1.13–1.15) Women: 1.08 (1.07–1.08)
Sex		Women: 3.38 per 1,000 person-years Men: 2.36 per 1,000 person-years	1.27 (1.14–1.43) women > men (Yian 2012) 1.40 (1.38–1.43) women > men
Parkinson's disease		12.7 % (Riley et al. [32]) 19.8 % (Kim et al. [20]) 6 % (Rana et al. [30])	RR = 2.180 ($p = 0.010$) (Kim et al. [20])
Family history			6.03 (2.43–10.50) (Wang et al. [42])
Ethnicity			2.25 (1.17–4.32) (Wang et al. [42])

Conclusion

In conclusion, the epidemiology of frozen shoulder is still not completely understood, and treatment results vary by study and population. There is some evidence that genetic factors may be associated with development of frozen shoulder, but little is known and further studies are needed.

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Part II

Etiology

Frozen Shoulder: Etiology (Idiopathic)

3

Richard Angelo, Alberto de Castro Pochini, Carlos Vicente Andreoli, and Benno Ejnisman

3.1 Definition

Frozen shoulder is a condition with a functional restriction of both active and passive shoulder motions for which radiographs of the glenohumeral joint are normal. Classification used by Zuckerman [1]: (A) Primary: Primary frozen shoulder is considered a diagnosis for all cases for which an underlying etiology or associated condition cannot be identified (as specified for the secondary types). (B) Secondary: The secondary types of frozen shoulder include all cases of frozen shoulder in which an underlying etiology or associated condition can be identified. This is further subdivided into three categories. *Intrinsic*: This category includes limitation of active and passive range of motion that occurs in association with rotator cuff disorders (tendonitis and partial- or full-thickness tears), biceps tendonitis, or calcific tendonitis (in the case of calcific tendonitis, an acceptable radiographic finding would include calcific deposits within the subacromial space/rotator cuff tendons). *Extrinsic*: Cases in this category are those in which there is an association with an identifiable abnormality remote to the shoulder itself. Examples would include limitation of active and passive motion found in association with previous ipsilateral breast surgery, cervical radiculopathy chest wall tumor, previous cerebrovascular accident, or more local extrinsic problems, including previous humeral shaft fracture, scapulothoracic abnormalities, acromioclavicular arthritis, or clavicle fracture. *Systemic*: These cases occur in association with systemic disorders, including but not limited to diabetes mellitus, hyperthyroidism, hypothyroidism, hypoadrenalism, or any other condition that has been documented to have an association with development of frozen shoulder.

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Primary or idiopathic frozen shoulder occurs when no findings on history or examination explain the onset of disease [2]. The causes could be to immunologic, biochemical, or hormonal imbalances. Secondary frozen shoulder is found after stiffness and immobility, previous shoulder trauma, or surgery.

Frozen shoulder commonly occurs in patients with certain medical comorbidities [2–11] Most well known is the strong association between diabetes and frozen shoulder. Bridgman [5] first described this association after observing a 10.8 % incidence among 800 diabetic patients and only a 2.3 % incidence in 600 nondiabetic patients. The cardiac, endocrine, and neurologic disorders have a higher incidence than in the general population.

3.2 Predisposing Factors

3.2.1 Diabetes Mellitus

Epidemiologic data has established the relationship between diabetes mellitus (DM) and various rheumatologic manifestations, with shoulder and hand disorders being the most common. Frozen shoulder is the most recognized complication and is characterized by pain and moderate to severe limitation of active and passive range of motion. While the etiology is presumed to be related to micro- and macrovascular disease, definitive pathophysiological correlations have been difficult to establish. Earlier diagnosis of diabetes may provide opportunities to limit its musculoskeletal manifestations.

3.2.1.1 Incidence

When a diagnosis of frozen shoulder is made, it may be that it is the first indication of a diabetic condition. In one study of 88 patients with frozen shoulder and no prior history of known DM, blood samples revealed the prevalence of diabetes to be 38.6 % and prediabetes 32.95 % resulting in a total prevalence of a diabetic condition of 71.5 % [12].

Patients with established diabetes have a greater likelihood of developing frozen shoulder than the normal population. A cohort of 78,827 newly diagnosed patients with diabetes mellitus were compared with 236,481 age- and sex-matched subjects without diabetes. Over a 3-year period, there was a 1.333 increased likelihood of patients with diabetes developing frozen shoulder when compared to the control population [13]. In a different group of 297 type II patients consecutively presenting to an outpatient diabetic clinic, frozen shoulder was detected in 86 patients, 29 % [3–14]. In addition, statistically significant associations of frozen shoulder with the age of the patient ($p = 0.000$) and duration of diabetes (0.03) were noted. In a third review, a cross-sectional study of 291 type I (mean age 33.2) and type II (mean age 61.1) diabetic patients revealed a prevalence of frozen shoulder in 10.3 % of type I patients and 22.4 % of type II subjects [4–15]. Frozen shoulder was associated with age in type I ($p < 0.01$) and type II (< 0.05) and with the duration of diabetes in type I ($p < 0.01$).

3.2.1.2 Pathology

It has been challenging to determine why patients with diabetes are at higher risk for developing frozen shoulder. The relationship of frozen shoulder and glycemic control using hemoglobin A1c levels was studied in 1,150 diabetic patients with frozen shoulder and none was found [16]. Insulin-dependent diabetic patients, regardless of whether they did or did not also use oral hypoglycemics, were 1.93 times more likely than non-insulin-dependent patients to have frozen shoulder. Patients taking only oral hypoglycemic medications were 1.5 times more likely to develop frozen shoulder. The probability of developing frozen shoulder was 1.85 times greater for patients with more than 10 years of insulin use when compared with those with less than 5 years of use. These remain associations and don't support a causal relation for the lack of serum glucose control being as a cause for the development of frozen shoulder.

The relationship between the components of metabolic syndrome (insulin resistance, hypertension, dyslipidemia, and obesity) was evaluated [17]. In a cohort of 150 consecutive patients being treated for frozen shoulder, the use of anti-glycemic medication was noted in 18.4 % patients with frozen shoulder, over twice the national average of 7.6 %. The use of antihypertensive medications was higher than the national average as well, 31.3 % vs. 21.6 %. The presence of hypertension is presumed to be related to the vascular disease associated with diabetes, but the way in which this relates to insulin resistance is unclear. The use of anti-lipid medications and the presence of obesity were similar whether or not frozen shoulder was present.

Hyperemic synovitis and capsular thickening, particularly of the inferior region and rotator interval, have been noted to be a cardinal feature of frozen shoulder [18, 19]. Synovial inflammation is controlled, at least in part, by a variety of cytokines and growth factors. Intercellular adhesion molecule-1 (ICAM-1) plays a central role in the inflammatory response mediated by the immune system. In studying the relationship between frozen shoulder and diabetes, an upregulation of the ICAM-1 level in patients with diabetes has been shown [15, 20]. ICAM-1 has been found to be increased in the capsular tissue and fluid of the glenohumeral joint as well as the serum of the patients with frozen shoulder and diabetes mellitus compared with normal control patients [21]. However, it has been noted that many patients with diabetes mellitus who have a high ICAM-1 concentration in the serum show no symptoms of frozen shoulder. The effect of ICAM-1 on synovial cells may be reinforced by high levels of glucose, and this could be one explanation for the higher prevalence of frozen shoulder in the diabetic population.

A study of synovial biopsies that were obtained from 13 patients with frozen shoulder, four of whom had diabetes and 10 control patients with a diagnosis of subacromial impingement, was conducted [22]. Cytogenic analysis revealed that fibrogenic cytokine matrix metalloproteinase 3 (MMP 3) and inflammatory cytokine interleukin 6 (IL 6) were both elevated in patients with frozen shoulder. Diabetic patients, however, actually exhibited a decrease in the level of expression of inflammatory cytokine and monocyte colony-stimulating factor compared to nondiabetic patients with frozen shoulder. Although cytokines are involved in the process related to frozen shoulder, the regulation and specific role of cytokines in the development of inflammation and tissue healing remain unclear.

Little histological data are available for patients with frozen shoulder, and it is unknown how the tissues exhibit change during the various phases, i.e., "freezing, frozen, and thawing." In one report, 11 of 172 patients undergoing shoulder arthroscopy had a diagnosis of established frozen shoulder with five of them also having diabetes [23]. Neither intra-articular adhesions nor obliteration of the axillary recess were identified in any of these patients. A significant increase in fibroblast cells was noted next to loose areas of connective tissue on histological evaluation of capsular biopsies of the rotator interval region. Thickened vessel walls and augmentation of the synovial surface were noted, but no histological evidence typical of inflammation was identified, although the phase of the frozen shoulder was unspecified. On transmission electron microscope examination (TEM), the collagenous tissue exhibited a loss of fibril order and a twisting of collagen fibrils resulting in up to a fourfold increase in the diameter of the collagenous fibrils. Thin, elastic-like filaments could be seen between the thickened collagen bundles [23]. As thickened capsular tissue and loss of the axillary recess are not infrequently seen on MRI in the early phases of frozen shoulder, it may be that the patients in this cohort were more mature in the expression of their disease. It is possible that histological specimens from patients earlier in the disease process may exhibit more consistent signs of inflammation.

3.2.1.3 Steroid Injection in Diabetic Patients

A group of 45 diabetic patients were randomized to either receive an intra-articular cortisone injection consisting of 40 mg of triamcinolone acetonide or no injection [24]. Both were given a home stretching program. The corticosteroid injection group showed significant improvement in pain at 4 weeks and in forward elevation and internal rotation at 12 weeks, but there was no significant difference between the groups at 24 weeks. Thus, the addition of an intra-articular steroid injection may act primarily to accelerate the resolution of the inflammatory process and the recovery of shoulder motion.

The effect of an intra-articular steroid injection on serum glucose levels in patients with diabetes is unclear. In one study of 18 diabetic patients who had reasonable control of their sugars and also had self-monitoring devices for serum glucose, 15 also had frozen shoulder. The effect of an injection of 35 mg of methylprednisolone acetate (MPA) into the glenohumeral joint on blood glucose levels was evaluated [25]. Patients recorded blood glucose levels six times each day, every other day for 1 week prior, and 2 weeks following the corticosteroid injection. No significant changes in mean glucose levels after the injection were detected when compared to those obtained before the injection. Reports of steroid injections in the knee, however, have been documented to cause acute hyperglycemia for 2 or 3 days in patients with diabetes who otherwise have good glucose control [26]. The hyperglycemia, however, was not clinically significant. With the limited available data, there appears to be little risk for significantly affecting the patient's blood glucose levels following an intra-articular shoulder steroid injection.

3.2.2 Non-shoulder Surgery

Stiffness can occur after all sorts of shoulder surgery. Frozen shoulder also may occur after breast cancer surgery [31]. Surgical interventions with a higher risk of development of frozen shoulder are axillary node dissection and neck dissection, combined with radiation therapy and breast cacer surgery [30]. Cardiological procedures in association with frozen shoulder are cardiac catheterization in the axilla, coronary artery bypass grafting, thoracotomy [27], cardiac catheterization through the brachial artery [28], and placement of an ipsilateral subpectoral cardiac defibrillator [29]. The incidence of frozen shoulder developing in a male patient population undergoing cardiothoracic surgery has been estimated at 3.3 % [30].

3.2.3 Immobility

Shoulder immobilization is a risk of developing shoulder stifness [9, 32–34]. In a review of patients referred to Binder et al, 75 % were initially told to rest the shoulder rather than to perform gentle exercises [35].

3.2.4 Thyroid Disorders

Bilateral frozen shoulders can occur in patients with hyperthyroidism or hypothyroidism [4, 36–38]. Wohlgethan felt that hyperthyroidism, frozen shoulder, and shoulder-hand syndrome were linked disorders [39]. Resolution of shoulder stiffness has been shown to occur after thyroidectomy and stabilization of thyroid hormone levels [11, 39].

3.2.5 Cardiac Disease

The association between atherosclerotic coronary vascular disease and shoulder stiffness is well documented [40–43]. In a review of 133 consecutive cases of myocardial infarction, Ernstene found 17 patients originally, who complained of unrelenting pain in the shoulder region [42]. Shoulder-hand syndrome, an autonomic dystrophy, may be a sequel of myocardial infarction in 10–30 % of cases [10].

Bunker identified increased serum lipid levels in a frozen-shoulder group [44].

3.2.6 Pulmonary Disorders

In 1959, Johnson reported that the incidence of frozen shoulder was 3.2 % in a population of sanatorium patients with tuberculosis [9]. Saha reported that frozen shoulder occurred more frequently in patients with emphysema and chronic bronchitis [45].

3.2.7 Neoplastic Disorders

Bronchogenic carcinoma and Pancoast tumor of the lung can cause severe shoulder pain that can mimic the early phase of frozen shoulder [6, 31]. Other occult neoplastic tumors can be masked by symptoms attributed to a frozen shoulder. These include chest wall tumors and primary or metastatic carcinoma of the humerus [43, 46–48].

3.2.8 Neurologic Conditions

The incidence of frozen shoulder in patients with Parkinson's disease is significantly higher than in age-matched controls [49]. In 8 % of patients, the first symptom of Parkinson's disease was shoulder stiffness. This can occur up to 2 years before the onset of generalized symptoms [49]. Patients with cerebral hemorrhage and cerebral tumors are also at increased risk for frozen shoulder [36].

At least 30 % of patients with hemiplegia have shoulder pain and are susceptible to the development of shoulder stiffness [50, 51].

Brachial neuritis, a painful neuritic condition also known as Parsonage-Turner syndrome, has been associated with stiff shoulder [4–52].

3.2.9 Reaction to Medication

Grasland and coworkers described eight cases of frozen shoulder developing after treatment of human immunodeficiency virus (HIV) infection with protease inhibitors [53]. Specifically, all patients received the drug indinavir as part of their regimen and had no other risk factors for frozen shoulder [54–57]. Other medications in association with frozen shoulder include barbiturates 62, fluoroquinolones 63, nelfinavir 64, and isoniazid [3, 58].

3.2.10 Genetic Aspects

A report demonstrated a genetic association of 42 % for frozen shoulder, but no specific gene has been identified [59].

The genetic factor seems to be involved in many cases, but the basic science in this topic needs more time to prove that. In a recent paper 2 or 3 times higher incidence of stiff shoulder in female twins in every 1,000 patients [59].

3.2.11 Psychological Factors

In some cases of frozen shoulder, psychological factors can play a role. Psychological or physical trauma could be involved. In psychological cases, the diagnostic could be done after psychological evaluation [75].

Some articles did try to associate idiopathic frozen shoulder and personality traits or mechanical aspects, but no statistical association was established [75, 76].

3.3 Natural History

The frozen shoulder is a poorly defined entity. Despite extensive research, the etiology is still unknown. As a consequence, the management and evaluation of results in patients with stiff shoulders are confusing. The disease is characterized by a spontaneous onset of shoulder pain accompanied by progressive limitation of both active and passive glenohumeral joint movements. The natural history of idiopathic frozen shoulder syndrome is considered benign. The majority of authors state that a resolution of symptoms occurs from 6 weeks to 10 years. Codman [60] and Grey [61] concluded that the course of frozen shoulder is benign and self-limiting with complete recovery of pain and range of motion within a maximum of 2 years from the onset of symptoms. Shaffer et al. [62] on the other hand reported persistence of symptoms and impaired range of movement in over 50 % of their cases when followed up at 3 and 11 years. This long period of pain and disability has been the reason for many different types of intervention. Intensive physical therapy, mobilization and stretching, intra-articular injections, distension arthrography, manipulation under anesthesia, and open surgical or arthroscopic release have been performed with good results [6, 46, 55, 60, 63, 64]. Diercks [65] reported good results from gentle exercises within the painless range of motion without any form of passive stretching.

Frozen shoulder is a condition of an underlying synovial inflammation with subsequent reactive capsular fibrosis [55, 61, 64, 68, 70, 72, 74]. The management of frozen shoulder has been a field of controversy in orthopedics since the original use of the term by Codman in 1934. It is considered a benign condition lasting 18–30 months, but high percentages of patients are left with some shoulder impairment. A correct diagnosis is the first and most important step: other pathologies need to be ruled out. Diagnostic criteria are not so far from the ones published in 1969 by Lundberg [71]: painful stiff shoulder for at least 4 weeks that interfered with daily living or work activity, painful restriction of both active and passive elevations to less than 100 and 50 % restriction of the external rotation (at the side), and normal radiological appearance.

3.4 Etiology

The etiology of the frozen shoulder is still unknown. Brue [3–6, 9, 11–68] described the increased deposition of cytokines as transforming growth factor, platelet-derived growth factor, and tumor necrosis factor-alpha in the synovium and in the capsule of the frozen shoulder group compared to a control group. They postulated that cytokines might be involved in the fibrotic and inflammatory process. Especially, the Matrix-bound transforming growth factor-beta may act as a persistent stimulus, resulting in a capsular fibrosis. Bulgen in 1976 [69] found HLA B27 more common in patients with frozen shoulder, but this has not been confirmed in subsequent studies [44], and the same authors [70] in 2000 analyzed the expression of cytokines, growth factors, and metalloproteinase in frozen shoulders comparing the findings with normal shoulders and Dupuytren's contractures in patients. Tissue from frozen shoulders presented just a slightly higher presence of cytokines and growth factors compared to the control groups. The notable exception in comparison with both control groups was the absence of the metalloproteinase MMP-14 required to activate the proteolytic enzyme gelatinase A. The association between frozen shoulder and Dupuytren's contracture has been identified by multiple authors dating back to 1936 [19]. These reports identify rates of association ranging from 18 % to as high as 52 %. More recent investigators have likened the histologic changes seen in the glenohumeral joint capsule to those seen in Dupuytren's contracture of the hand [19, 68]. Bunker and Anthony performed manipulation and open excisional biopsy of the coracohumeral ligament and rotator interval capsule in patients who failed to improve with nonoperative treatment of frozen shoulder [19]. Tissue specimens revealed active fibroblast proliferation amidst thick nodular bands of collagen accompanied by some transformation to a smooth muscle phenotype. As in Dupuytren's disease, in frozen shoulder there is neither inflammation nor synovial involvement. Fibrosis is most evident in later phases of inflammatory response, and collagen and matrix synthesis takes place after chemotactic and cellular responses have occurred. These findings, therefore, might reflect a later phase of the disease, and an inflammatory phase might occur earlier [68] Uhthoff and Bolileau confirmed the presence of vimentin, a cytocontractile protein known to be present in Dupuytren's disease in histologic sections of only the anterior capsular structures in patients with frozen shoulder. This protein was not seen in specimens of the posterior capsule. The type I and III collagens were found, in both anterior and posterior capsular specimens. Maybe that contracture selectively involves only the anterior capsule, and the fibroplasia involves the entire capsule [46]. Hannafin and colleagues attempted to correlate the three histopathologic phases of fibroplasia detected in biopsy samples of patients with frozen shoulder, as related by Neviaser et al., with clinical examination and arthroscopy. They found that hypervascular synovitis provokes a progressive fibroblastic response in the adjacent capsule that results in diffuse capsular fibroplasia and contracture [64]. Based on immunohistochemical and histologic findings, these investigators proposed a cellular pathway, which eventually results in the clinical scenario of a frozen shoulder. Some authors suggest that there may be a common immunological or genetic basis for primary hypoparathyroidism and frozen shoulder. Alternatively, frozen shoulder may be a manifestation of hypoparathyroidism (artigo hipopara). Neviaser in [64] utilized open surgery to investigate the pathogenesis of idiopathic loss of shoulder range of movement. He found that the capsule was adhered to the humeral head and concluded that capsular adhesions caused the decrease in motion. On the contrary, Lundberg [71] documented periarticular inflammatory changes and thickening of the joint capsule without intra-articular adhesions. Rizk et al. [72] discovered thickening and constriction of the capsule. Ozaki [73] found a contracted and hypertrophied coracohumeral ligament. Neviaser [46, 64] described the arthroscopic stages of frozen shoulder supporting the hypothesis that the underlying pathological changes are synovial inflammation with subsequent reactive capsular fibrosis.

The etiology to some authors seems different between idiopathic and secondary frozen shoulder; however, some pathologic findings and stiffness occur in both cases [8].

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Post-traumatic Shoulder Stiffness

4

Knut Beitzel and Andreas B. Imhoff

4.1 Introduction

Physiologic shoulder motion is dependent on the correct function of the glenohumeral and scapulothoracic joints. The glenohumeral motion is based on rotation of the articulating surface of the humeral head on the glenoid and facilitated by the mutual sliding of a set of bursa-lined surfaces (deep sides of the deltoid, acromion, coracoid process, and its tendons against the proximal humerus, rotator cuff, and long biceps head). The scapulothoracic motion is dependent on proper gliding of the scapula on the thoracic wall as well as a coordinated and well-timed function of the scapulothoracic muscles. Both motion interfaces may be affected by post-traumatic changes, which result in limitations of the shoulder motion (stiff shoulder).

Post-traumatic shoulder stiffness (PTSS) is regarded as one type of the general syndrome of the "frozen shoulder" or "adhesive capsulitis." As for all types of "frozen shoulder," no general consensus exists on the etiology of PTSS. It usually is correlated to one specific trauma to the shoulder joint, which the patient is able to remember. This is seen in contrast to the "idiopathic" stiffness of the shoulder. In addition, its onset is often combined with a certain period of immobilization, because patients who experienced such a direct trauma to the shoulder commonly protect their shoulder joint due to the pain they feel when actively moving their shoulder.

If structural damage of the joint or the motion interface is caused by a trauma, this initiating factor can often be defined and treatment can be specifically initiated. Sometimes patients correlate the onset of clinical symptoms to some minor trauma, which might not be the "real" causative factor. Additionally, in a specific group of

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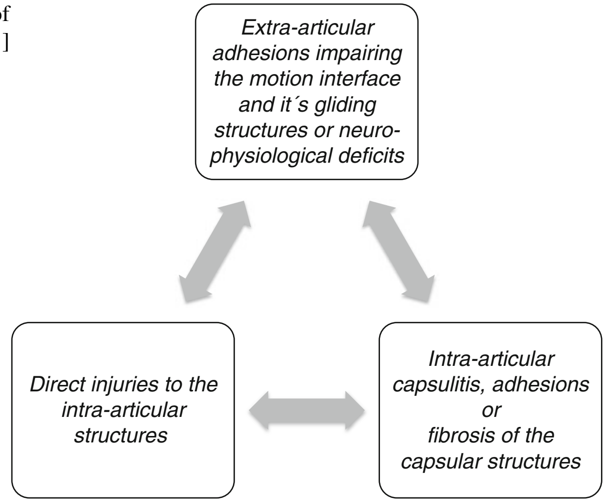

Fig. 4.1 Three major groups of causes for the onset of PTSS [1]

patients, no structural changes responsible for PTSS can be diagnosed by today's clinical or image-based methods.

The authors believe that all types of stiff shoulders show significant overlapping and that the process of PTSS is rather a very dynamic and complex process, which passes through different phases. It can be difficult to distinguish between the various groups, and there is clearly a considerable overlap between the different conditions and potential for progression from one to the other. In the authors' opinion and according to previous classifications, PTSS is defined as a stiffness of the shoulder joint, which can be linked to a causing trauma. The causes for the onset of PTSS can grossly be structured into three major groups [1] (Fig. 4.1):

- *Extra-articular adhesions impairing the motion interface and its gliding structures or neurophysiological deficits (e.g., injuries to the cervical spine, plexus, etc.)*
- *Direct injuries to the intra-articular structures (e.g., fractures, tendon ruptures)*
- *Intra-articular capsulitis, adhesions, or fibrosis of the capsular structures*

4.2 Extra-articular Adhesions Impairing the Motion Interface and Its Gliding Structures or Neurophysiological Deficits

Extra-articular adhesions or neurological deficits are seen as secondary, extrinsic causes [1]. According to the observations of Neviaser [2], patients may present with stiff shoulders that occur after trauma, who appear to not have any capsular contracture. These cases may be explained by the other extra-articular causes. Extra-articular adhesions might be caused due to soft tissue injury of the surrounding structures. For example, large burning injuries might result in excessive scarring of the skin, which then limits range of motion and therefore leads to a stiff shoulder joint. In these cases, the scarring of the skin layer directly causes the limitations. Hematomas located in the deltoid muscle or the subacromial space can cause direct adhesions and scarring within these structures and prevent proper function of the fascia, gliding of the soft tissue.

It is interesting to mention that if the glenohumeral joint is restricted for a longer period, deficits in motion might persist, even if the causing problem is solved (e.g., plastic surgery for restrictive burned skin). This is in contrast to the classic ideas of the "frozen shoulder," which, as a self-limiting condition, resolves after a certain time and patients regain motion.

Injuries to the neurophysiologic structures like the cervical spine or the brachial plexus may also cause compromised function of the humero-scapular or scapulo-thoracic muscles resulting in limited function and therefore reduced motion. This might also lead to a persisting shortening of the joint structures and a limitation of the range of motion.

4.3 Direct Injuries to the Intra-articular Structures

If mechanical barriers contributed to the loss of motion of the joint, reasons might be fractures of the bony structures, tendon ruptures, free joint bodies, injuries to the cervical spine with combined neurological symptoms, etc. which have also been defined as "secondary intrinsic" [1]. In these cases, the etiology of the limited range of motion is clearly identified by clinical examination and imaging. Most of these cases can be directly treated and allow the removal of the causing pathology. Some authors exclude these cases from their definition of PTSS, since the causing pathomorphology is known. However, we believe that, under a comprehensive view, these cases should also be included into the definition of PTSS.

If the causing pathology can be sufficiently treated, ROM can be recovered (e.g., fracture and displaced healing). However, it has to be noted that regaining ROM might be very difficult, if the immobilization of the joint persisted for a longer period and capsular fibrosis has already started to limit joint excursions and the intracapsular volume [3]. In these cases, the etiology might be rather combined with type three, and this makes the authors believe that all different types have wide overlapping as their etiology.

4.4 Intra-articular Adhesions/Intra-articular Fibrosis

Specific etiology of PTSS caused by intra-articular adhesions or intra-articular fibrosis without structural injury to the joint is yet unknown, and literature lacks clear data on the possible causes. In our opinion, it is seen closely correlated to the idiopathic stiff shoulder, with the only significant difference of a defined trauma to which the patient refers the onset of the clinical symptoms. Possible comorbidities (diabetes, hyperthyroidism, etc.) have already been discussed in detail within the specific previous sections and chapters.

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Postoperative Shoulder Stiffness After Rotator Cuff Repair

5

Benjamin Barden, Jessica DiVenere, Hardeep Singh, and Augustus D. Mazzocca

5.1 Introduction

Clinical failures after repair of the rotator cuff are usually attributed to disruption of the repair itself and can be associated with pain and loss of active motion. However, postoperative stiffness, characterized by loss of motion, may occur with or without disruption of the repair. In fact, postoperative shoulder stiffness (POSS) has been shown to be one of the most frequent complications of surgical repair of the rotator cuff (RCR) [7]. Shoulder stiffness may result from any combination of anatomic, neurologic, physiologic, or biomechanical maladies. Although its incidence is not well established, available studies report an incidence of 4.9–32.7 % [31] (Table 5.1). Although POSS is considered one of the most frequent complications after RCR, most studies report excellent outcomes with good mobility 1 and 2 years after surgery [3, 54].

In studies exploring etiologies of shoulder stiffness, Beaufils, Holloway, and Schenk found that patients with POSS demonstrated significantly inferior outcomes when compared to those patients with posttraumatic or idiopathic stiffness [3, 18, 29]. The prevention and management of POSS after RCR is critically important to achieving good functional outcomes where patients are capable of regaining a normal passive and active range of motion (ROM) with sufficient strength to perform activities of daily living, work, and sporting activities (Fig. 5.1) [16].

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Table 5.1 Incidence of POSS after RCR

Study	No. of patients	Incidence
Incidence and treatment of postoperative stiffness following arthroscopic rotator cuff repair (Huberty et al. [31])	489	4.9 %
The factors affecting stiffness occurring with rotator cuff tear (Seo et al. [49])	117	32.7 %
Complications after arthroscopic rotator cuff repair (Brislin et al. [7])	263	8.75 %
Reduction of postoperative stiffness after arthroscopic rotator cuff repair: Results of a customized physical therapy regimen based on risk factors for stiffness (Koo et al. [36])	79	0 %
Stiffness and frotator cuff tears: incidence, arthroscopic indings, and treatment results (Tauro [54])	72	4.2 % resistant stiffness
Functional outcome of arthroscopic repair with concomitant manipulation in rotator cuff tears with stiff shoulder (Cho and Rhee [12])	45	3 %
Does slower rehabilitation after arthroscopic rotator cuff repair lead to long-term stiffness? (Parsons et al. [45])	43	23 %

Resistant stiffness that did not resolve with prolonged period of physical therapy

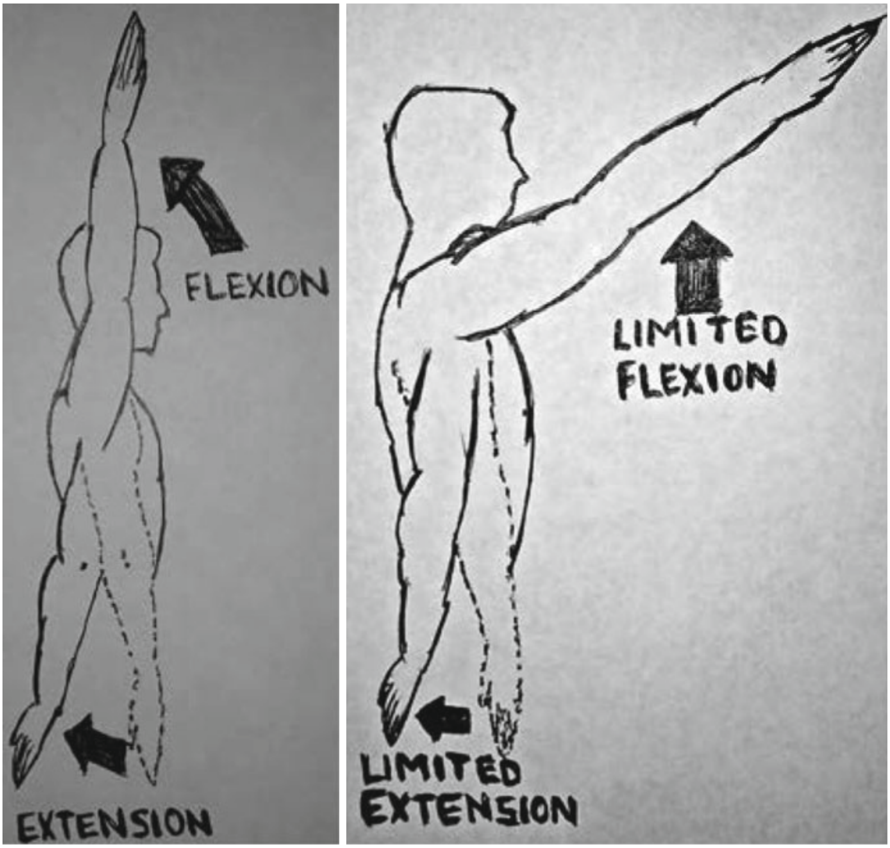

Fig. 5.1 Standardized range of motion assessment

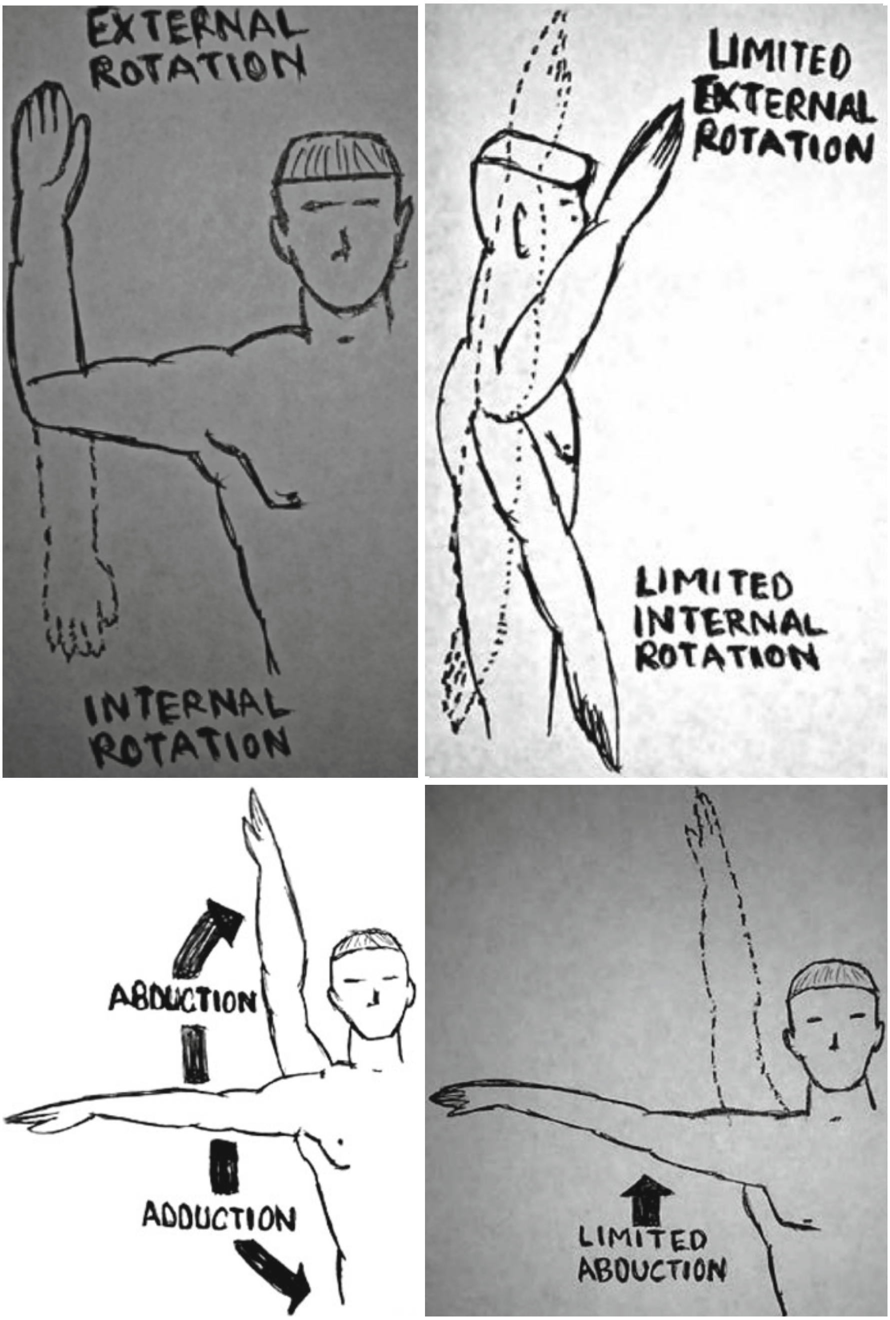

Fig. 5.1 (continued)

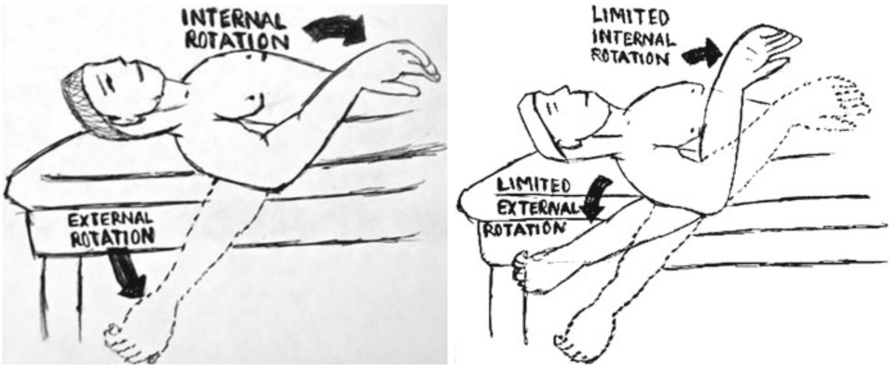

Fig. 5.1 (continued)

Unfortunately, surgeon consensus is still lacking regarding the etiology of POSS and the most efficacious method(s) by which to predict and prevent its occurrence in the setting of RCR. This should come as no surprise as there is a paucity of adequately powered scientific studies to empirically define the incidence and etiology of POSS after RCR (Table 5.1). Current theories postulate that POSS may be related to postoperative immobilization, scapulohumeral adhesions, tear morphology, errors in surgical technique, capsular contracture, or underlying predisposing patient comorbidities such as diabetes to list just a few.

It is therefore extremely challenging to distill consensus from the literature regarding the true incidence and etiology of POSS after RCR. This chapter aims to objectively provide the reader with a summary of the current body of literature addressing this topic and subsequently provide a committee consensus.

5.2 Incidence

In a 2011 systematic review of the literature, Burkhart et al. aimed to define the incidence of stiffness as a primary outcome after arthroscopic rotator cuff repair (ARCR) and found only three articles providing adequate description. Papalia et al. undertook a similar literature review to Burkhart's and identified only five publications relevant to the topic in 2012. Burkhart el. al reported an incidence of transient stiffness responsive to nonoperative management of 10 % and permanent stiffness requiring capsular release of 3.3 % [17] (Table 5.1). All three studies defined stiffness differently, thus preventing meaningful comparisons between them. In general, objectively evaluating the incidence and etiology of POSS after RCR in the literature is made difficult by studies that have been confounded by a focus on postoperative rehabilitation protocols (which have yielded conflicting results), the role for surgical interventions such as capsular release, and preoperative risk factors as it relates to tear size and morphology. Furthermore, there remain very little basic science data with only a few published studies reporting findings in an animal model [33, 46].

Table 5.2 Shoulder stiffness definition by authors

Study	Definition of shoulder stiffness
Seo SS, et al., The factors affecting stiffness occurring with rotator cuff tear. *J Shoulder Elbow Surg*. 2012. [49]	Restriction of active and passive motions of 100° of elevation or less, <50 % of external rotation when compared with the motion of the contralateral shoulder and internal rotation only to the sacrum
Parsons, B.O., et al., Does slower rehabilitation after arthroscopic rotator cuff repair lead to long-term stiffness? *J Shoulder Elbow Surg*. 2010. [45]	Passive forward elevation was <100° and passive external rotation was <30° in the operated-on shoulder
Brislin KJ, Field LD, Savoie FH. 3rd, Complications after arthroscopic rotator cuff repair. *Arthroscopy*. 2007. [7]	Total passive external rotation with the arm at the side of <10°, total passive external rotation with the arm in 90° abduction of <30° or total passive forward flexion of <100°. Diagnosis was made only when these motion deficits persisted for 90 days postoperatively
Tauro JC, Stiffness and rotator cuff tears: incidence, arthroscopic findings, and treatment results. *Arthroscopy*. 2006. [54]	Total passive ROM deficit (abduction, forward flexion, external rotation and internal rotation added together): 0–20° = mild stiffness; 25–70° = moderate stiffness and >70° = severe stiffness
Hsu SL, et al., Surgical results in rotator cuff tears with shoulder stiffness. *J Formos Med Assoc*. 2007. [30]	Active and passive limitation of motion of equal to or more than half the normal range for at least 3 months. The ranges of motion were flexion 90°, abduction 90°, external rotation 25°, and internal rotation sacral level

From Papalia et al. [44]

5.3 Definition

In an effort to further explore the relationship between rotator cuff repair and postoperative stiffness, we must first define shoulder stiffness – a task easier said than done. The definition of what constitutes a stiff shoulder is controversial in and of itself. Table 5.2 demonstrates the variability in definitions employed by current authors on this topic.

Postoperative stiffness is most likely the result of the surgical violation of tissue planes resulting in pathologic connections between motion interfaces, contractures of the soft tissue surrounding the articulations, and/or shortened muscle-tendon unit excursion. The sources of stiffness can occur independently of each other or in combination. From a strictly biomechanical perspective, a stiff shoulder is ultimately one in which at least one of the shoulder's motion interfaces has been compromised, which subsequently limits maximal excursion. Several authors submit that stiffness after rotator cuff surgery is typically global but with an accentuated posterior capsular stiffness pattern. However, it is not yet clear in the literature if POSS after RCR has a predilection for motion in a singular plane or impacts global ROM. The history given by the patient is critical to identifying the etiology of stiffness. This may or may not be accompanied by clinically apparent pain and/or weakness.

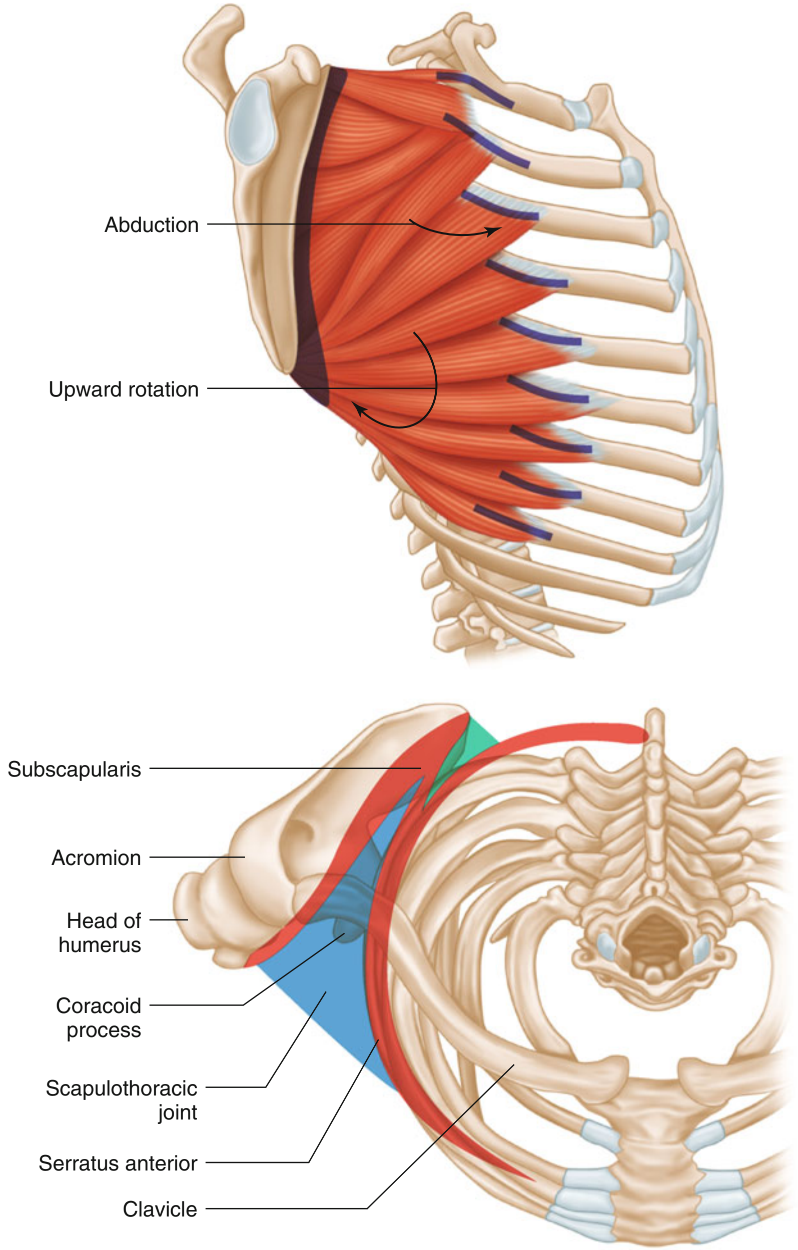

Fig. 5.2 Scapulothoracic interface

We Propose the Following Definition of Shoulder Stiffness At its core, we believe that a definition of stiffness in the postoperative setting is ultimately defined by a loss of passive motion in one or multiple planes. Thus, when joint surfaces are normal and stably aligned, and have no skeletal block, a restriction in passive ROM in any plane, as compared to the contralateral side, is classified as stiffness. This definition is conducive to defining range of motion via a standardized, quantitative, and objective measurement in degrees. These range of motion values must then be clinically contextualized by comparison to physiologic/biomechanical norms established in the literature and perhaps even more importantly, to the patient's own physiologic control via comparison to the contralateral limb.

Postoperative changes from the violation of soft-tissue planes can cause a limitation of glenohumeral rotation in all planes, leading to a global or specific loss of active and passive motion. Comprehensive range of motion testing should include measures of forward flexion, abduction, internal and external rotation with arms at the side, and in 90° of abduction, horizontal adduction, and abduction in the scapular plane. Accurate measurements are best obtained with use of a goniometer. Physical examinations confounded by pain can benefit from targeted subacromial or intra-articular 1 % lidocaine injections to allow the clinician to discriminate between deficits in active versus passive ROM.

Finally, not all shoulder stiffness is of clinical significance and a diagnosis of shoulder stiffness must be contextualized within the clinical framework of each patient to arrive at its etiology, prognosis, and successful treatment.

It should be noted that cumulative shoulder arc of motion is also highly dependent upon a functional scapulothoracic interface. The scapula has three degrees of freedom for rotatory motion; it can move in three planes around three axes (see Fig. 5.2). Scapular movement is inherently dependent upon functional acromioclavicular and sternoclavicular joints. For every 2° of glenohumeral motion there is approximately 1° of scapular motion.

5.4 Etiology of Postoperative Shoulder Stiffness After Rotator Cuff Repair

A major challenge to the identification of the etiology(s) of POSS after RCR is the historically common practice of including all patients with reduced postoperative shoulder motion into the same study cohort. In particular, it is important not to lump all postoperative stiffness into a diagnosis category of "frozen shoulder" as this nomenclature often refers to the intra-articular pathologic condition known as primary or idiopathic stiff shoulder. Although the exact etiology of those with true idiopathic stiff shoulder is not yet clear, its resulting stiffness should likely be considered a distinct pathophysiologic process.

Unlike frozen shoulder, POSS after RCR represents an *acquired* loss of motion that occurs after a known surgical or traumatic event. Authors have proposed a multitude of organizational schema in an attempt to identify and classify potential factors contributing to the etiology of acquired postoperative stiffness. For example, Warner JP et al. have suggested that acquired stiffness after RCR is attributable to one or more of the following elements: (1) a preexisting comorbid condition; (2) the operative technique; or, (3) the postoperative rehabilitation protocol. Matsen et al. propose that three different basic forms of acquired shoulder stiffness can be defined: (1) intra-articular capsular contracture; (2) contractures and/or adhesions of gliding tendons such as the rotator cuff or biceps; and (3) adhesions within the

Table 5.3 Possible causes of postoperative shoulder stiffness after RCR

Extra-articular	**Intra-articular**
Subacromial adhesions [17]	Tear morphology [14, 17]
Subdeltoid adhesions	Musculotendinous unit shortening due to tendon tissue loss
Shortening of myotendinous unit due to muscle atrophy	Secondary/acquired adhesive capsulitis [31]
Scapulothoracic dysfunction	Capsular contracture [7, 31, 54]
Mechanical impingement	Intrinsic osseous disease (osteoarthritis, deformity)
Untreated A/C arthrosis	Repair failure
Scapulothoracic dysfunction	
Technique	**Other**
Open vs. arthroscopic repair [14, 50]	Preoperative stiffness [12, 54]
Over tensioning of tendon repair	Infection
Postoperative rehabilitation protocol [36, 45]	Neurologic injury
	Endocrine disorders (ex. Diabetes) [11, 14]
	Comorbidities

extra-articular humeroscapular motion interface [25, 39]. McLaughlin believed that "acute synovitis represents one phase of the life cycle of this condition [27]." In the postoperative setting of rotator cuff repair, Harryman et al. have suggested that patients with a stiff shoulder can be categorized broadly into one of four subgroups: (1) stiffness without re-tear; (2) stiffness with re-tear; (3) stiffness with untreated osteoarthritis; and, (4) stiffness with deltoid or neurovascular injury with or without re-tear [27]. Bigliani et al. attributed stiffness to inadequate postoperative rehabilitation [6].

Although these schemas provide for a simple and structured approach to POSS, currently available literature has yet to validate this approach. Postoperative shoulder stiffness after RCR may result from a multitude of both intra-articular and extra-articular causes, including but not limited to the following (Table 5.3).

5.5 Postoperative Surgical Changes: Adhesions

Postoperative adhesions represent pathologic connections between motion interfaces responsible for unencumbered shoulder motion. The actual incidence of postoperative adhesions after rotator cuff repair and their direct impact on postoperative shoulder stiffness are unknown. It has been our experience that these adhesions, particularly in the subacromial space and subdeltoid recess reside in the location of local tissue trauma secondary to portal or incision placement. Rarely are they present when a diagnosis of idiopathic frozen shoulder is responsible for shoulder stiffness.

5.6 Biomechanical Impact of Postoperative Adhesions on Shoulder Motion

Given the potential multifactorial etiology of postoperative shoulder stiffness, a basic understanding of shoulder biomechanics can aid the clinician in accurately defining stiffness and then identify the causative agent(s).

The humeroscapular motion interface is composed of a set of gliding surfaces (Fig. 5.3):

- Glenohumeral joint where the humeral head interfaces with the glenoid surface.
- Periarticular interface(s) where the proximal humerus, covered by the rotator cuff tendons, interact with overlaying structures attached to the scapula, including the deltoid, acromion, coracoarcomial ligament, coracoid, and the tendons of the coracoid muscles.

The glenohumeral joint is inherently lax to allow for a wide range of multiplanar motion that is necessary for work, daily living, and athletic activity. A relative capsular laxity in the mid-ranges of motion is a feature of normal shoulder motion. For example, the translational laxity of the normal shoulder facilitates 1 cm of multiplanar excursion when the glenohumeral joint is in mid-range positions. Acting primarily as passive restraints, the glenohumeral ligaments work to limit excessive rotational or translational ranges. The major stabilizing force throughout the majority of mid-range positions is imparted by the dynamic compressive forces generated by glenohumeral joint musculature contractions.

Maximal physiologic multiplanar range of motion about the shoulder is dependent upon four articulations and two bursal-lined interfaces of the shoulder girdle. These interfaces must have stable alignment, smooth articular and motion plane surfaces, and must be free from contractures in the tissues about the joint. To fully mobilize the extremity, the surrounding musculotendinous units must retain both their flexibility and anatomic excursion. This has been well documented by Matsen et al. when they demonstrated that approximately 4 cm of motion takes place in the subacromial and subdeltoid space(s) during normal shoulder movement [39] (see Fig. 5.4).

Scar tissue in any one or combination of the four articulations and two bursal-lined interfaces of the shoulder girdle can result in a mechanical tether. Tauro et al. reviewed a series of open rotator cuff repairs and found a 4 % incidence of significant postoperative adhesions [61]. Acting as a tether, adhesions can limit bony and soft-tissue excursion. Matsen et al. postulate that scarring and adhesions are particularly likely to occur after previous surgery or injury causing bleeding around the shoulder [40].

Matsen et al. speculated that these adhesions, or spot welds, appear to be particularly common after acromioplasty, cuff surgery, or fracture surgery, when early postoperative motion was not implemented (Great picture of Spot Welds scar tissue Matsen Shoulder Surgery, p. 45, Figs. 6–12). Although the literature remains inconclusive regarding the role postoperative motion plays in adhesion formation

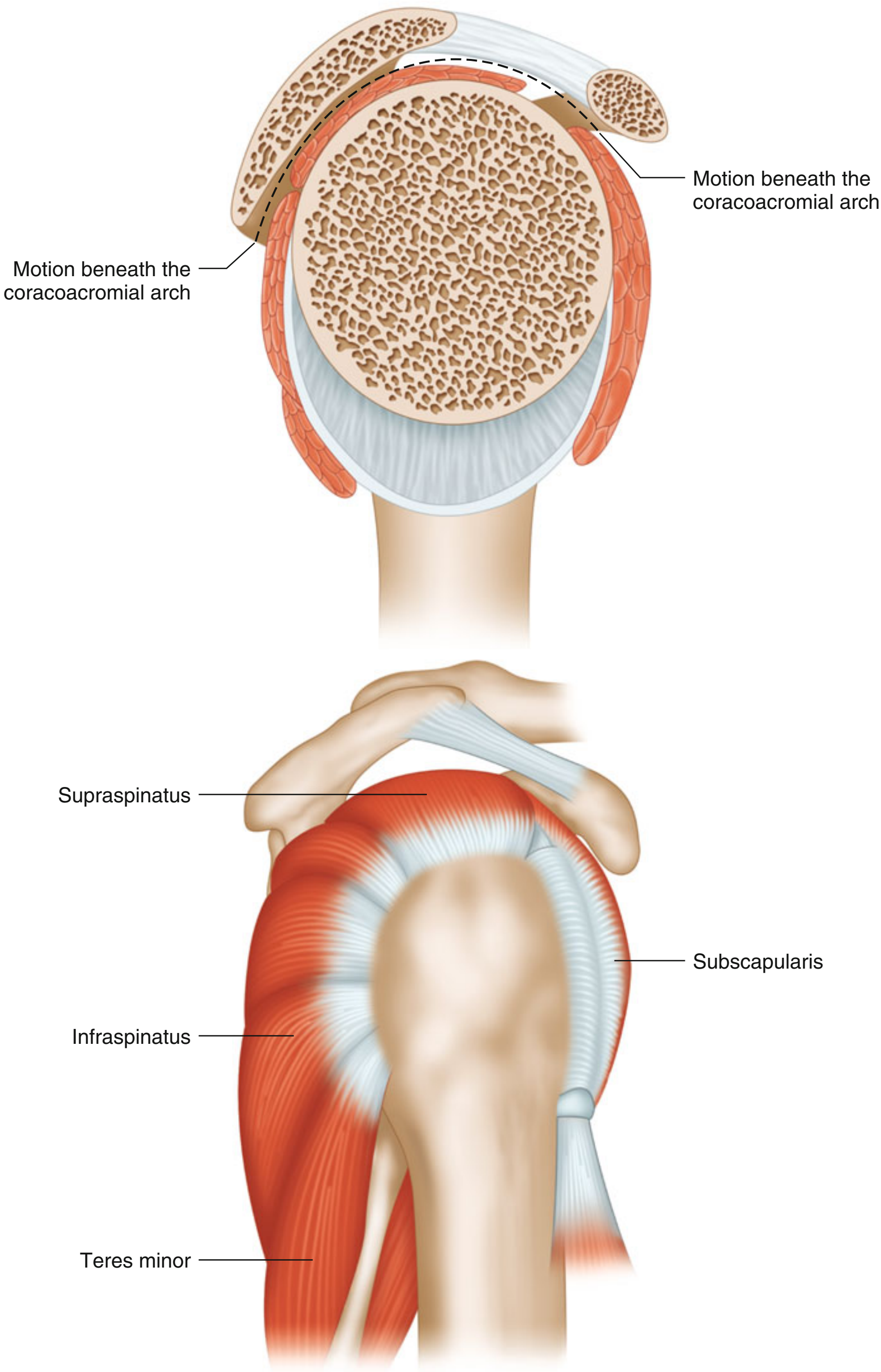

Fig. 5.3 Humeroscapular motion interfaces

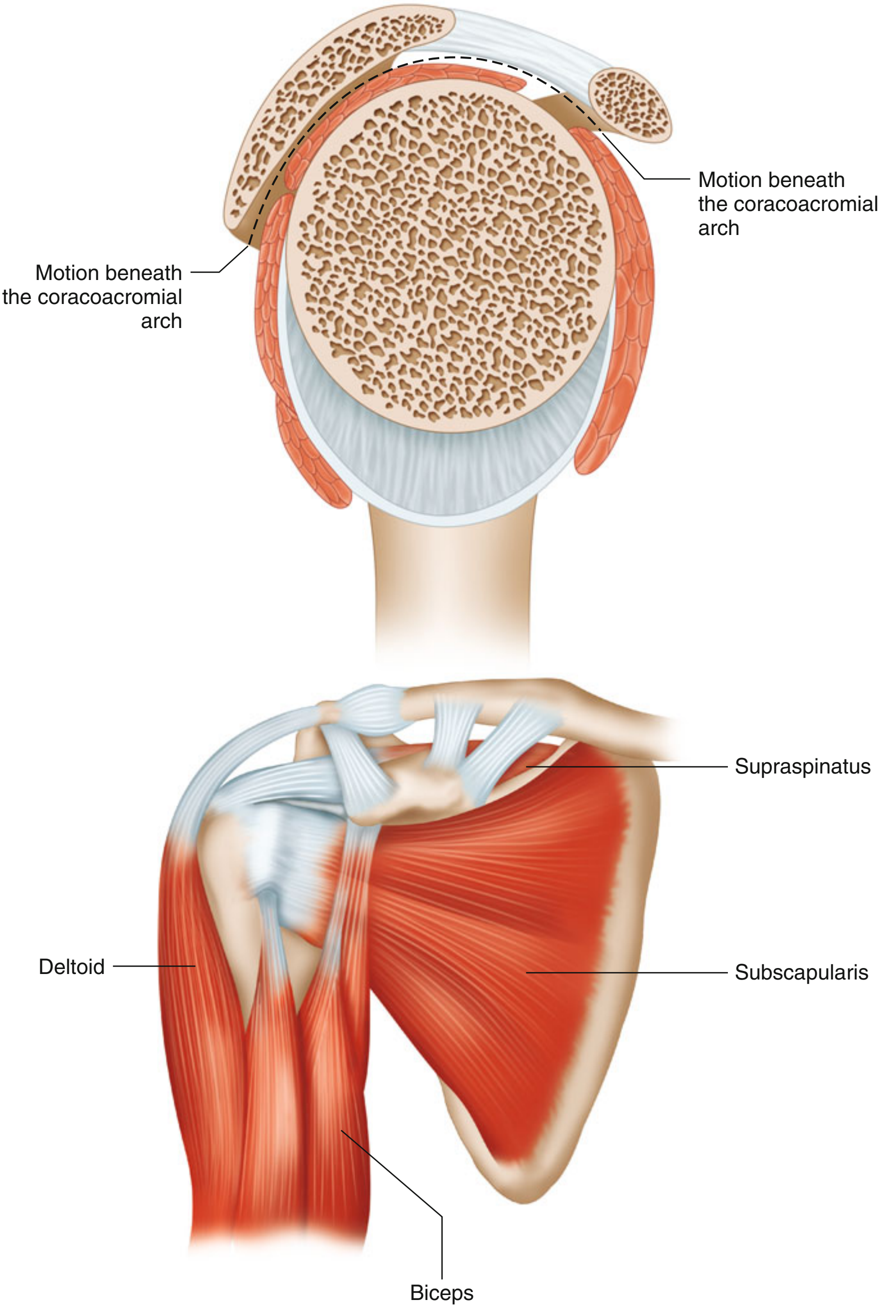

Fig. 5.4 Anatomic excursion in the subacromial and subdeltoid spaces

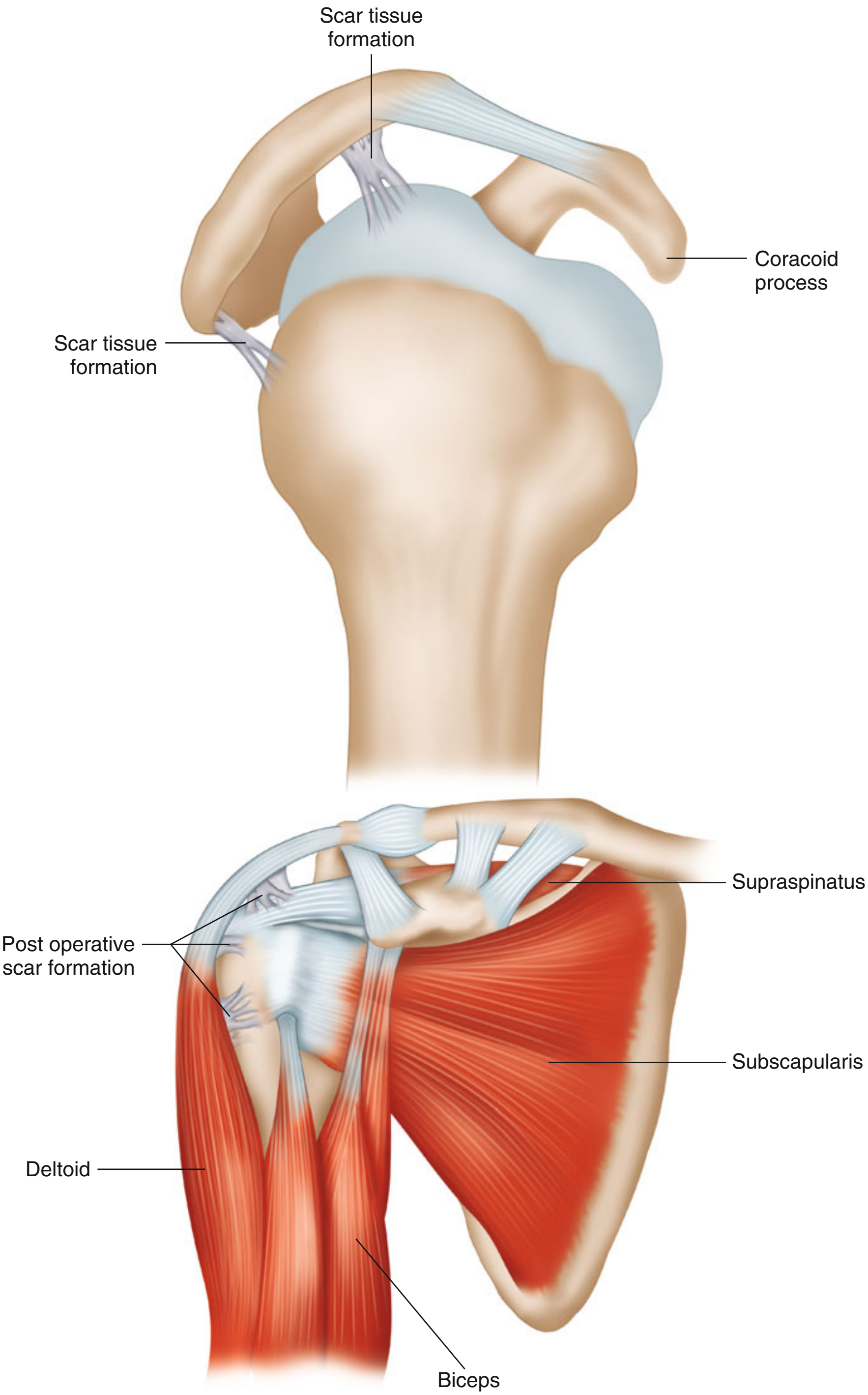

Fig. 5.5 Adhesions of the subacromial and subdeltoid spaces

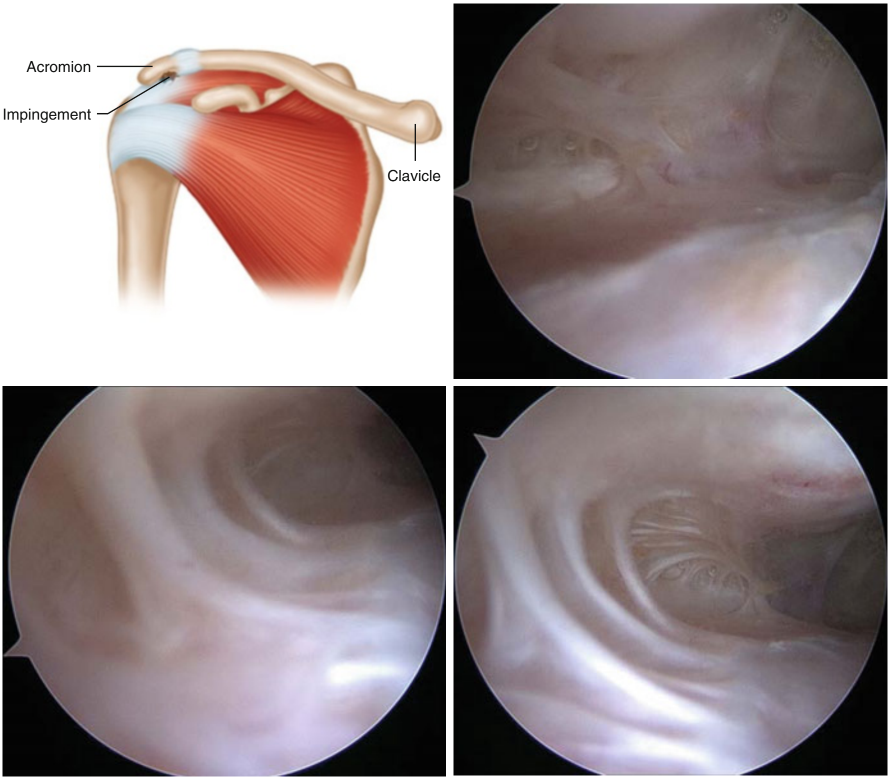

Fig. 5.5 (continued)

(see section 5.7), we do know that postoperative tissues are often inflamed and undergo hypertrophic changes. Many surgeons will attest to the thickness of postoperative adhesions in the shoulder and that attempting to lyse them with manual manipulation can require a significant force that may jeopardize the integrity of the humerus or rotator cuff.

It is difficult to quantify the incidence of POSS after RCR in our practice. However, it is a rare occurrence in our shoulder practice that a patient must return to the operating room for POSS after RCR that is refractive to extensive physical therapy. If a postoperative patient still has persistent activity-limiting stiffness 9 months postoperatively, arthroscopic lysis of adhesions with capsular release and subsequent manipulation under anesthesia is a treatment option offered to the patient. Anecdotally, a common intra-operative finding in this patient group has been extensive subacromial and subdeltoid bursal adhesions. These adhesions are particularly prevalent at the sites of previous soft-tissue trauma where tissue planes have been violated by arthroscopic cannulas or instrumentation. Often, a pervasive pattern of scar tissue formation will span the majority of the subacromial space. These adhesions are impressively thick and under dynamic arthroscopic visualization demonstrate robust tethers to cuff excursion (Fig. 5.5).

In our opinion, there is a subset of patients with POSS after RCR that also present with disproportionate pain in conjunction with stiffness. We recognize that capacity for pain tolerance varies among patients. However, it has been our suspicion that patients with subjectively disproportionate pain in conjunction with POSS after RCR, may have had preoperative underlying idiopathic frozen shoulder with subsequent postoperative exacerbation (see section "Secondary adhesive capsulitis").

5.7 Postoperative Therapy Protocols

A review of the literature demonstrates conflicting reports in regard to postoperative range of motion protocols and their impact on the incidence of postoperative stiffness. Furthermore, there are very few clinical studies on this subject. Most of the published literature evaluating the role of postoperative physical therapy after RCR has focused on therapy's impact on rotator cuff healing rates and its influence on the rate of re-tear. These data often cite clinical experience and focused on open surgery, not arthroscopic surgery [34].

Ideally, the most efficacious postoperative rehabilitation program is one that facilitates the best clinical outcome possible where repair integrity is maintained with motion and strength optimally restored.

5.7.1 Early Postoperative Motion Protocols

Historically, postoperative stiffness was one of the most devastating complications for shoulder surgeons. "Acquired shoulder stiffness is most commonly encountered after surgery or trauma, especially after prolonged periods of immobilization" [29, 47, 60, 61]. Efforts to avoid stiffness led to the popularization of early passive range of motion after rotator cuff repair [15, 19, 28, 41]. Many surgeons are of the opinion that postoperative positioning in an abduction brace can help keep the inferior glenohumeral joint capsule stretched out, and passive stretching exercises can be initiated immediately after surgery if the repair is felt to be strong enough to withstand the stress. Proponents of early postoperative range of motion, such as Matsen et al., theorize that adhesions appear to be particularly common after acromioplasty, cuff surgery, or fracture surgery when early postoperative motion is not implemented. They theorize that stiffness may result from pathologic connections between motion interfaces, a contracture of soft tissues surrounding the articulations, or a shortening of the musculotendinous unit. A primary motivating force behind this postoperative approach was the extrapolation of flexor tendon repair data to the shoulder. Other authors, such as Abrams et al. have supported early postoperative motion protocols but have cautioned against advancing motion protocols too rapidly as this can lead to an inflammatory response [1, 62] (see Fig. 5.6). As such, Abrams et al. suggest that early postoperative motion routines may need to be modified if a patient encounters significant pain during therapy.

A review of the literature suggests that most studies addressing rotator cuff repair and rehabilitation protocols incorporated immediate passive ROM exercises for the first 3–6 weeks with the patient wearing an abduction shoulder brace during that time [2, 10, 21, 26, 32, 37, 53].

5.7.2 Delayed Postoperative Motion Protocols

Denard and Burkhart (*The Shoulder*, p. 65) advocate that the notion of early passive range of motion to avoid stiffness is a misconception that is a carryover

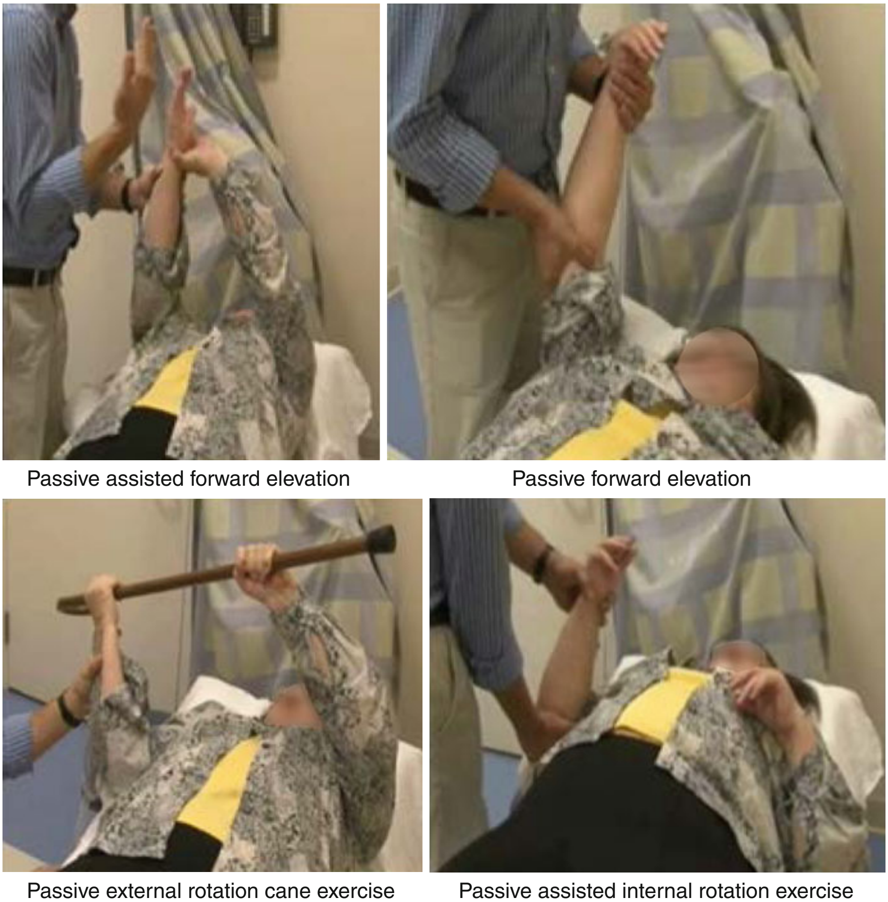

Fig. 5.6 Passive range of motion exercises

from the days when open rotator cuff repair created greater soft-tissue damage. Burkhart et al. have reported that using their protocol of delayed range of motion, stiffness was observed in only 2 % of massive rotator cuff tears [9]. Clinical studies have shown that only 4.5 % of patients develop clinically important stiffness with a 6-week immobilization protocol following arthroscopic rotator cuff repair [17, 31, 45]. Burkhart's data are supported by a more recent 2014 Level I study by Koh et al. (the only Level 1 study of its kind addressing POSS after RCR) in which 100 patients were randomized to either 4 or 8 weeks of postoperative immobilization in a sling after RCR. In patients without preoperative stiffness or diabetes, they reported a 4 % incidence of stiffness in the 4-week immobilization group [34]. At the time of final follow-up, there was no significant difference in range of motion or clinical scores among the 4-week and 8-week immobilization groups. However, it should be noted that the proportion of patients showing POSS was higher in the 8-week immobilization group (38 % compared to 18 %).

Denard and Burkhart assert that aggressive early passive range of motion is detrimental to healing (*The Shoulder*, Denard and Burkhart, Page 65). Interestingly, Koh et al. have demonstrated that an immobilization period of 4 or 8 weeks does not result in significant long-term stiffness and their re-tear rate (identified by 6-month postoperative MRI) was only 10 % – a significantly lower incidence than comparable reports in the literature [34]. Although their re-tear rate was very small, Koh et al. reported that early aggressive passive ROM could result in twice the re-tear rate when compared to limited passive ROM but that this difference was not statistically significant in their cohort [38].

Recent basic science investigations support the assertion that early mobilization produces strain on the rotator cuff and compromises healing [4, 5, 8, 24]. Animal models used to evaluate the effects of postoperative immobilization or early range of motion exercises have demonstrated improved healing and improved mechanical properties when immobilization without passive ROM was used after rotator cuff repair [22–24, 35, 46, 51, 56, 58]. Addressing POSS after RCR, there are several basic science investigations reporting that the inflammatory response in the animal model is higher following open rotator cuff repair, and immediate passive range of motion may actually increase postoperative adhesions in an animal model [46]. This is not to say that POSS does not occur. In fact, POSS is relatively common but Sarver et al. reported that immobilization following rotator cuff repair in an animal model led to stiffness that was transient [48]. Thus, there is growing basic science evidence that delayed therapy after arthroscopic rotator cuff repair improves tendon healing and does not impair motion [45]. It should be noted, however, that much of these data has been produced in the context of open RCR and conclusions may have different implications in an arthroscopic model where there is less soft-tissue dissection and trauma.

The role of preoperative stiffness and its impact on postoperative stiffness shoulder should be noted (see Preoperative Stiffness and POSS After RCR). In Koh et al.'s Level I study, they found that patients with preoperative stiffness had approximately a 50 % rate of postoperative stiffness regardless of their duration of immobilization periods studied (4 weeks or 8 weeks) [5].

In an interesting study published by Koo et al. [36], the authors aimed to determine the benefits of a modified rehabilitation protocol incorporating early closed-chain passive overhead stretching for reducing the incidence of POSS after RCR in patients with at least 1 of 5 risk factors previously established by Huberty et al. [31]: (1) calcific tendinitis; (2) frozen shoulder; (3) PASTA repair; (4) concomitant labral repair; and (5) single-tendon repair [31]. They compared results of this modified rehabilitation protocol to their conservative protocol utilizing 6 weeks or sling immobilization. Among the 152 patients with primary rotator cuff tears in this study, there were 73 who did not have any risk factors for stiffness; 79 patients had 1 or more risk factors and were allocated to the modified rehabilitation protocol. Postoperative stiffness developed in none of the 73 conservative therapy patients or those patients with risk factors enrolled in the modified rehabilitation program. This is significantly lower than an incidence of 7.8 % reported in their historical high-risk controls.

An evaluation of postoperative immobilization protocols in our practice appears to be highly variable and is not only surgeon-dependent, but it shows tremendous variability among patients of individual surgeons. This variability stems from a general consensus that postoperative immobilization protocols should be custom-tailored to each patient in an effort to maximize their both objective and patient-centered clinical outcomes taking into account preoperative comorbidities, tear morphology, tissue quality, repair characteristics, and capacity for patient compliance.

Limitations on ROM parameters are dictated to the patient and physical therapist based on the surgeon's intraoperative assessment of repair and tissue quality. In relatively younger patients with partial thickness articular-sided rotator cuff tears (PASTA), or small full thickness tears involving relatively robust tissue quality, our surgeons tend to initiate early supine passive ROM in an effort to prevent POSS. Active-assist and active motion is deferred until at least 4–6 weeks when initial tendon healing can be expected adequate enough to tolerate minor cuff stresses. For elderly patients and patients with exceptionally osteoporotic bone, and/or patients with significant degradation of cuff tissue quality such that there is an increased risk of anchor or suture pull-out, we tend to utilize a prolonged period of immobilization up to 6 weeks in an abduction sling. Like many others, this practice is based on clinical experience/opinion and has not yet been validated in the orthopedic literature.

5.8 Risk Factors for Postoperative Shoulder Stiffness

In their systematic review of the literature, Burkhart and Denard found only three studies that examined both the incidence of POSS and risk factors for POSS [7, 31, 45]. Two of the articles described a "transient" and a "resistant" stiffness [7, 45]. One of the articles described only resistant stiffness [31]. The combined sample between the three articles was 795 patients. They concluded that tear size contributed to the development of both transient and resistant stiffness. In fact, one study reported that patients with tears less than 3 cm in dimension were more likely to have transient stiffness, although the difference did not reach statistical significance with the sample size of 43 patients [45]. In the two larger studies reviewed, there was a trend toward a higher incidence of stiffness in patients with smaller tears [12] and significantly higher rates of stiffness in partial articular-sided tears versus 3- or 4-tendon tears (13.5 % vs. 2 %, $P < .05$) [31]. Two studies analyzed surgical fixation technique and did not find a positive correlation to POSS [7, 31]. Additional statistically significant risk factors for the development of POSS were described in one study and included workers' compensation (8.6 %), age less than 50 years (8.6 %), calcific tendinosis, frozen shoulder (15.6 %), or concomitant labral repair (11 %) [31]. Several studies support a higher incidence of POSS after RCR in patients with diabetes [11]; however, specific correlates to HbA_1C have not been reported.

Other authors have identified similar risk factors including Huberty et al. who identified the following factors: (a) calcific tendinitis, (b) frozen shoulder, (c) single-tendon cuff repair, (d) PASTA repair, (e) age < 50, (f) workers compensation insurance, and (g) partial thickness articular-surface tendon avulsion [31]). Namdari and Green identified the following factors: (a) limited preoperative active forward elevation, active external rotation, and passive internal rotation; (b) diabetes; (c) open repair; (d) size of the tear; (e) duration of symptoms; (f) subscapularis tear; (g) biceps tears; (h) workman's compensation; and (i) involvement of the dominant extremity [31]. Trenerry et al. reported that a preoperative limitation in "hand behind the back" motion was a factor associated with an increased risk of stiffness development [57].

5.9 Preoperative Range of Motion and Postoperative Stiffness After Rotator Cuff Repair

Many patients with rotator cuff tears have some degree of stiffness prior to surgery [55] with the literature reporting rates of 17.4–32.7 % [12, 36, 43, 54]. There are only a few articles in the literature that specifically evaluate the incidence of POSS after RCR in relation to preoperative stiffness. In Burkhart's systematic review, he found two articles that focused on preoperative stiffness in patients undergoing ARCR [12, 54]. There is no general consensus among surgeons if preoperative stiffness will greatly increase the difficulty in regaining full postoperative range of motion. Tauro et al. have reported that only patients who have frozen shoulder at the time of surgery will fail to recover satisfactory postoperative ROM [54, 57]. In Koh et al.'s Level I study, they found that patients with preoperative stiffness had approximately a 50 % rate of postoperative stiffness regardless of their duration of immobilization (4 weeks or 8 weeks) [5]. Other authors note that patients who start out with mild or moderate stiffness before surgery will have persistent stiffness after surgery, but the prognosis for ultimate recovery of near full motion has been found to be very good [55]. Several of these studies demonstrate that preoperative stiffness in patients when managed concomitantly during rotator cuff repair does not alter their clinical outcomes [13, 43].

Harryman et al. retrospectively categorized 72 patients with full thickness tears undergoing ARCR into having a mild (0–20°), moderate (20–70°), or severe (>70°) deficit in total preoperative range of motion [28]. No capsular releases were performed at the time of surgery. Final deficits in each plane of motion were not provided. They reported that mean total range of motion deficits decreased from 10 to 4° in the mild group, 36–12° in the moderated group, and 89–31° in the severe group. In patients with a total deficit of less than 70°, they reported that there was no resistant postoperative stiffness. Of the six patients with a preoperative deficit of more than 70°, three had resistant postoperative stiffness.

Nicholson et al. prospectively compared 15 patients with preoperative stiffness (passive forward flexion <100° or external rotation <40°) with 30 patients without preoperative shoulder stiffness. Preoperatively, the mean passive motion was forward flexion of 118° and external rotation of 35° in the stiffness group compared with 163° and 55°, respectively, in the group without stiffness. In contrast to the study by Tauro et al. [55], a manipulation under anesthesia was performed in the patients with stiffness before ARCR. At final follow-up of more than 2 years, there was no significant difference in forward flexion (167° vs. 170°, $p = .157$) or external rotation (49° vs. 53°, $P = .384$) between the groups with and without preoperative stiffness. The rate of motion recovery, however, was slower in the group with preoperative stiffness. External rotation took 3 months to become equal between both groups. Recovery was slower for forward flexion, with patients with stiffness preoperatively having 13° less at 3 months, 9 less at 6 months, and 6° less at 1 year compared with the normal group ($P = .21$).

It has been our experience that patients presenting with preoperative shoulder stiffness in association with rotator cuff tear benefit from concomitant capsular release followed by gentle manipulation under anesthesia at the time of RCR.

5.10 Operative Techniques and Postoperative Stiffness: Open Versus Arthroscopic Techniques

Open shoulder procedures can be complicated by postoperative stiffness. One review of open rotator cuff repairs showed a 4 % incidence of significant postoperative adhesions [61]. Open shoulder surgery almost always violates the motion planes external to the glenohumeral joint. Observations at the time of surgical intervention for shoulder stiffness often include adhesions between the bursal surface of the rotator cuff and proximal humerus, the deep surface of the deltoid, CA arch, and conjoined tendons (Fig. 5.7).

Recent basic science investigations have suggested that the inflammatory response is higher following open rotator cuff repair. In an interesting study, Shinoda et al. randomized 32 patients to open repair or ARCR and demonstrated a threefold increase in interleukin-6 levels postoperatively in the open repair group compared to ARCR [50]. Franceschi et al. demonstrated threefold increases in levels of substance P in patients who developed postoperative stiffness [20]. Thus, from a basic science perspective, surgical technique, open versus arthroscopic repair, plays a role in the development of postoperative stiffness after rotator cuff repair.

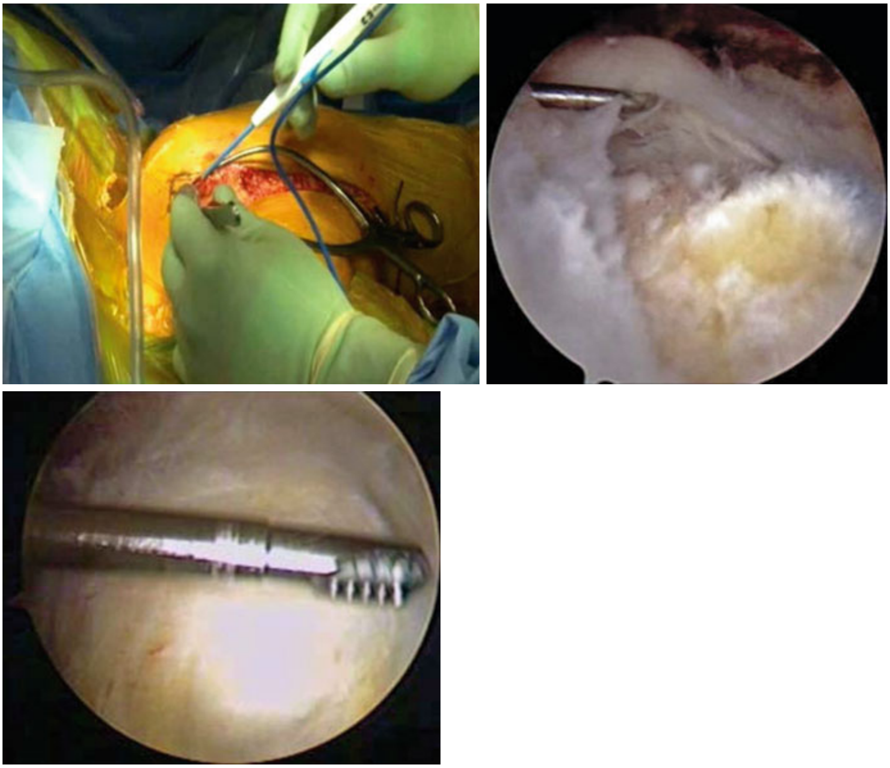

Fig. 5.7 Open vs. arthroscopic repair technique

Irrespective of surgical approach, incorrect operative technique puts a patient at risk for POSS. Technical errors include inadequate soft-tissue releases, especially of the coracohumeral ligament, and tight closures of the rotator interval with the glenohumeral joint in a position of internal rotation. Repairs achieved by excessively advancing the musculotendinous unit will often result in a loss of internal rotation if the posterosuperior cuff was advanced, and decreased external rotation if the subscapularis was over advanced. Finally, failure to address any associated pathologic processes contributing to shoulder stiffness, such as biceps disease or acromioclavicular arthropathy, can put the patient at risk for POSS.

5.11 Tear Morphology and Postoperative Stiffness

Stiffness is unusual after repair of massive tears, because most of the capsule was off with the tendon and both are often thin and insubstantial [42]. Conversely, patients with small tears, with thick, robust tendon (and attached capsule), not infrequently may become stiff after repair.

5.12 Postoperative Secondary/Acquired Shoulder Stiffness

Adhesive capsulitis etiology has been described as idiopathic or secondary, sometimes referred to as acquired shoulder stiffness. The disease process is characterized by inflammation and subsequent scarring of the joint capsule, decreased intra-articular volume, and a reduction in capsular compliance contributing to motion limitations in multiple planes [60]. Inflammation of the joint capsule and subsequent scarring may also occur as an *acquired* entity after surgery or trauma. This has been a commonly reported postoperative finding after surgical treatment of proximal humerus fractures.

Patients at risk for secondary shoulder stiffness after RCR are those with diabetes and thyroid disorder, previous history of frozen shoulder, large or massive rotator cuff repairs in whom therapy was delayed in order to maximize the tendon healing potential, and acute, traumatic tears repaired in the first week following injury. Another school of thought suggests that any pathologic condition that causes primary or referred pain to the shoulder can potentially initiate an inflammatory cascade leading to an acquired shoulder stiffness [55]. Stollsteimer, G.T. and F.H. Savoie have reported that it is possible for patients who undergo rotator cuff repair to develop secondary postoperative shoulder stiffness [52]. Postoperative stiffness after rotator cuff repair surgery may be the result of capsular contracture, with or without adhesions between tissue planes, which alters the scapulohumeral motion interface. Tauro et al. have reported that only those patients that have frozen shoulder at the time of survey will not recover satisfactory ROM [54, 59]. On the contrary, Savoie and Stollsteimer et al. report that in patients without a preoperative history of frozen shoulder, postoperative stiffness is rarely a significant long-term problem – even for patients in whom formal stretching is delayed 4–6 weeks after surgery [52].

5.13 Clinical Findings of Postoperative Acquired Adhesive Capsulitis

After surgery, some patients will develop severe pain that cannot be explained by the underlying condition – this type of shoulder stiffness seems similar to the idiopathic form of the disease. Symptoms usually do not occur immediately after surgery, but 2–6 weeks after. The patient's pain is constant, often worse at night, and may be refractive to oral analgesics. On physical examination, any manipulation of the shoulder is extremely painful for the patient. There is a loss of passive range of motion particularly in abduction. Active range of motion is limited by pain as well. In these patients, attempts to initiate range of motion physical therapy protocols may exacerbate their chronic pain. Some authors caution that the pain mimics the symptoms of subacromial impingement, limits active range of motion, and is often localized on the lateral side of the arm. Other authors suggest that unlike patients in the acute inflammatory stage of primary frozen shoulder, pain is usually present, especially in the end-range of motion, but motion limitation is the main complaint.

The literature is confounded by variable usage of the terms “acquired” and “secondary” shoulder stiffness when referring to stiffness that can be attributed to a known extrinsic cause such as fracture. It is not yet clear if there is a subset of patients with POSS after RCR that demonstrate capsular histopathology analogous to that seen in idiopathic frozen shoulder. Our clinical experience suggests that there is a subgroup of patients with rotator cuff tears and concomitant capsular changes consistent with idiopathic frozen shoulder. Physical examination findings in this subgroup are suggestive of rotator cuff tear and are often confounded by pain. This pain is often attributed to the rotator cuff tear but may represent an underlying pathologic process idiopathic frozen shoulder. Surgical intervention for cuff tears in this population may exacerbate this underlying frozen shoulder resulting in painful POSS.

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Postoperative Shoulder Stiffness Following Surgical Repair of Shoulder Instability

6

Kevin D. Plancher and Stephanie C. Petterson

6.1 Introduction

Loss of external rotation range of motion is a complication following both open and arthroscopic instability repair procedures [4, 5, 8, 9, 12, 13, 16, 19, 22, 23, 27]. In the hallmark paper by Rowe demonstrating the success of the Bankart procedure, 24 % of patients achieved only 75 % of external rotation range of motion compared to the nonsurgical side [24]. Loss of motion following arthroscopic procedures is still debated. Some authors demonstrate minimal or no loss of range of motion [10, 14, 30], while others show some degree of reduction in motion [5, 22, 27]. In general, range of motion loss following arthroscopic Bankart repair is typically less than 5°; however, many authors only report total range of motion and do not compare it to the accepted normal range [10, 28]. Range of motion loss has also been shown to be greater following revision surgery compared to primary repair [3, 25].

The higher incidence following an open repair has been attributed to compromise to subscapularis muscle and capsular contracture. The use of a subscapularis splitting approach may help to minimize this complication. Following an open repair, longer periods of specific immobilization are warranted to protect the repair of the subscapularis and its anatomy and function, thus also contributing to the greater incidences of loss of external rotation range of motion [3].

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The most important independent risk factor for patient satisfaction following open Bankart repair is loss of range of motion [21]. However, we believe re-dislocation is a much worse prognosis and always strive to achieve stability in a shoulder as the primary goal. Rahme et al. in 2010 reported a fivefold increase in the risk for poor or fair outcome measured by questionnaire in patients with loss of external rotation postoperatively [21]. While the ultimate goal is to minimize at risk positions to prevent a recurrent dislocation, loss of external rotation or forward flexion may hinder functional outcomes particularly in overhead athletes or workers. Therefore, it is important for the surgical and rehabilitation teams to work closely to manage the expectations of each patient and ultimately yield the most successful outcome.

6.2 Etiology

The most common areas for the development of fibrosis in frozen shoulder include the coracohumeral ligament, the interval between the base of the coracoid and the top of the subscapularis, known as the rotator interval, between the conjoined tendon and the subscapularis, between the rotator cuff and the overlying acromion, and the deltoid bursae and musculature in the scapulothoracic region [31]. Higher rates of contracture are seen with procedures that tighten the anterior glenohumeral capsule or expose the deltopectoral interval (i.e., open procedures). Loss of motion following both arthroscopic and open Bankart repair has also been associated with excessive imbrication of the anteroinferior capsule including the anterior band of the inferior glenohumeral ligament [16, 17].

Mengiardi et al. conducted a quantitative analysis of MR arthrogram in patients with severe frozen shoulder and those without clinical or arthroscopic indications of frozen shoulder [15]. The blinded quantitative analysis showed a significantly thicker coracohumeral ligament and thicker capsule in the open rotator interval. The authors also found that patients with severe frozen shoulder had a significantly smaller capsular volume in the area of the axillary recess; however, there was no difference in capsular thickness in the axillary recess. We have previously shown that the rotator interval is dynamic with glenohumeral motion [18]. The rotator interval closes with internal rotation and opens with external rotation. Therefore, if the arm is positioned in internal rotation during imbrication, overtightening will occur and subsequent loss of external rotation [18]. A case-controlled study by Lee et al. demonstrated a thicker coracohumeral ligament and unlike the by Mengiardi et al, study they found the capsule in the axillary recess was thicker in patients with frozen shoulder. Loss of internal and external rotation range of motion was only correlated with a thickened coracohumeral ligament [11].

Rotator interval closure is often indicated for patients with inferior or multidirectional instability. The rotator interval acts to control posterior (in vitro only) and inferior (in vivo) glenohumeral translatory motion [7]. While imbrication of the rotator interval minimizes translation posteriorly and inferiorly, it may also restrict motion in flexion and external rotation [2, 7]. Computer-simulated models have demonstrated that when imbrication of the anterior band of the inferior glenohumeral ligament was increased by 3, 6, and 9 mm, a loss of external rotation range of motion of 10°, 22°, and 36° was seen, respectively [26].

Precise surgical placement of the anterior band of the inferior glenohumeral ligament is a key component in ensuring an excellent instability repair. Similar computer models, previously mentioned, have shown that when the anatomic insertion deviates superiorly by 3, 6, and 9 mm, a loss of external rotation of 5°, 11°, and 13°, can be seen respectively. When the anatomic insertion deviates inferiorly by the same amount, there is increased anteroinferior humeral head displacement [26].

Postoperative immobilization or restrictions in range of motion can contribute to frozen shoulder following surgery [3]. A key element to consider in the immediate postoperative period to minimize the incidence of secondary loss of motion following surgical intervention for repair of shoulder instability is immobilization time. Immediate postoperative external rotation with limits placed on the exercises in a controlled environment may help to overcome shoulder stiffness following instability repair.

6.3 Treatment

When faced with postsurgical shoulder stiffness following instability surgery, physical therapy is always the first line of management. An individualized approach to rehabilitation is important when treating shoulder stiffness in patients following instability repair [20]. The duration of each phase of the rehabilitation program may need to be lengthened depending on the patient's progress. The focus must be on early restoration of shoulder range of motion without stretching the repaired capsule. Aggressive stretching should be avoided for fear of exacerbating pain and stiffness. Restrictions at both the glenohumeral and scapulothoracic joints must both be addressed in order to restore ranges of motion. Furthermore, return to functional activities should be dictated by the patient's tolerance rather than on the phase of the rehabilitation program in order to maintain gains in range of motion. Oral steroids and nonsteroidal anti-inflammatories or intra-articular steroid injections should be administered for pain control, to help decrease inflammation, to calm the tissues, and to aid in maximizing gains in physical therapy.

While rehabilitation can be successful in the early stages of idiopathic frozen shoulder, its success may be limited following surgical intervention. If conservative management fails, surgical intervention may be warranted. Manipulation under anesthesia, while a successful approach for primary, idiopathic frozen shoulder, is cautioned in patients with postsurgical shoulder stiffness. Arthroscopic capsular release is typically indicated when no progress has been made within

4 months for acute cases and 3 months for chronic cases [29]. Arthroscopic intervention allows these contractures to be released in a controlled fashion to minimize risk of the primary repair. The success of arthroscopic capsular release is largely dependent on the degree of stiffness regardless of etiology [6]. Anterior capsule and rotator interval releases are indicated with range of motion deficits in external rotation with the arm by the side, whereas, anterior and inferior capsule releases are indicated with range of motion deficits in external rotation with the arm in abduction [29]. We have found that release of the anterior capsule will paid in gains of forward flexion and release of the posterior capsule can help to increase internal rotation and extension of the glenohumeral joint in an adducted or abducted position. Loss of cross-body adduction can also be corrected with a posterior capsular release.

Arthroscopic rotator interval capsular release is indicated for cases of overtightening the capsule in instability repair procedures. Release of the rotator interval can improve flexion and external rotation range of motion; however, associated increases in inferior translatory motion may compromise the primary repair is cautioned in this population, though to date, the senior author has never experienced this [7].

The arthroscopic procedure for restoration of the anterior transverse sliding (RATS) mechanism of the subscapularis tendon has also been described in patients with loss of external rotation range of motion after surgical stabilization of anterior glenohumeral instability [1]. The RATS procedure involves releasing the adhesions of the subscapularis tendon to the glenoid neck with the goal of movement restoration of the subscapularis tendon. Itoi and colleagues in 2012 demonstrated the success of the RATS procedure in restoring range of motion in seven patients with loss of external rotation range of motion following primary repair to restore anterior glenohumeral instability. Five patients had undergone arthroscopic Bankart repair and rotator interval closure, one patient had undergone open Bankart repair, and one patient had undergone an open Bristow procedure. A thick fibroust issue covering the rotator interval and the anterior aspect of the glenohumeral capsule was found in all patients, which prevented the normal sliding of the subscapularis tendon during rotation of the arm. Following the RATS procedure, all patients demonstrated improved forward arm elevation, external rotation, and internal rotation range of motion, as well as improved function measured by Constant and UCLA scoring systems.

Conclusion

In conclusion, loss of external rotation range of motion is a known complication following surgical intervention for anterior shoulder instability. While the etiology may be the result of the surgical repair technique or prolonged postoperative mobilization, the goal is to restore full functional range of motion to promote return to full activities of daily living, work, and sporting activities. Following a failed conservative attempt to restore range of motion, surgical intervention may be warranted to yield the best outcomes in patients with range of motion deficits following instability repair procedures.

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Postoperative Shoulder Stiffness: Arthroplasty and SLAP Repair

7

Felix H. Savoie III and Michael J. O'Brien

7.1 The Stiff Shoulder Post Replacement

7.1.1 Introduction

Shoulder replacement surgery remains an excellent procedure for osteoarthritis of the shoulder. Although results are quite good, complications may occur [9]. In a recent report, the French group found significant stiffness to be relatively rare in unconstrained shoulder arthroplasty patients. Only 0.9 % of total shoulder arthroplasty (TSA) patients had a problem with stiffness. The purpose of this chapter is to discuss this relatively rare complication of stiffness. In most cases, stiffness will be associated with either posttraumatic arthroplasty or in the presence of subtle infection, most commonly *Propionibacterium acnes* (*P.acnes*) [15]. The most common complications following TSA are infection, neurologic injury, wound issues, and fracture. The true incidence of stiffness without infection is unknown, especially in light of recent papers by Pottinger et al. from the Seattle group indicating a 70 % positive culture rate for *P.acnes* in revision surgical cases that were not thought to be infected.

It is inherent to rule out subclinical infection in the stiff TSA. Aspiration has been routinely recommended but its effectiveness in only 17 % [15]. Routine lab studies including white blood cell (WBC) counts, C-reactive protein (CRP), and erythrocyte sedimentation rate may be inconclusive. Arthroscopic or open evaluation with culture and tissue biopsy has been shown to be effective in diagnosing infection, but with the dilemma of leaving a stable prosthesis in place while awaiting final reports. The loose infected prosthetic replacement has the same problem in determining whether to do a one- or two-stage exchange.

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7.1.2 History

The patient will present with a loss of motion. It is important to understand whether there was preoperative motion loss. Another salient factor is whether motion was gained postoperatively and then stiffness occurred or if motion was never regained. History of subsequent systemic illness, or other factors such as diabetes, may also be important both in determining etiology as well as treatment.

7.1.3 Physical Examination

Inspection for redness, healing of the tissue and atrophy is initially noted. The position of the shoulder on the chest wall should be noted at rest, as it is commonly protracted in the stiff shoulder. There may be obvious tightness of the trapezius muscle. Palpation for swelling or adhesions in the subdeltoid, subcoracoid, and subacromial bursa should be performed. The pectoral minor tendon can be palpated for stiffness and rigidity. The passive and active range of motion (ROM) should be well documented. The functional ability of the rotator cuff musculature should similarly be noted in allowable range. Each muscle should be tested as much as possible. The deltoid can similarly be tested.

7.1.4 Imaging

Plain radiographs should include standard posteroanterior (PA), scapular Y, and axillary radiographs. These should be evaluated for position of the components and for the presence of any residual spurs that may be restricting motion. Additional studies may include ultrasound of the rotator cuff and bursa to look for areas of tearing or inflammation, computed tomography (CT) arthrogram with special technique to minimize distortion from the prosthesis, including 3D-imaging, and magnetic resonance imaging (MRI) scanning.

7.1.5 Management

7.1.5.1 Initial Management

The stiff, replaced shoulder may have many etiologies. Once the shoulder has been fully evaluated, treatment should proceed in a cautious manner [3, 5, 9, 13–15, 17, 19]. The initial management may simply include a trial of steroids (oral and injected) and a change from manual therapy to aquatic therapy. An evaluation of the therapy protocol being utilized at home or in physical therapy (PT) may allow important adjustments that improve the results.

7.1.5.2 Manipulation

Although a standard treatment for the primary stiff shoulder has risks of complications, especially periprosthetic fracture, may outweigh the benefit in TSA patients. If performed, the classic Nevaiser technique of initial abduction while keeping the "forcing" hand above the elbow, followed by flexion with the same hand placement, then gentle external and internal rotation should be used. In no case should the hand or lower arm be utilized to obtain motion as the risk of humeral fracture is too great.

7.1.5.3 Arthroscopic Release

The advantages of arthroscopic release are obvious. Minimal skin incision and the lack of rotator cuff takedown minimize perioperative morbidity. The procedure can be done on an outpatient basis, with decreased cost. Deep cultures and tissue samples are easily obtained, and the prosthesis itself can be assessed for loosening. However, there may be inherent difficulty in accessing the shoulder initially. One should also be careful to differentiate scar and rotator cuff tissue and avoid inadvertently removing rotator cuff or deltoid tendon.

The procedure may be performed in either lateral decubitus or beach chair position. Examination under anesthesia should document exactly the range of motion (ROM). The arthroscope is initially inserted through a posterior portal. One may have to direct the initial small blunt trocar slightly superiorly to access the joint. Initial evaluation may be confusing due to reflection of the soft tissue by the humeral head (Fig. 7.1). Initial evaluation should include an evaluation of the integrity of the subscapularis muscle. A rotator interval portal is then established. A side effect type cautery device can be inserted and the interval released to the coracoid base

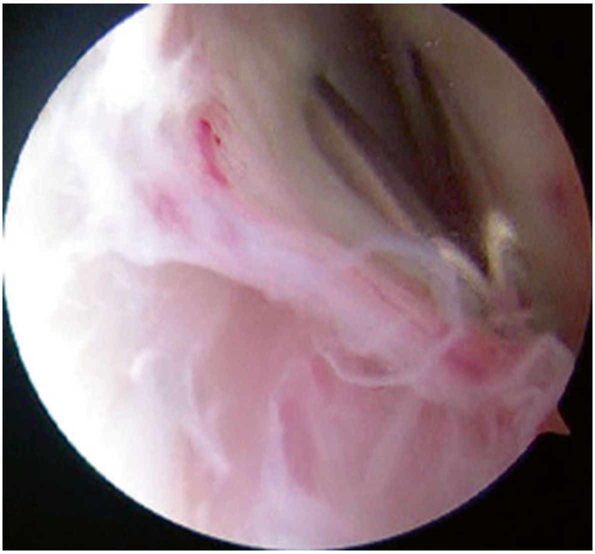

Fig. 7.1 Initial arthroscopic evaluation may be confusing due to reflection of the soft tissue by the humeral head

Fig. 7.2 A side effect type cautery device can be inserted and the interval released to the coracoid base

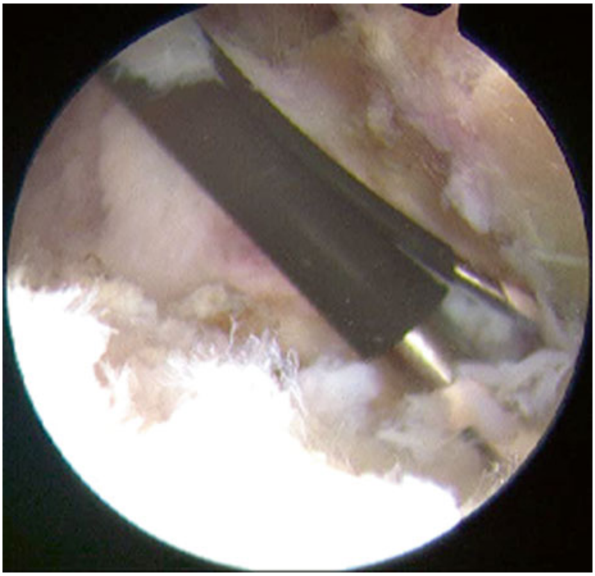

Fig. 7.3 The cautery is used just outside the normal (for humeral head replacement – HHR) or prosthetic (for TSA) glenoid to excise the anterior capsule and labrum down to the 4 o'clock position

(Fig. 7.2). The cautery device is used just outside the normal (for humeral head replacement – HHR) or prosthetic (for TSA) glenoid to excise the anterior capsule and labrum down to the 4 o'clock position (Fig. 7.3). This dissection always stays right along the bone of the glenoid neck to avoid damage to the subscapularis muscle and the axillary nerve. The scar and capsule must be released quite a bit medially along the glenoid neck to insure the subscapularis muscle is free to track laterally.

The superior capsule beneath the supraspinatus is similarly released, but the dissection is limited to stay within 1 cm of the joint to avoid the suprascapular nerve (Fig. 7.4).

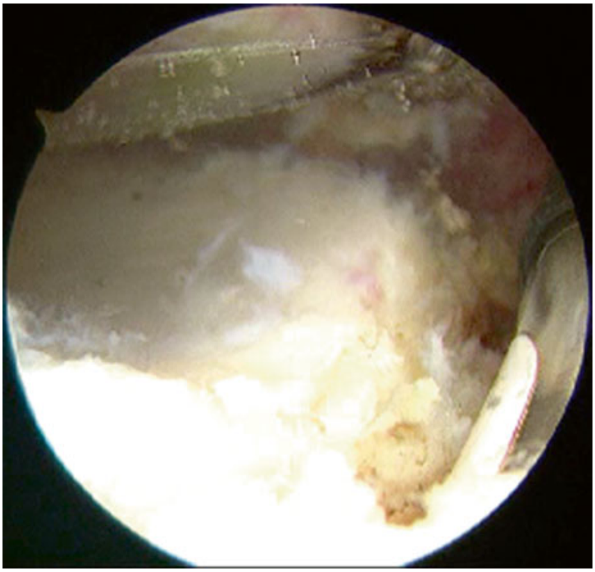

Fig. 7.4 The superior capsule beneath the supraspinatus is similarly released, but the dissection is limited to stay within 1 cm of the joint to avoid the suprascapular nerve. The humeral prosthesis is at top, the prosthetic glenoid at bottom, and the cautery to the right going over the superior glenoid neck

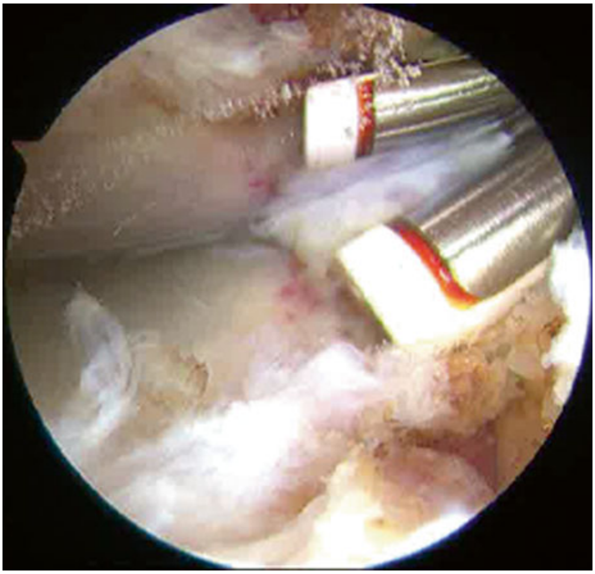

Fig. 7.5 The posterior capsule is released with the cautery staying right on the bone of the glenoid neck down to the 7 o'clock position posteriorly

The viewing portal is then transferred to the anterior portal and the cautery placed in the posterior portal. The release is continued beneath the infraspinatus superiorly without attempting to find the scapular spine. The posterior capsule is released with the cautery staying right on the bone of the glenoid down to the 7 o'clock position posteriorly (Fig. 7.5).

A posterior, inferior portal is then created. The arthroscope may be left anteriorly or placed in the standard posterior portal. The inferior capsule and labrum are carefully dissected off the glenoid, again using the cautery (Fig. 7.6). In this 5–7 o'clock

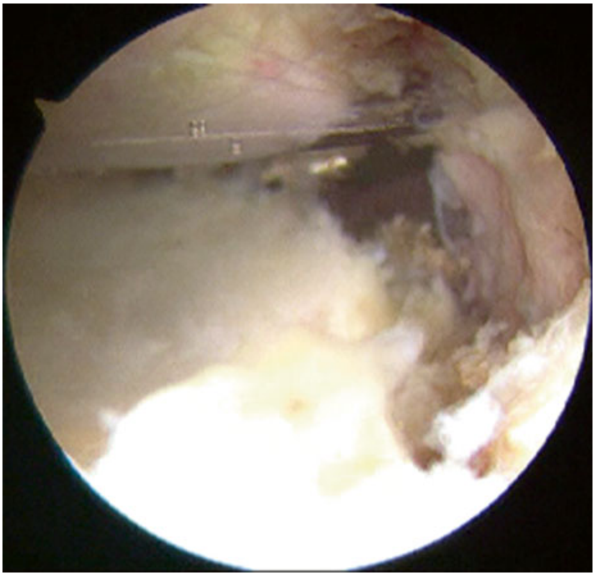

Fig. 7.6 A posterior, inferior portal is then created. The arthroscope may be left anteriorly or placed in the standard posterior portal. The inferior capsule and labrum are carefully dissected off the glenoid, again using the cautery. It is important to recognize the proximity of the axillary nerve during this part of the procedure

position, the axillary nerve may be quite close, or even scarred to the capsule, so caution is essential. If there is any question, a small posterior incision with dissection down to the quadrangular space will allow direct inspection of the axillary nerve and retraction of it away from the capsule.

Once the capsule has been completely and circumferentially released from the glenoid, the rest of the rotator interval tissue may be excised. At this point, an evaluation of the glenoid for possible loosening can be performed (Fig. 7.7). There should be no motion between the prosthesis and the bone of the glenoid. The arm is then removed from the holding device and motion rechecked.

The arthroscope is inserted into the subacromial bursa. A lateral portal is established and initial dissection is in the subdeltoid bursa in order to avoid injury to the rotator cuff (Fig. 7.8). There are usually considerable adhesions in this area. Excision of all of these adhesions is essential, so one must debride distally while staying close to the humerus and always keeping the shaver facing away from the deltoid muscle to avoid injury to the axillary nerve. In some cases, a blunt dissector placed via the anterior portal may be used to hold the muscle and nerve up and away from the shaver.

Once the subdeltoid debridement has been completed, the dissection can be continued proximally to find the acromion. The cautery device is used on the undersurface of the acromion so that the rotator cuff tissue can be preserved. The arthroscope is transferred to the lateral portal and using anterior and posterior instrument portals, all soft tissue is dissected free from the acromion, the coracoacromial (CA) ligament, the coracoid and subcoracoid bursa and the conjoined tendon, the acromioclavicular (AC) joint, and the scapular spine. Posteriorly, adhesions between the deltoid and the rotator cuff should be excised.

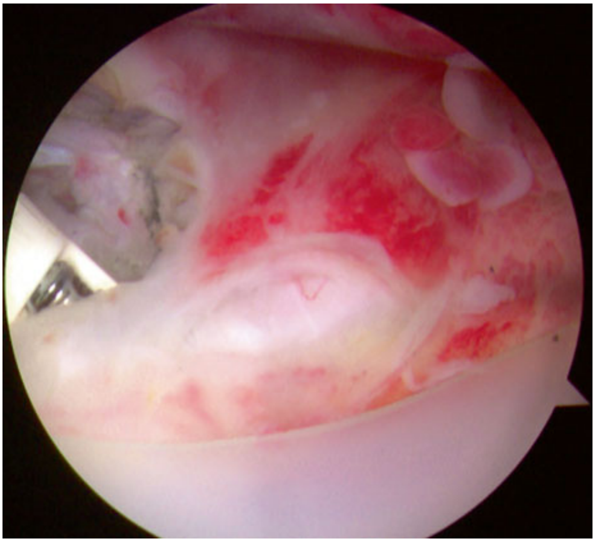

Fig. 7.7 Once the capsule has been completely and circumferentially released from the glenoid, the rest of the rotator interval tissue may be excised. At this point, an evaluation of the glenoid for possible loosening can be performed. The glenoid is the white plastic on the inferior aspect of the figure

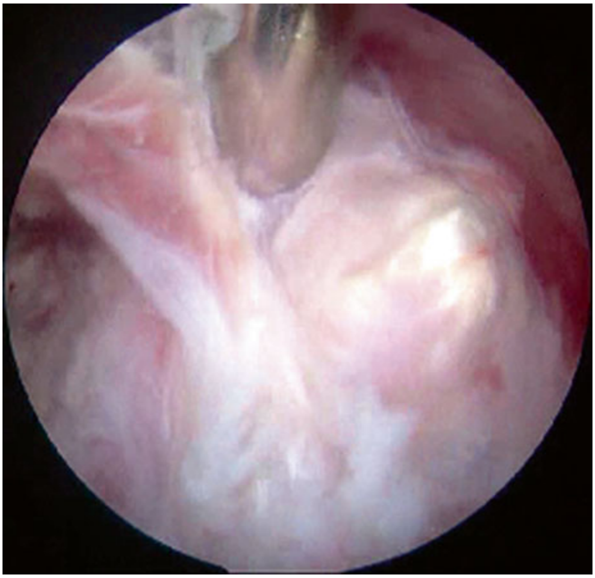

Fig. 7.8 The arthroscope is inserted into the subacromial bursa. A lateral portal is established and initial dissection is in the subdeltoid bursa in order to avoid injury to the rotator cuff

Motion is carefully reassessed and should be normal. In some cases, a further dissection of the soft tissue around the subscapularis and rotator interval may be necessary to achieve full motion. We usually document the motion with the arthroscope to give to the patient (Fig. 7.9a–c). Fluoroscopy may be used in the operating room (OR) or radiographs may be obtained either in the OR or recovery rooms to ensure no fractures or instability have occurred.

Fig. 7.9 (**a**) Full abduction is achieved with no stress on the right shoulder post arthroscopic release. (**b**) Full external rotation is achieved in 90° abduction with no stress on the shoulder post arthroscopic release. (**c**) Full internal rotation is achieved in 90° abduction with no stress on the shoulder post arthroscopic release

7.1.5.4 Open Release

The patient is placed in the same position as for TSA. The previous deltopectoral approach is utilized. The coracoid is identified and the subscapular muscle evaluated. Subcoracoid, subdeltoid, and subacromial adhesions are removed. The axillary nerve is identified and carefully protected, and if necessary dissected from the inferior capsule. The rotator interval is completely released and excised.

7.1.5.5 Postoperative Course

Radiographs should be taken in the recovery room to make sure no inadvertent fractures or dislocation have occurred. The patient is usually admitted overnight for continuous passive motion (CPM) and therapy. Outpatient PT for maintenance of motion gained in surgery is continued for at least 4 weeks.

7.1.6 Results

There is little information on results of post arthroplasty release. However, in writing this chapter, the senior author had the opportunity to review 12 surgical patients referred for noninfected post arthroplasty stiffness; 6 patients had sustained a proximal humerus fracture treated with humeral head replacement, 2 patients had simultaneous surface replacement and rotator cuff repair, and 4 patients were stiff total shoulder arthroplasty patients. All 12 patients had failed extensive nonoperative management including injections and therapy; 4 patients also failed manipulation under anesthesia. The average preoperative visual analogue scale (VAS) for pain was 8 (range 5–10) and the average preoperative motion was flexion of 100°, abduction of 80°, external rotation of 0°, and internal rotation of 0°. All 12 patients underwent arthroscopic release by the described technique; 5 patients regained completely normal range of motion, 4 patients 90 % of normal, one patient 75 %, and in 2 patients (one HHR for fracture, 1 SRA with RCR) motion gains were relatively modest. The VAS pain scale decreased to an average of 3 (range 0–10) with one patient remaining at 10. Eleven of the 12 felt surgery was beneficial.

7.1.7 Conclusions

Stiffness post total shoulder arthroplasty is a rarely reported complication with an unknown incidence. Open or arthroscopic release may be indicated to improve the restricted motion and will allow assessment of the prosthetic stability and help rule out infection by providing deep culture and tissue biopsy. Limited evidence exists to evaluate the success of this treatment.

7.2 The Stiff Shoulder Post SLAP Repair

7.2.1 Introduction

The superior labrum has been an area of interest since the advent of arthroscopy. The initial report of superior labrum anterior to posterior (SLAP) tears by Snyder et al. [20] in 1990 that followed on the report by Andrews et al. [1] in 1985 on tears at the base of the biceps has focused quite a lot of interest in the subsequent years on this area of the shoulder. Both Andrews and Snyder documented a very low incidence of significant tears, and both managed their series with debridement only.

In the ensuing years, reports of repair became more common. Initial repair techniques were limited by available equipment to staples [21] and transglenoid sutures [8]. The development of suture anchor and suture passing techniques made this area much more accessible to repair. Unfortunately, initial reports of success with labral repair and the development of advanced imaging of this area has led to an explosion in the number of SLAP repairs being performed. The variable anatomy of this area makes it quite difficult to determine on imaging and exam when a superior labrum actually is the source of the problem and needs to be repaired. Repair of a normal superior labrum may result in overtightening of the shoulder, with subsequent motion loss. Additionally, there are age-related changes that occur in the superior labrum and the base of the biceps that may be best managed nonoperatively or with biceps tenotomy/tenodesis. This chapter attempts to determine the incidence and appropriate treatment of the stiff shoulder post SLAP repair.

7.2.2 Anatomy

The superior labrum extends from approximately the 10 o'clock position posterior–superior to the 1–2 o'clock position anterior superiorly. It includes attachment of the biceps tendon, as it progresses on to the supraglenoid tubercle and the attachments of the superior and sometimes middle glenohumeral ligaments. The stability of the attachment is quite variable; while some patients have a fairly tight attachment to the superior glenoid, others have more variability, with a large cleft beneath the labrum. In most situations, the superior labrum is thought to be somewhat mobile, with the superior labrum "rolling" medially with overhead movements.

7.2.3 Pathology of the Stiff Shoulder Post SLAP Repair

In these patients, there are several areas noted to be a problem [11]. The most common pathology noted in the failed SLAP repair was stiffness of the entire capsule and rotator interval [2, 4, 6, 7, 10–12, 16, 18]. In a similar study of military age population, Provencher et al. had similar findings of stiffness and pain as the main cause of failure of the procedure rather than recurrent instability complaints [16]. Almost all of the patients in both series had developed additional biceps pathology, chondromalacia, contracture of the rotator interval and a tight capsule [4, 11, 12, 16, 18]. In the Katz study [11], there were multiple cases of severe chondromalacia from anchors and sutures, with at least one patient requiring total shoulder arthroplasty.

7.2.4 Imaging

Radiographs should usually be performed to assess the status of the glenohumeral joint. More advanced imaging may be quite helpful, and in these painful postsurgical patients an arthrogram followed by either MRI or CT scan will provide the most helpful information.

7.2.5 Treatment

7.2.5.1 Nonoperative Treatment

The hallmark of management remains physical therapy. Despite the potential risks to articular cartilage, these patients may respond to injections, oral steroids, and a combination of water therapy and manual stretching. In the Katz study [11], 28 % of patients responded to nonoperative treatment measures.

7.2.5.2 Surgery

Surgery may be indicated when the nonoperative protocol is unsuccessful. In patients with post SLAP repair shoulder stiffness case, there are three basic principles to follow: remove the entire old fixation, tenodese the biceps away from the glenohumeral joint, and perform a complete capsule and rotator interval release.

7.2.5.3 Surgical Technique

The patient may be positioned in either the lateral decubitus or beach chair position. An exam under anesthesia is performed to assess motion and laxity. The arthroscope is then placed into the glenohumeral joint to assess the situation. It is certainly reasonable to perform a manipulation prior to insertion of the cannula, but we prefer to assess the pathology before any manipulation is performed. In most cases, the pathology is fairly consistent; the superior labrum is stable but often ragged (Fig. 7.10). There is usually chondromalacia associated with the sutures (Fig. 7.11). The biceps tendon will show wear/tearing at both the attachment to the superior labrum and as it enters the groove (Fig. 7.12a, b). There is often a significant contracture of the superior glenohumeral ligament and tight scarring in the rotator interval (Fig. 7.13).

The procedure involves initially removing all of the old anchors and sutures. Microfracture and debridement of the anchor holes is also performed. The biceps is then assessed and transected at the base. The rotator interval is then released, and the posterior inferior glenohumeral ligament (PIGHL) is also released. Additional capsular release is performed as needed.

7.2.6 Results

Katz et al. showed significant return of motion, with the best results in patients without additional chondromalacia [11]. Provencher et al. showed improvement in motion and decrease in pain in a large series of failed SLAP repairs managed with tenodesis [16].

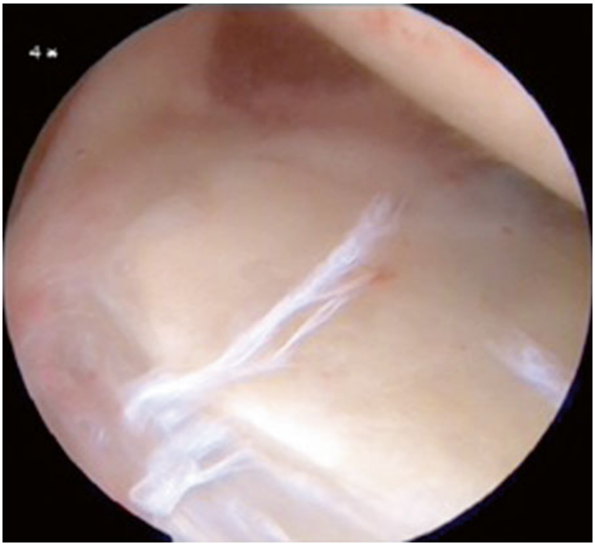

Fig. 7.10 In most cases, the pathology is fairly consistent; the superior labrum is stable but often ragged

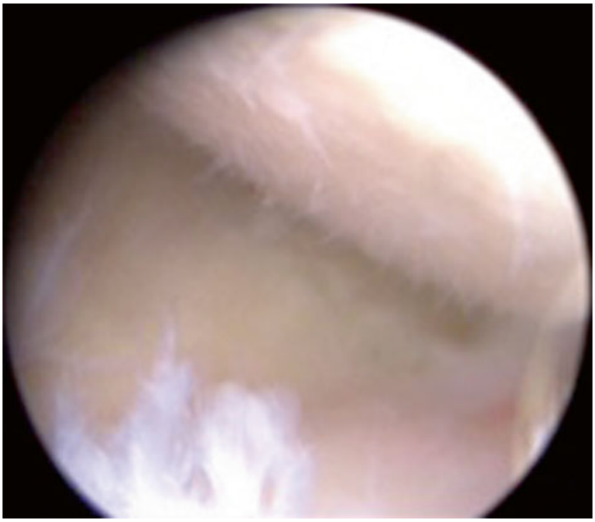

Fig. 7.11 There is usually chondromalacia associated with the sutures

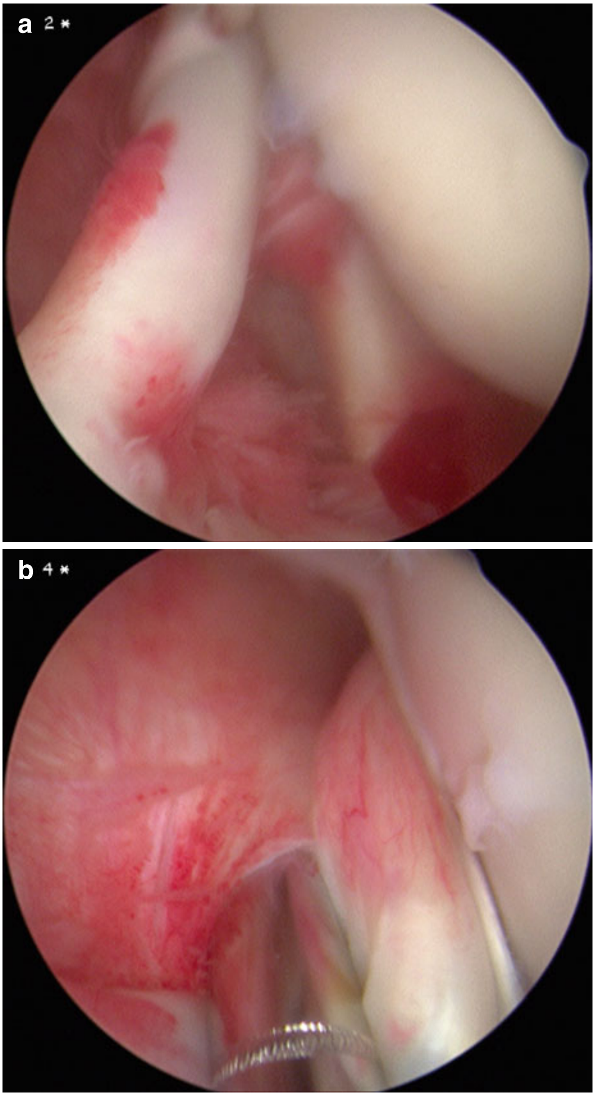

Fig. 7.12 (**a**, **b**) The biceps tendon will show wear/tearing at both the attachment to the superior labrum and as it enters the groove. (**a**) Biceps damage post SLAP. Biceps inflammation at groove: (**b**)

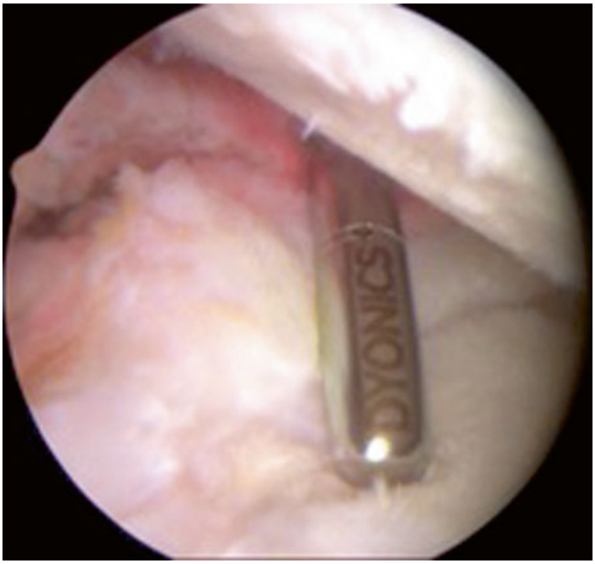

Fig. 7.13 There is often a significant contracture of the superior glenohumeral ligament and tight scarring in the rotator interval. Contracted SGHL post failed SLAP repair

Conclusions

Shoulder stiffness post SLAP repair may be more of an iatrogenic problem due to confusion over indications rather than a true postoperative capsulitis. This problem may be best addressed by improving education as to when a SLAP repair should be performed.

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Other Causes of Shoulder Stiffness

8

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and Augustus D. Mazzocca

8.1 Neurological Causes

The neurological system controls the activation and coordination of the muscles, the motors that drive the motion about the shoulder. Pathology affecting the neurological system can result in shoulder stiffness. The neurological system is divided into upper and lower motor neurones. The upper motor neurons comprise the cerebrum and spinal cord. These neurons are responsible for the planning and coordination of movement. The lower motor neurones are the peripheral nerves that arise from the spinal cord roots. These are the wires that deliver the signal from the upper motor neurons to the target muscle fibers.

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8.1.1 Etiology

The mechanisms of upper motor neuron damage may be cardiovascular (cerebral palsy, cerebrovascular accident), traumatic (brain or spinal cord injury), or progressively degenerative conditions (multiple sclerosis, amyelotrophic lateral sclerosis).

Lower motor neuron damage commonly results from external trauma (crush, stretch, division, etc.), but other causes such as inflammatory (brachial neuritis, Parsonage-Turner syndrome) or compressive mechanisms (paralabral cyst, suprascapular notch stenosis) may be responsible.

Differing patterns of dysfunction will occur depending on the level of the neurological lesion.

8.1.2 Upper Motor Neuron

8.1.2.1 Spasticity

Definition

Spasticity is a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes ("muscle tone") with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex, as one component of the upper motor neuron syndrome [1].

Pathology

Spasticity occurs because the injury to the upper motor neurons affects their modulating function on lower motor neurons. The exact pathoanatomical mechanisms by which this occurs are still not well understood [1–3]. Several potential mechanisms have been proposed and it is likely that the mechanisms differ depending on the location of the injured neurons (e.g., cerebral cortex compared to spinal cord tracts).

The location of the neuronal injury dictates the region of the body that is affected. However, even in a hemiplegic patient not all muscles of the affected limbs are equally involved. Spasticity in the upper limb typically affects antigravity muscles (e.g., biceps brachii) and around the shoulder it affects pectoralis major, latissimus dorsi, subscapularis, and teres major. The classic posture of upper limb hemiplegia with spasticity is adduction of the arm, internal rotation of the shoulder, and flexion of the elbow, wrist, and fingers [4]. The deformity arises from an imbalance between the strongly contracting muscle affected by the spasticity and the remaining muscles, which are weak.

8.1.2.2 Secondary Changes

Contracture

Contracture develops because of changes within the affected muscle tendon unit due to the spasticity and immobilization [2].

Immobilization of connective tissue allows cross-linking of collagen fibers to occur, resulting in shortening and loss of elasticity. This reduces the compliance of the connective tissue both within the muscle-tendon unit as well as in the capsule-ligamentous surrounding the adjacent joints [1].

Prolonged spasticity results in shortening of sarcomeres as well as a loss of sarcomeres at the ends of the muscle fibrils, which is in excess of that seen with immobilization alone [2].

Joint Changes

Prolonged muscle shortening places joints at the extremes of their normal range of motion. This can result in persistent subluxation leading to pain and joint deformation [3, 4]. The classic adducted and internally rotated position of the spasticity affected shoulder results in posterior wear of the glenoid.

8.1.2.3 Management

Clinical Presentation

As described above, the distribution of the affected muscles depends on the location and extent of the neurological injury. The patient usually presents for treatment because of functional and hygiene difficulties due to the contracture.

Spasticity due to cerebral injury (e.g., stroke) can develop pain as well as contracture. This pain can be due to a combination of factors such as complex regional pain syndrome, glenohumeral subluxation secondary to posture, and possibly tendinopathies [4].

Spasticity must be clearly distinguished from weakness, as the latter is unlikely to be improved by antispastic treatments and may even be made worse.

Treatment

Treatment should be reserved for patients with functional disability or hygiene difficulties due to their contracture, or those with pain [5].

Nonoperative

Physical therapy addresses the secondary changes resulting from spasticity. Passive stretching reduces muscle tone [5] and maintains the compliance of the muscle and periarticular structures. Muscle strengthening exercises can also be performed to strengthen the spastic and synergistic muscle groups [3].

*Orthoses*s can be used to maintain stretch on a contracted joint.

Oral medication has been used including baclofen, tizanidine, benzodiazepines, clonidine, and gabapentin. However, a meta-analysis of oral antispastic medications in nonprogressive neurological diseases reported their efficacy was, at best, weak, and the evidence to support their use was marginal [6].

Botox (botulinum toxin A) is a neuromuscular blocker that inhibits the release of acetylcholine from the presynaptic membrane. It is injected locally into the affected muscle and the effect lasts 2–6 months [3, 5]. Paralysis of the muscle allows greater passive movement for physical therapy and reduces pain. It can also be used as a "trial tenotomy" to test the outcome of surgery on the patient. In the spastic shoulder, botox injection into the subscapularis and/or pectoralis major has been reported to improve pain but not range of motion [4, 7].

Alcohol and phenol nerve injections have also been used to paralyze spastic muscles. In contrast to botox, the effect of these agents on the nerve is permanent.

Extracorporeal shockwave therapy (ESWT) delivers electrical current to the affected area. The exact mechanism by which it reduces spasticity is unclear, but it may stimulate neurons that reduce excitability of the spastic neuromuscular circuits or stimulate antagonistic muscles [3, 7].

Operative

Options for the surgical management of spasticity include nerve ablation, capsular releases, and muscle release or lengthening procedures.

Nerve ablation (e.g., selective dorsal rhizotomy) aims to reduce the spasticity by sectioning either the motor or sensory arm of the reflex arc. It has been reported that at least 25 % of the motor fibers must be preserved to prevent unacceptable weakness [3].

Releases of the contracted capsular tissues and removal of muscular adhesions is only of benefit if the muscle spasticity has been addressed. Lengthening of the muscle tendon units allows an increase in the arc of motion of the joint but the risk of both procedures is creating unacceptable secondary weakness.

For patients with a spastic internal rotation contracture, a subscapular tenotomy can be performed. The anterior capsule must be preserved to prevent instability.

8.1.3 Lower Motor Neuron

8.1.3.1 Pathology

Unlike the upper motor neuron lesions that may produce spasticity, lower motor neuron injury results in muscle wasting, loss of reflexes, and *flaccid paralysis*.

The flaccid paralysis and areflexia are apparent almost immediately. Loss of innervation will result in fibrillation of the denervated muscle fibers. With prolonged denervation, degradation of the motor end plates will occur with associated muscle atrophy and fatty infiltration. Once the end plates have been lost re-innervation of the muscle fibers cannot occur [8].

Muscle Imbalance

For patients with a complete plexus injury this will result in a flail extremity, but a partial injury will result in *muscle imbalance*. The posturing of the upper limb following an upper truck brachial plexus injury (C5, 6) is similar to that seen in spasticity (internal rotation, adduction). However, the key difference is that the muscle imbalance in spasticity is between a spastic (over active) muscle and a weak muscle, whereas in lower motor neuron injury the imbalance is between a weak/paralyzed muscle and a normal one.

8.1.3.2 Secondary Changes

As with spasticity, secondary changes to the capsule, joint surface, and articular congruity can occur in chronic cases of lower motor neuron pathology [9]. The classic example is glenoid retroversion as a result of chronic brachial plexus birth injury.

8.1.3.3 Management

Clinical Presentation

The key issues are the location of the lesion in the lower motor neuron system and the time elapsed since the lesion occurred, as these dictate the management options.

The absence of neurological stimulation will eventually result in the irreversible loss of the motor end plates, where the nerve fibers synapse with the muscle sarcomere.

Treatment

Physical Therapy and Orthoses

The role of stretching, strengthening, and splinting in lower motor neurone conditions is similar to that in upper motor neurone lesions: to address the secondary contractures. In the case of a lower motor neuron lesion that has some chance of recovery, physical therapy is essential to prevent joint contracture developing whilst the nerve and therefore muscle function is recovering.

Surgical

Surgical options can be grouped into nerve procedures and salvage procedures.

Nerve Procedures

These include *decompression or release* of an external structure compressing the nerve, such as fascia or scar tissue. In the case of complete nerve division (Sunderland type 4 or 5 [10]), the nerve can be *repaired* or a cable *graft* may be considered. The potential success of this depends on the distance from the site of the repair to the motor end plate, the time left before the end plate will be degenerate, and the age of the patient [8].

Finally, in some cases *nerve transfer* may be performed. In this procedure, fascicles from a separate motor nerve are separated out and grafted on to the denervated muscle. An example of this is the Oberlin transfer of ulnar nerve fascicles to the musculocutaneous nerve. This transfer has been used to restore elbow flexion following brachial plexus injury [11, 12].

Capsular Releases

If nonoperative measures fail to restore the compliance of the capsule, then soft-tissue releases may be required.

The most common region of the shoulder capsule to be contracted is the anterior portion. Capsular release frequently must be accompanied by lengthening of the subscapularis tendon to achieve adequate external rotation.

Tendon Transfers

Tendon transfers can be employed to address the muscle imbalance created by a partial plexus injury.

In pediatric brachial plexus injury, the latissimus dorsi tendon can be transferred to the greater tuberosity to provide both abduction and external rotation [9].

In adult patients the trapezius, levator scapulae, and serratus anterior have all been described as donor tendons to restore active shoulder motion [13].

Salvage Procedures
Tendon transfers are unlikely to be successful in cases with advanced glenohumeral deformity, as they cannot address the loss of passive movement due to the misshapen articular surfaces.

In this situation, a *derotational osteotomy* can improve the functional position of the hand. By doing so the humeral head is left in a permanently subluxed or dislocated position, thus this procedure is generally reserved for patients with no remodeling potential left.

8.2 Arthropathy

These are conditions that affect the articular cartilage. The cartilage changes may be due to an inflammatory condition affecting the joint (e.g., rheumatoid arthritis, seronegative arthropathy, crystal arthropathy) or degenerative processes (osteoarthritis).

8.2.1 Osteoarthritis

8.2.1.1 Definition

Osteoarthritis (OA) is a condition characterized by focal and progressive loss of the articular cartilage, thickening of the subchondral bone, and the formation of marginal osteophytes.

8.2.1.2 Etiology

Osteoarthritis is classified into primary (idiopathic) and secondary groups. Secondary OA can have multiple etiologies including posttraumatic, postcongenital abnormality, and metabolic, endocrine, and neuropathic conditions. The possible role of chronic, low-grade infection with *Proprionibacterium acnes* in the etiology of shoulder OA has also been reported recently [14].

8.2.1.3 Pathology

Arthritis

The primary pathological process in shoulder OA is thinning of the articular cartilage. When the full thickness of the articular cartilage has been lost, the exposed subchondral bone is also subject to erosion. Walch et al. [15] described five characteristic glenoid wear patterns in shoulder arthritis (Fig. 8.1).

The loss of the normal lubricating and load-bearing properties of the cartilage produces abnormal loads on the subchondral bone plate. The bone responds by remodeling, producing the sclerotic appearance on radiographs.

Synovial fluid can pass through microfractures of the subchondral bone plate, creating subchondral cysts (Fig. 8.2). Osteophytes form at the articular margins. Late-stage OA can develop osteonecrosis, leading to collapse of the humeral head.

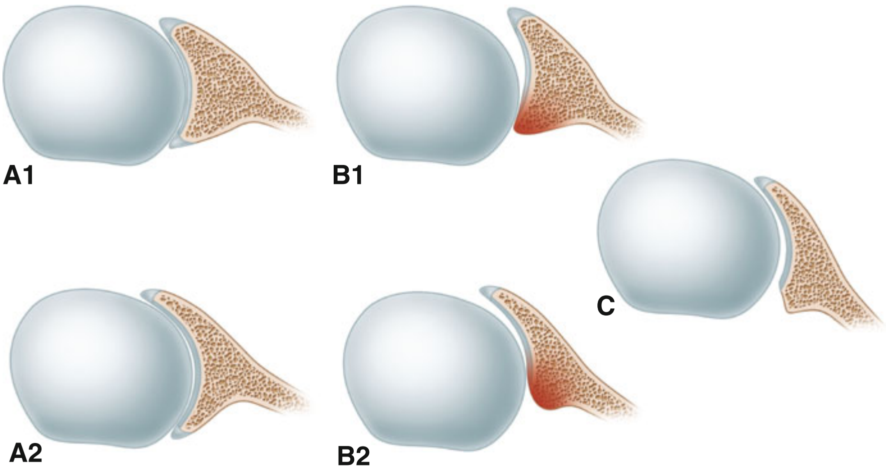

Fig. 8.1 Walch classification of glenoid morphology. *A1* Centered humeral head, *A2* central glenoid erosion, *B1* posterior subluxation of humeral head, *B2* posterior glenoid erosion, *C* dysplastic glenoid (>25° retroversion) (From Walch et al. [15])

A block to movement of the glenohumeral joint can arise from the increased retroversion of the joint (glenoid wear), flattening and incongruity of the humeral head (Fig. 8.3), and impingement against osteophytes.

Secondary Contracture

Wear of the posterior aspect of the glenoid sets up an environment for progressive contracture of the anterior soft tissues. The posterior capsule is stretched over the posterior osteophytes and by the internally rotated and posteriorly translated position of the humeral head [16]. The stretched capsule allows further posterior subluxation and internal rotation.

On the anterior side, the reverse occurs: the lack of external rotation of the humeral head decreases the resting tension in the anterior capsule and contracture develops in the anterior ligamentous structures (capsule and coracoclavicular ligament) [17]. The subscapularis muscle displays both shortening of the muscle tendon unit and adhesions to the surrounding structures [18]. Three distinct zones of the subscapularis tendon have been described: the superior tubular tendon (STT), the middle tendon, and, inferiorly, the muscle fibers that insert directly onto the humerus [17].

8.2.2 Inflammatory Arthropathy

8.2.2.1 Etiology

A large number of conditions may cause inflammation of the synovial lining of the joint capsule. These may be autoimmune syndromes such as rheumatoid arthritis and the seronegative arthropathies or crystal arthropathy syndromes such as gout.

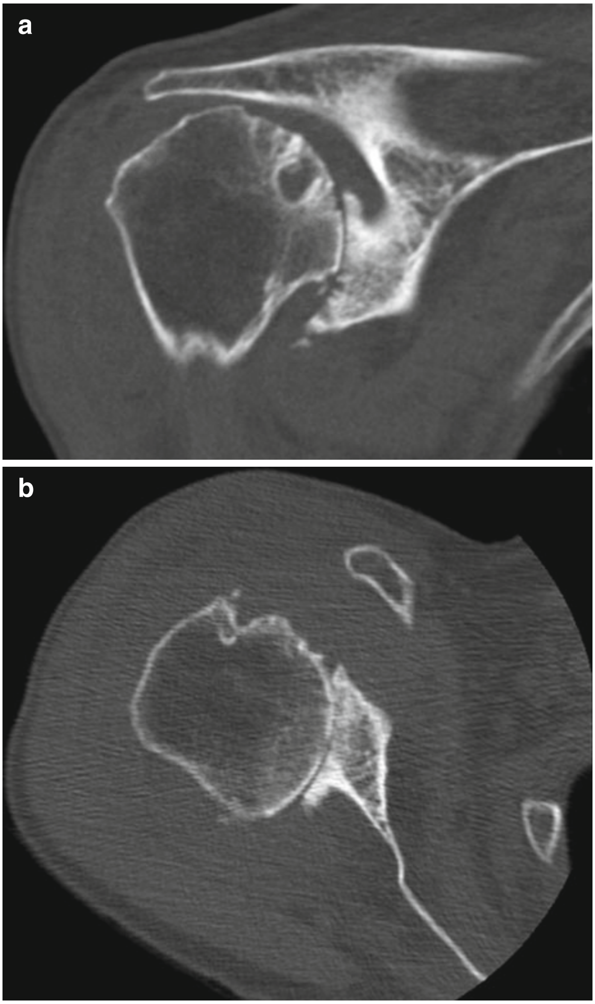

Fig. 8.2 Computed tomography images of rotator cuff tear arthropathy of the shoulder. Multiple factors contributing to joint stiffness are present: complete loss of joint space (**a**, **b**), collapse of subchondral bone (**a**), osteophyte formation (**b**), and subluxation of the humeral head in both superior (**a**) and posterior (**b**) directions (Copyright GI Bain. Used with permission)

8.2.2.2 Pathology

The specific effects vary according to the different etiologies, but in all cases the chronic inflammatory processes result in cytokine release and macrophage activation. This results in destruction of both cartilage and soft tissue.

In rheumatoid and seronegative inflammatory conditions, the changes are typically present throughout the entire joint, compared to the relatively focal nature of joint changes in osteoarthritis.

The disease process affects multiple joints. The presence of cervical spine instability due to inflammatory arthritis will limit the patient's ability to undergo surgery. Multiple other body systems may also be affected, including the lungs, cardiovascular system, and skin.

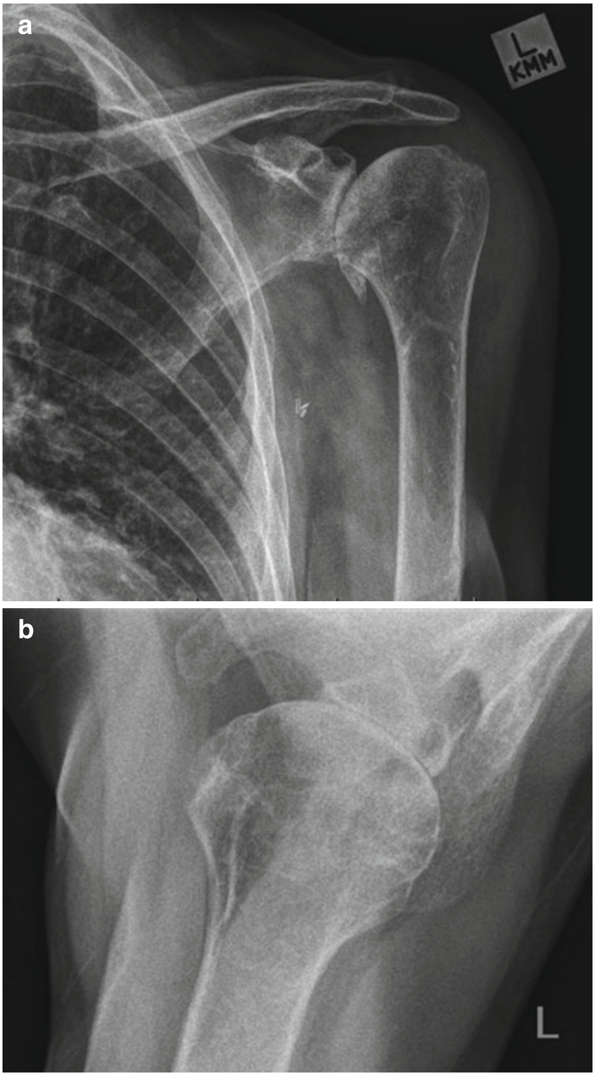

Fig. 8.3 Plain radiographs of osteoarthritic shoulder. Note multiple factors contributing to shoulder stiffness. (**a**) Osteophyte formation around inferior border of humeral articular surface. (**b**) Loss of joint space, erosion of posterior glenoid, posterior subluxation of humeral head on glenoid face (Copyright GI Bain. Used with permission)

8.2.2.3 Secondary Contracture

The damage to the capsular and surrounding tissues can affect the fulcrum function of the joint, resulting in reduced active and passive motion. Severe capsular attenuation and disruption of the rotator cuff tendons can result in loss of normal constraint, with subluxation of the humeral head on the glenoid. Persistent inflammation will not only destroy the articular cartilage but can also precipitate collapse of the subchondral bone, resulting in a loss of articular congruency.

8.2.3 Management

8.2.3.1 Clinical Presentation

The patient has pain with activity and potentially at rest. In inflammatory arthritis the pain is classically noticeable in the morning, whereas in osteoarthritis the pain becomes more of a problem as the day progresses. There is reduced active and passive glenohumeral range of motion in all movements.

In osteoarthritis, there is typically contracture of the anterior and inferior soft tissues resulting in a block to both abduction and external rotation. In severe cases, the patient may develop a fixed internal rotation contracture.

In inflammatory arthropathy, there may be other body systems affected and these must all be assessed when determining the patient's suitability for treatment. Skin lesions present an infection risk, especially if arthroplasty is planned. These may be a result of the inflammatory disease process (e.g., psoriasis) or a result of underlying joint deformity (foot ulcers in rheumatoid arthritis). Specific attention must also be paid to the presence of any cervical spine instability.

8.2.3.2 Investigation

Plain radiographs demonstrate the key findings of reduced joint space, osteophytes, subchondral sclerosis, and subchondral cysts. An axillary lateral view will demonstrate the glenoid version (Fig. 8.2). If there is any concern regarding the glenoid morphology or bone stock, a CT scan is required to further quantify this.

8.2.3.3 Treatment

Various *nonoperative* treatments have been described for shoulder OA including physical therapy, oral cartilage supplements, and injections of either steroid or viscosupplements. There is little published evidence on the effectiveness of these modalities in managing the established secondary soft-tissue contracture.

Medical management of inflammatory arthritis can prevent joint destruction, but cannot reverse changes that have already occurred.

Surgical options for glenohumeral arthritis include arthroscopic debridement with capsular release, open debridement procedures, arthroplasty procedures, and arthrodesis.

Arthroscopic surgery for arthritis involves debridement of unstable cartilage fragments, removal of loose bodies, and excision of impinging osteophytes. A capsular release can also be performed and in most cases of OA this would involve the anterior and inferior capsule. Any adhesions around the subscapularis muscle can also be released, and a partial release of the tendon can also be performed.

The majority of patients with symptomatic shoulder arthropathy require an *arthroplasty* procedure. Concerns regarding progressive lucent lines around glenoid components [19] have led some surgeons to favor hemiarthroplasty procedures, especially in younger patients. However, the results of hemiarthroplasty, with or without glenoid reaming [19, 20] or with soft-tissue interposition arthroplasty [21], are generally inferior to those of total shoulder arthroplasty (TSA).

Total arthroplasty prostheses are either an anatomic design or a reverse prosthesis. Anatomic TSA requires strict rebalancing of the rotator cuff and capsular tissues. This can be achieved with a combination of capsular release, posterior capsular plication, and alteration of the humeral version [22].

The subscapularis muscle can also be addressed by release of any surrounding adhesions and lengthening procedures. Lengthening can be achieved by release of the superior tubular tendon component [17] or repair of the tenotomized tendon to a lateral stump of capsule [16, 18].

Reverse TSA has traditionally been indicated for patients with rotator cuff insufficiency. The component design has greater containment than anatomic TSA, allowing a greater margin for soft-tissue release without compromising stability. This is also an advantage in inflammatory arthroplasty where the soft tissues may be compromised. The uncemented glenoid component of a reverse TSR allows combined bone grafting procedures to address excessive retroversion [1, 22, 23]. Conversely, uncemented glenoid components have shown poor results in anatomic TSA [1–3, 24] .

8.2.4 Outcomes

Arthroscopic procedures are potentially attractive for addressing capsular contracture, especially in the setting of lower-grade articular cartilage changes. However, literature on these techniques is limited to case series. A recent review [4, 25] could not find any high-level evidence to support the use of arthroscopic debridement in the management of shoulder OA.

Anatomic TSA has demonstrated good to excellent functional scores [2, 26] and 90–95 % survival at 10 years [2, 22]. A recent systematic review [1, 27] reported that complications occur in 23 % of unconstrained shoulder arthroplasty cases. However, 20 % of these complications were related to hemiarthroplasty procedures [3, 4, 27]. The most common complication in anatomic TSA is glenoid loosening, although this may be present on radiographs but clinically silent [4, 27–29]. Gonzalez et al. [5, 27] reported 29 % of patients with glenoid loosening required revision.

Failure to correct glenoid retroversion, or to rebalance and stabilize the soft tissues can result in higher rates of instability and prosthesis failure [5, 22]. An intact rotator cuff is also necessary for the normal functioning of an anatomic TSA and rotator cuff insufficiency is a common cause for instability of the prosthesis [3, 30].

Reverse TSA has been reported to have a high complication rate. A meta-analysis of 782 reverse TSA cases [6, 31] reported a 44 % incidence of postoperative problems and a 24 % complication rate. However, this study included revision procedures, which showed a much higher complication rate than primary procedures [3, 5, 31].

Specific problems with reverse TSA include scapular notching (potentially resulting in accelerated implant wear), instability, and acromial fracture.

Concerns regarding the longevity of the implants have led to recommendations that they are only performed in elderly, low-demand patients [4, 7, 32]. However, Reverse TSA has increasingly been used in younger and more active patients because of the other potential advantages including avoiding problems with rotator cuff insufficiency and the ability to address glenoid retroversion. The revision rate for Reverse TSA is similar to that of anatomic TSA in registry studies [3, 7, 33] and the outcomes of Reverse TSA in patients aged <60 years have been reported to be similar to those of older patients [3, 34].

8.3 Extra-articular Conditions

8.3.1 Heterotopic Ossification

8.3.1.1 Definition

Heterotopic ossification (HO) is the deposition of bone in extraskeletal tissue. The presence of the cellular, protein, and mineral components of bone in HO differentiates it from dystrophic calcification, which is deposition of mineral components only [8, 35].

HO adjacent to a joint usually does not directly involve the nearby articular surfaces. Thus, HO is a cause of joint stiffness in the setting of a normal articular surface. HO is most common around the hip, followed by the elbow. Intervention for HO of the shoulder is seldom required. Pansard et al. [9, 36] reported 539 cases of HO associated with neurological injury that required surgical intervention. Only 3.5 % of the procedures were for shoulder involvement. The authors of that study concluded that HO of the shoulder in this setting was “very rare, or perhaps rarely troublesome” [10, 36].

8.3.1.2 Etiology

HO has been linked to genetic, neurological, and traumatic (postsurgical and postinjury) etiologies. More than one may be present in a single patient.

Congenital

Hereditary HO is a rare genetic disease that has at least three distinct subtypes: Fibro ossificans progressiva (FOP), progressive osseous hyperplasia (POH), and Albright’s Osteodystrophy. Hereditary HO is a progressive disease that usually presents in the first decade of life; however adult-onset FOP has also been reported [8, 37].

Neurological

HO is a relatively common complication following injury to the central nervous system. HO has also been reported following a period of chronic sepsis and coma of 2-weeks duration [11, 12, 38]. The reported incidence is 11–73 % following traumatic brain injury (TBI) and 10–78 % following spinal cord injury (SCI) [9, 39]. The incidence is highest if the joint injury is concomitant to the neurological injury [13, 40].

Traumatic

The incidence of HO following open fractures sustained in combat has been reported to be 38 %, with the hip, elbow, and forearm most commonly requiring treatment [14, 41]. High injury severity scores and injuries to the shoulder, hip, and femur have been shown to be independent risk factors for HO formation in this setting [15, 41].

HO has also been reported to affect 1–3 % of burn patients [16, 40].

Postsurgical

The incidence of HO following shoulder arthroplasty has been reported to be between 14 and 54 % [17, 42–45]. Almost all cases were low grade and had minimal functional limitation. Tanner et al. [18, 42] reported that grade III lesions (appearance of complete bone bridge from humerus to glenoid on radiographs) had reduced abduction but no difference in rotation compared to lower grades and shoulders with no HO.

HO has been reported after less-invasive surgical procedures, including arthroscopy. Cases of HO following arthroscopy are rare and have involved the deltoid muscle and subacromial bursa [17, 46, 47].

8.3.1.3 Pathology

Heterotopic Ossification

The underlying pathological process that produces HO is poorly understood. Genetic studies of hereditary HO have implicated a receptor for bone morphogenic protein (BMP) type I [19, 37].

Neurological injury has been postulated to activate BMP and prostaglandin (PG) pathways. This induces mesenchymal stem cells in the periphery of the muscle to differentiate into osteoblasts [19, 20, 48]. It has also been that suggested that loss of joint proprioception due to neurological injury has a role in the pathogenesis of HO [21, 49].

Stimulation of mesenchymal stem cells by the local inflammatory response is also thought to be the underlying mechanism of HO due to trauma [22, 39].

The ectopic bone is a heterogeneous mixture of cortical and cancellous bone with distinct regions of fibrocartilage. Evidence of bone remodeling has been reported in lesions that have been present for as long as 3 years [17, 50].

Secondary Contracture

Contractures and musculotendinous adhesions can occur in the surrounding soft tissues and can compress surrounding neurovascular structures. As the HO mass slowly grows, a groove forms around these structures and may compress them. Nerve rupture secondary to the HO mass has not been reported.

Risk Factors

There are very little published data regarding the shoulder, but several risk factors for the development of HO about the elbow have been proposed. Patient factors include genetic predisposition, metabolic bone disease, and ankylosing spondylitis. Injury-related factors include severity of initial trauma and concomitant neurological injury. Treatment factors include timing of surgery, surgical approach, and hematoma formation [16, 18, 40]. A delay of ≥8 days from injury to surgery has been reported to have an odds ratio of developing post-traumatic elbow HO that is 12 times the odd ratio for a delay of <1 day. Immobilization >15 days has also been reported to have higher odds of HO compared to immobilization for <7 days [51].

Table 8.1 Kjaersgaard-Andersen classification of HO about TSA [43]

Grade	Description
0	No ossification
I	<50 % of the space[a]
II	>50% but <100 %
III	Ossifications roentgenographically bridging the space[a]

HO Heterotopic ossification, *TSA* total shoulder arthroplasty
[a]Space between lateral border of glenoid and the medial border of the humeral shaft and/or the inferior acromion

8.3.1.4 Classification

The classification of HO about total hip arthroplasty was described by Brooker in 1973 [52]. This comprised four classes, from islands of bone in the soft tissues to bone spurs that did not connect and finally to apparent anklylosis. Kjaersgaard-Andersen et al. [43] used a similar concept to classify HO about total shoulder arthroplasty (Table 8.1).

8.3.1.5 Clinical Presentation

History

The most common presentation of HO is loss of joint movement. This can cause difficulties with function and hygiene. Pain and/or swelling are uncommon in HO following shoulder arthroplasty [42], but may be a feature of HO following neurological injury [48].

Examination

The patient may show evidence of prior trauma such as scars (burns, wounds, or surgical) or deformity. The affected joint may be swollen in the case of neurological injury. However, the most common examination finding is a loss of both passive and active movement.

Investigation

Plain radiographs will reveal the HO lesion (Fig. 8.4). Proper orthogonal views may not be possible because of inability to position the limb appropriately due to the contracture.

CT scans with three-dimensional (3D) reconstructions are useful for defining the extent and morphology of the HO lesion if surgical treatment is being considered. The proximity of neurovascular structures can also be assessed [36].

Neurological disturbance due to HO is almost always due to compression and stretch caused by the mass effect of the lesion. Nerve rupture due to HO has not been reported. Nerve conduction and electromyography (EMG) studies are only required if there is clinical evidence of a preoperative axillary nerve injury [36].

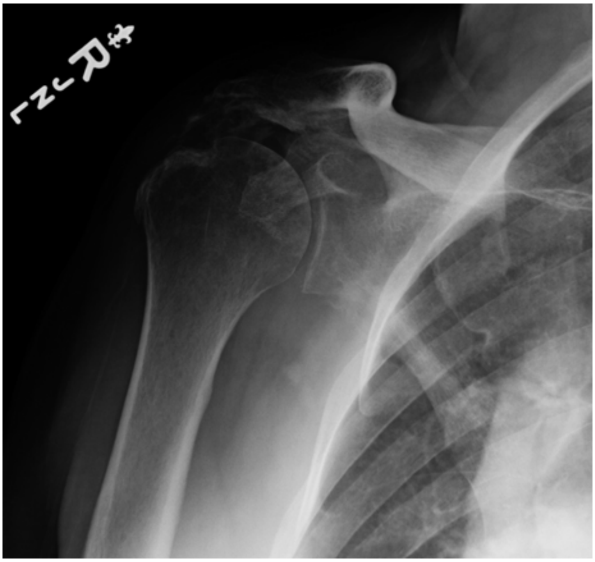

Fig. 8.4 Plain radiograph of the shoulder demonstrating heterotopic ossification between the humeral head and acromion (Image courtesy of Dr Felix Savoie III. Used with permission)

8.3.1.6 Treatment

There is comparatively little published literature on the management of HO affecting the shoulder. The majority of published treatment recommendations have been extrapolated from data published on the hip and, to a lesser extent, the elbow. In reality, HO of the shoulder is rarely a problem and treatment of any kind is seldom required.

Prophylaxis

Primary Prophylaxis

Primary prophylaxis as treatment may be given to patients with no evidence of HO but a high risk of developing the problem (e.g., open fractures).

Primary prophylaxis regimes have been shown to be effective in the hip [53] but not the upper limb. Radiotherapy has been associated with nonunion following fixation of humerus fractures [54], and NSAIDs have not shown to be of any benefit for preventing HO following primary shoulder arthroplasty [43, 55].

Secondary Prophylaxis

Secondary prophylaxis is treatment given to prevent recurrence of HO following surgical excision [56].

Most reports of surgical treatment for shoulder HO include a secondary prophylactic regime of either NSAIDs or radiotherapy, used individually or in combination. There is a paucity of literature on the effectiveness of secondary prophylaxis regimes in the upper limb. A systematic review of the role of RT in the management of elbow HO found only weak evidence to support its use [40]. Pansard et al. [36] reported that there is no evidence for the efficacy of radiotherapy in the prophylaxis of neurological HO.

Modalities

Radiotherapy (RT)

The postulated mechanism by which RT prevents HO formation is inhibition of the bone morphogenic protein signaling cascade. This cascade is involved in the differentiation of mesenchymal stem cells into osteoblasts [56].

Most dose regimes provide a single dose of 7Gy to the target area [40]. RT can produce gastrointestinal symptoms, fever, and carries the risk of causing a secondary malignancy [53].

Using a mathematical model, it has been reported that radiation prophylaxis in the shoulder region may result in an increased risk of cancer development in the skin and organs close to the irradiation field [57].

Nonsteroidal Anti-inflammatory Drugs (NSAIDs)

NSAIDs are thought to prevent HO formation by nonspecific inhibition of the prostaglandin mediated inflammatory response [56].

Approximately, 20 % of patients treated with NSAIDs prophylaxis will develop side effects [53]. The risk profile of NSAIDs is not insignificant. Gastrointestinal bleeding, renal impairment, and cardiac failure are all associated with NSAID usage.

Surgery

Closed manipulation under anesthesia has been reported for the management of this problem. The HO mass fractures to allow restoration of movement and the fragments eventually reabsorb [38]. However, the most common treatment for established HO is surgical excision.

Historically it has been the practice to wait for the HO to mature before excision is carried out. However, it has been shown that early surgery is not a risk factor for recurrence [36, 48]. In fact, a treatment delay of >12 months predisposes the patient to poorer postoperative outcomes [36, 48, 58].

The *indications* for surgical excision are loss of functional movement, problems with hygiene, neurovascular involvement, and pain [48]. In contrast, surgery for hereditary HO syndromes has been reported to be counterproductive as there is a high rate of recurrence [37].

The *surgical approach* is dictated by the position of the HO lesion. The most common location is inferomedial [36, 38] but any region of the shoulder may be affected. In certain situations, more than one incision may be required and the surgeon must be comfortable performing approaches to all regions of the shoulder.

Associated capsular contracture may require substantial release once the HO has been removed. Muscle-tendon units may be adherent to surrounding tissues or contracted as part of associated spasticity. Concurrent release or lengthening of spastic muscle-tendon units can create an iatrogenic muscle imbalance about the joint [36]. It has been recommended that such procedures be performed in a staged manner once the relative contributions of spasticity and adhesions have been delineated [36].

8.3.1.7 Outcomes

There are very little data on the outcomes of treatment of *posttraumatic or postsurgical* HO of the shoulder. Excision of posttraumatic elbow HO has been reported to produce similar results to excision for other etiologies, with a clinically significant improvement in the arc of rotation and functional scores generally rated as "Good" [58].

In appropriately selected patients with *neurological HO*, surgical excision can result in a clinically significant improvement in function. Pansard et al. [36] reported the outcomes of 19 cases of neurological shoulder HO that required surgical excision with 10 days of NSAIDs prophylaxis. Compared to preoperative measurements, the mean gain in arc of abduction was 60° and forward elevation was 69°. The external rotation arc improved by 13°. One case had significant intra-operative bleeding precluding complete resection of the HO and another developed shoulder stiffness. There were no other reported complications.

8.3.1.8 Senior Author's Approach

The senior author has worked in a Level 1 Trauma Center in which patients with head injuries, spinal cord injuries, burns, and complex trauma would often be managed. This provided a unique opportunity to experience some of the more complex cases of heterotrophic ossification of the upper limb.

Preoperative Assessment

The preoperative assessment of these patients is important. It is critical to identify and understand the associated comorbidities, especially those related to spasticity and burns. Preoperative 3D CT scans nicely demonstrate the detail of the heterotrophic bone, its exact distribution, and the associated structures that are likely to be involved. The surgeon needs to have an accurate understanding of the exact position of the major neurovascular structures with regard to the heterotrophic ossification. In those cases in which the major vessels may be involved, a 3D CT angiogram will identify the exact relationship of the vessels to the heterotrophic ossification. Heterotrophic ossification can develop around nerves, so that the nerve will be ultimately encased in the heterotrophic ossification. Prior to performing the surgery, the surgeon also needs to have an understanding of the involvement of the articulation. This includes where the joint surface is intact, and whether the associated ligamentous structures that stabilize the joint are intact or would be violated by excision of the heterotrophic ossification.

Timing

Many texts recommend delaying surgery until complete maturation of the HA, which can be an extended period of time. However, it is the author's experience that the heterotrophic ossification can be excised once it is well defined on the CT scan. This is often as early as 3 months. A further delay will allow secondary contractures to develop and only compromises the rehabilitation of the patient.

Prophylaxis

Primary Prophylaxis

We have witnessed cases of severe HO in patients with significant injuries to the affected limb in association with major burns or spasticity (e.g., cervical spine or head injury). It is our experience that often these patients will have significant swelling of the area that is developing HO. This acute phase resolves over a few weeks. We have seen severe HO develop despite patients having documented prophylactic nonsteroidal anti-inflammatory medication.

As a consequence of witnessing these cases, it is our practice to provide prophylactic radiotherapy to those joints that are very high risk such as an associated head or spinal injury. This can usually be provided as a single dose of 700 gray to the area at risk.

Secondary Prophylaxis

For patients who have an established heterotrophic ossification for which surgery is being considered, we would include intravenous anti-inflammatories at the time of surgery, and then proceed with oral NSAID for 2 weeks.

For patients who have associated spasticity we would add prophylactic radiotherapy. The best way to provide this radiotherapy is a single dose of radiotherapy performed the day prior to surgery. The advantage of preoperation radiotherapy is that the patient is able to be assessed electively by the Radiotherapist, and have the radiotherapy provided on an out-patient basis before the surgery when the arm is not painful. No cases of secondary malignant change have been identified in patients who have had a dose of 700 gray or less. We have not witnessed any cases of abnormal wound healing in this patient population.

Surgical Approach

The surgical approach required is dictated by the position of heterotrophic ossification; however, standard surgical approaches are usual.

Major neurovascular structures must be identified before they enter the region of the heterotrophic ossification. These neurovascular structures are then followed from the normal tissue to the heterotrophic ossification. If the neurovascular structures pass through the HO, then they can be decompressed by using neurosurgical Kerrison rongeurs. These are designed to remove impinging bone while protecting the adjacent nerve roots.

Once the neurovascular structures have been safely identified and released, the surgeon can then divert their attention to removing the offending heterotrophic ossification. The easiest way to perform this is in the subperiosteal plane. However, this increases the chance of recurrence of the heterotrophic ossification as new bone can form below the periosteum.

The best way to remove the heterotrophic ossification is to develop an extra-periosteal plane. Usually this can be performed by blunt dissection with the surgeon's glove and a simple right-angled retractor. The heterotrophic ossification should be exposed so that a clear identification of the interval between normal bone and heterotrophic bone can be identified. Then an osteotome can be used to perform an osteotomy at this level. The abnormal bone and its periosteum are then removed.

Once the heterotrophic ossification has been removed, the adjacent neurovascular structures are again assessed to ensure that they have not been violated. The joint is also examined and a gentle manipulation may be required to release tight capsular tissue. The surgeon must be prepared to perform a soft-tissue reconstructive stabilization procedure if the joint is unstable.

It is important to obtain good hematosis to prevent a postoperative hematoma which may also ossify. Any bone debris should be removed and the wound irrigated. A deep drain is inserted to remove any subsequent hematoma.

Postoperative Phase

The patient is provided with intra-operative prophylactic intravenous anti-inflammatories and then a course of 2 weeks postoperative NSAID.

The patient needs to be closely observed in the postoperative phase. They can commence active mobilization in an attempt to regain a functional range of motion. The use of an interscalene block may be important for providing postoperative analgesics.

Patients who have continuous pain with progressive loss of motion in the postoperative phase need to be closely watched as these are the ones who are most likely to get a reoccurrence of the HO.

Those patients who develop a recurrence of the heterotrophic ossification often have associated pain and swelling of the joint. In a few cases where this has occurred, we have provided radiotherapy to the joint as late as 4 weeks. Interestingly, the patients described that the radiotherapy improved their pain, and they did not lose any further motion.

8.4 Shoulder Stiffness and Impingement

Subacromial impingement may be a factor in postoperative stiffness. Glenohumeral rotation can be limited by contact between the edge of the tuberosities and the acromion, or the edge of the glenoid (a phenomenon known as internal abutment). Mechanical impingement can reduce both passive and active range of motion about the glenohumeral joint. Fracture nonunion or malunion can result in modified osseous morphology that produces a mechanical impingent. Furthermore, the pain generated by mechanical impingement or associated subacromial bursitis can produce persistent guarding with subsequent loss of motion.

8.5 Shoulder Stiffness and Calcific Tendinitis

Adhesive capsulitis and posttraumatic changes are just two very specific types of rotator cuff disease processes that can result in shoulder stiffness. There exists, however, a multitude of rotator cuff disease entities that can also lead to shoulder stiffness. Any disease process, local or systemic, which disrupts normal rotator cuff excursion, tissue flexibility, or associated osseous anatomy, has the potential to result in shoulder stiffness. Rheumatoid arthritis and diabetes often contribute to shoulder stiffness. Calcific tendonitis of the rotator cuff is a well-recognized clinical disorder but of uncertain etiology. In fact, Brislin et al. have identified preoperative calcific tendinosis as a risk factor for developing postoperative stiffness. Categories of patients who are at risk for developing postoperative stiffness include single-tendon tears, concomitant labral repair, and calcific tendonitis [59].

Codman has previously described the calcific deposits that are characteristic of rotator cuff calcific tendonitis [60]. He and others hypothesized that degenerative changes in rotator cuff tissue induced calcific changes [61]. Others, such as Uthoff et al., have reported a nondegenerative mechanism characterized by a cell-mediated mechanism of calcification that results in tendon metaplasia [62, 63]. This is followed by a cycle of formative and resorptive calcification, which is mediated by multinucleated giant cells and ultimately remodels the tendon tissue [62, 63].

Symptoms of calcific tendonitis may be absent despite radiographic findings. However, there is a cohort of patients that will develop pain and stiffness. Nonoperative treatment modalities, such as physical therapy, anti-inflammatories, subacromial injection, aspiration, and high-energy extracorporeal shockwave therapy, have been described in the literature [64–69] 1, 5, 7, 8, 12, 15, 25, and 31. Hurt and Baker have reported that approximately 90 % of patients can expect resolution with nonoperative treatment [68]. However, in a 100 patient matched-pair analysis, Wittenberg et al. [70] concluded that operative treatment produced better long-term results. Surgical treatment typically involves debridement of the calcific deposit or combined debridement of the deposit and subacromial decompression. However, it is not well understood if the subacromial decompression in addition to removal of calcific deposit improves patient outcome. The exact mechanism by which calcific tendinitis can lead to postoperative stiffness remains unclear.

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Part III

Anatomy and Biomechanics

Anatomy Related to the Range of Motion

9

Brian B. Gilmer and Dan Guttmann

9.1 Pathophysiology

The term "frozen shoulder" has historically been used to describe shoulder pain and limited motion that was originally referred to as periarthritis. Neviaser was the first to identify the pathology through histological and surgical examination of frozen shoulder patients [15]. He concluded that frozen shoulder was not periarthritis, but a "thickening and contraction of the capsule that becomes adherent to the humeral head," which he termed "adhesive capsulitis."

A recent histological study revealed that there was no adhesion in the shoulder joint [9]. Therefore, the term "adhesive" may not be appropriate. In this chapter, we use the term "frozen shoulder," which refers to primary shoulder stiffness. Frozen shoulder is characterized by pain, stiffness, and limited function of the glenohumeral joint. Patients typically describe onset of shoulder pain followed by a loss of motion in a particular pattern. The most common limitations in range of motion are flexion, abduction, and external rotation. Approximately, 70 % of frozen shoulder patients are women. Males with frozen shoulder are at greater risk for longer recovery and greater disability [1, 8, 23].

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Nevasier and others have reported thickening and contraction of the shoulder capsule as well as inflammatory changes through histologic analysis. This contracture causes a shortening and fibrosis of the shoulder ligaments, which decreases the volume of the capsule, thus limiting range of motion.

It is likely that limitations in range of motion and the pain associated with frozen shoulder are not only related to capsular and ligamentous tightness, but also fascial restrictions, muscular tightness, and trigger points within the muscles [15].

9.2 Muscular Imbalance and Altered Kinematics

Frozen shoulder patients may exhibit significant alterations in shoulder kinematics, including increased elevation and upward scapular rotation.

This may manifest as the classic "shrug sign" during glenohumeral joint elevation, where the scapula migrates upward prior to 60° of abduction.

This indicates compensation due to lack of capsular extensibility as well as a change in central nervous system motor patterning with adaptive postural deviations such as anterior shoulder compensation or increased thoracic kyphosis [13, 21, 22].

9.2.1 Trapezius

Shoulder muscle imbalances also lead to altered shoulder motion and mechanics. Upper trapezius muscles are more activated than lower trapezius, creating an imbalance of the scapular stabilizers. This causes increased elevation and upward rotation of the scapula during elevation of the glenohumeral joint in both the frontal and sagittal planes. Patients with frozen shoulder have higher EMG ratios of the upper trapezius to lower trapezius during arm elevation when compared to asymptomatic subjects, indicating a muscular imbalance [5, 12].

9.2.2 Subscapularis

The subscapularis may have an independent role in frozen shoulder that is separate from the known capsular contractures. When the glenohumeral joint is positioned up to 45° of abduction, the proximal portion of the capsular ligamentous complex and the subscapularis were found to limit external rotation.

At 0° of abduction, these authors found that the subscapularis limited external rotation primarily. Therefore, it has been proposed that a greater loss of external rotation at 45° of abduction versus 90° of abduction indicates more specific involvement of the subscapularis [7].

Clinically, the subscapularis has been noted to be inflamed and often demonstrates adhesions to the adjacent anterior capsule and glenohumeral ligaments (Fig. 9.1).

Fig. 9.1 This arthroscopic view from a posterior portal demonstrates thickening of the upper rolled border of the subscapularis with synovitis and inflammation of the adjacent anterior capsule

9.3 Capsular Ligamentous Complex

The glenohumeral joint capsule, coracohumeral ligament, and glenohumeral ligaments (superior, middle, and inferior) make up the capsular ligamentous complex. These structures connect the humerus and glenoid and also attach to the glenoid rim via the labrum [4].

9.3.1 Anterior Superior (The Rotator Interval)

The rotator interval is a confluence of tissue in a triangular shape located between the anterior supraspinatus leading edge and the upper border of the subscapularis tendon. The apex of the rotator cuff interval is located at the margin of the transitional ligament where the biceps sulcus is located. The rotator interval includes the coracohumeral ligament, the superior glenohumeral ligament, and the anterior-superior capsule. Contracture of these structures, the most commonly affected in frozen shoulder, results clinically in loss of flexion and external rotation in adduction [6, 10, 14, 17, 18] (Fig. 9.2).

Clinically, the rotator interval may be obliterated by fronding synovitis or inflammation, making initial appreciation difficult during arthroscopy (Fig. 9.3).

Fig. 9.2 Examination under anesthesia in a patient with frozen shoulder. (**a**) Forward elevation with the scapula stabilized is limited to 90°. (**b**) External rotation with the arm at the side (0° of abduction) is limited to 0° (The authors would like to acknowledge Dr. Bryce Wolf for his assistance with these images)

Fig. 9.3 In this arthroscopic view from a posterior portal, there is complete obliteration of the rotator interval by proliferative synovitis and capsular contracture

9.3.2 Anterior

The anterior-inferior quadrant of the glenohumeral joint contains the anterior-inferior capsule and the middle glenohumeral ligament (MGHL). These structures are also almost invariably involved in frozen shoulder. The anterior capsule is frequently scarred to the middle glenohumeral ligament and the subscapularis. This results in further limitations of external rotation at the side and with increasing degrees of shoulder abduction [6, 11, 16].

9.3.3 Inferior

In Neer's original description based upon open surgical dissection, he described scarring of the axillary fold to itself and to the anatomic neck of the humerus [15]. However, newer research based on arthroscopic findings suggests that no such scarring of the axillary recess exists and that instead there is contracture of the inferior capsular tissues [25].

Nonetheless, this results in significant losses of abduction, forward flexion, and both external and internal rotation due to involvement of the anterior and posterior bands of the inferior glenohumeral ligaments (AIGHL/PIGHL) as well as the remainder of the capsular tissue [6].

9.3.4 Posterior

Clinically, internal rotation is less affected in frozen shoulder. When involved, the posterior capsule results in loss of internal rotation of the adducted arm. Because this tissue frequently appears to be less involved, the need for surgical release of the posterior capsule during surgical treatment has been debated [2, 6, 24].

9.3.5 Posterior-Superior

There has been recent evidence to suggest the existence of a posterior-superior glenohumeral ligament (PSGHL) [19]. Whether this represents a discrete structure or simply a thickening of the capsule is unclear [3, 20]. The relevance of this finding and its relevance to frozen shoulder are unknown. The expected effect of contracture would be a loss of internal rotation in adduction – an uncommon clinical finding.

Conclusions

While frozen shoulder has been shown to involve all portions of the capsule, it affects certain quadrants more predictably and severely. Additionally, capsular contracture has effects on the surrounding periscapular musculature. Surgeons treating this difficult problem need to consider not only the intraarticular pathology but its associated effects on the entire shoulder girdle.

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Pathomechanics of Frozen Shoulder: A Basic Research Perspective

10

Yon-Sik Yoo

10.1 Introduction

There exist numerous studies indicating that the frozen shoulder results in decreased external rotation and abduction compared to the contralateral side of the arm [9, 21, 24, 26]. Other studies have reported abnormal scapular position and altering scapular kinematics in frozen shoulder [5]. These clinical and research findings suggest that capsular stiffness can compromise normal glenohumeral and scapulothoracic movement as well as a link between two movements. Surprisingly, although fibrosis and resulting stiffness to the capsular ligaments has been widely implicated as a cause of idiopathic frozen shoulder [8], very few scientific data exist to confirm the capsular mechanics that can influence the entire shoulder kinematics. An improved understanding of the pathomechanics of capsular stiffness can further enhance the efficacy of rehabilitation and surgical releaser strategies. This article will review existing data on the biomechanics of the glenohumeral capsule and examine potential mechanisms of glenohumeral capsule and its influence on scapular movement.

10.2 Function of Capsular Components

The capsule is relatively thin and by itself would contribute little to the stability of the joint. The integrity of the capsule and the maintenance of the normal glenohumeral relationship depend on the reinforcement of the capsule by ligaments and the attachment of the muscle tendons of the rotator cuff mechanism [7, 15, 25]. The superior part of the capsule, together with the coracohumeral ligament, is important in strengthening the superior aspect of the joint and resisting the effect of

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gravity on the dependent limb [1, 25]. Anteriorly, the capsule is strengthened by the glenohumeral ligaments and the attachment of the subscapularis tendon. The latter is a major dynamic stabilizer of the anterior aspect of the shoulder. Posteriorly, the capsule is strengthened by the attachment of the teres minor and infraspinatus tendons. Inferiorly, the capsule is thin and weak and contributes little to the stability of the joint. The inferior part of the capsule is subjected to considerable strain because it is stretched tightly across the head of the humerus when the arm is elevated. The inferior part of the capsule, the weakest area, is lax and lies in folds when the arm is adducted. The capsular tension in abduction compresses the humeral head into the glenoid fossa. As abduction progresses, the capsular tension exerts an external rotation moment. This obligatory external rotation allows further abduction [13]. Active humeral rotation may also have the effect of altering capsular tension through capsuloligamentous dynamization [23]. The joint capsule is adherent to overlying rotator cuff except rotator interval. Conceptually, active shoulder motion may "dynamize" the capsule and ligament, turning the capsule into a significant stabilizing factor in the midrange of rotation at which the ligament and the capsule are relatively lax. Therefore, glenohumeral capsule undergoes sequential stretching with its length change and is being "dynamized" by contraction of overlying muscles during active range of motion. This reflects how certain aspects of capsule are under varying amounts of tension to control the humeral movement [19, 23].

10.3 Scapulothoracic Joint Motion

Scapulothoracic mechanics are another important component of shoulder motion. Scapulothoracic motion is responsible for approximately one-third of total shoulder elevation [5, 20]. The motion of the scapula from resting position includes three rotations: upward/downward rotation, external/internal rotation, and anterior/posterior tipping. In normal situation, the scapula demonstrates a pattern of progressive upward rotation, decreased internal rotation, and movement from anteriorly to posteriorly tipped position as humeral elevation angle increases. Electromyographic activity of periscapularis muscles including upper, lower trapezius (upper and lower), levator scapular, and serratus (anterior and posterior) increased with increased humeral elevation angles.

10.4 Abnormal Biomechanics: What We Already Know

It is known that the capsule is essentially lax during motion in the midrange. The distinct ligamentous structures of the capsule only really become significantly tense at the end-range extremes of motion. Different regions of the joint capsule have specific functions in limiting particular end-range motions. It is well recognized that the inferior glenohumeral ligament functions as a checkrein for obligatory external rotation in abduction [3, 16]. In the meantime, ligamentous structures within the rotator interval are thought be more important in limiting external rotation in the adducted arm. Thus, external rotational limitation is thought to be a consequence of contracted structures in the rotator interval and inferior glenohumeral ligament.

It has been generally understood that the contracture of coracohumeral ligament [2, 18] is the main pathology that can cause a decrease in external rotation at 0° abduction in frozen shoulder. The contracture of rotator interval including superior glenohumeral ligament is also known to be responsible for the decreased motion in external rotation at 30–60°abduction. In the meantime, the decrease in external rotation at 90°abduction was thought to be associated with the contracture of anterior-inferior capsule. The decrease in internal rotation was known to be a consequence of contracture of posterior capsule [3, 16, 23]. Therefore, the pattern of motion loss is dependent on arm position although it was not consistent across all subjects. Likewise, there seems to be no consistent pattern of limitation on motion by the severity of capsular contracure because other factors including scapular stiffness and scapulohumeral muscular imbalance are linked with the restriction of glenohumeral joint motion.

Abnormalities in the scapular rest position [22] and an early [11] and significant increase [5, 21, 24] in scapular upward rotation have been noticed in frozen shoulder and have been attributed to increased upper trapezius and reduced serratus anterior activation [4, 6, 12, 14]. The compensatory scapulothoracic and acromioclavicular motions that may create additional symptoms [17] have also been reported. But the causative or compensatory nature of these kinematic alterations and their associated biomechanical implications remain speculative. As there is no firm evidence, there is a scope for understanding the pathogenic kinematic mechanisms contributing to these alterations.

10.5 Capsular Kinematics Associated with Frozen Shoulder

We intended to investigate the stress pattern of glenohumeral capsule according to various humeral motions. To perform this simulation study, we constructed the finite element model of glenohumeral capsule based on CT arthrogram of three patients who were diagnosed with frozen shoulder. According to our computational study, the abduction and external rotation caused an increase in the stress of the anteroinferior capsule rather than the anterosuperior capsule. The anterosuperior capsule or rotator interval (Fig. 10.1) known for primary lesion [2, 18] did not show prominent stress in various motions except early stages of internal rotation behind the back angle and external rotation at 0° abduction in our frozen shoulder model. Unlike previous studies, internal rotation behind the back angle was restricted by a portion of the anteroinferior ligament rather than the posterior capsule [3, 16, 23]. Hence, our study did not support the fact that posterior capsule was related to restriction of the internal rotation as was evident from previous studies. Consequently, it is practically difficult to find a consistent pattern of limitation of motion only by the degree of capsular contracure because other factors including individual difference of capsular shape, scapular stiffness and scapulohumeral muscular imbalance are linked with restriction of glenohumeral joint motion.

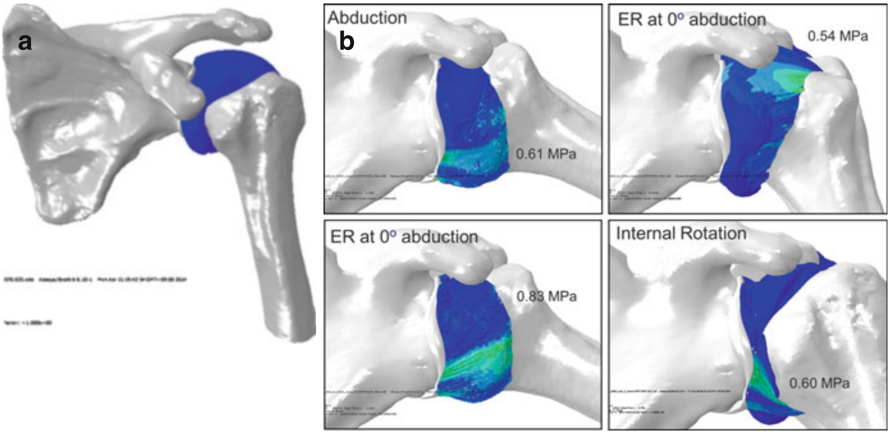

Fig. 10.1 (**a**) Zero-degree abduction of glenohumeral–capsular complex. (**b**) The capsule showing high stresses (*light blue color*) in various positions of representative patient with frozen shoulder

10.6 Altering Scapular Position Associated with Frozen Shoulder

Our study was also linked to evaluate the positional change of scapula and humerus with respect to the rib cage during two different shoulder abduction angles. A better understanding of positional differences of the scapula and humerus during glenohumeral movement is needed to better define the functional change of periscapula muscles and kinematic alteration of scapulothoracic force couple in patients with various degrees of frozen shoulder.

Thus, we compared the positional difference between scapulae obtained at maximum abduction position and 0° abduction in three patients with typical frozen shoulder. The averaged scapula–humeral angle of the affected shoulder and contralateral shoulder were $34 \pm 11°$ and $92 \pm 7°$, respectively. The abductions of the scapula of the affected shoulder and contralateral shoulder were $58 \pm 7°$ and $38 \pm 8°$, respectively. The posterior tipping of the affected shoulder was $25 \pm 5°$ (Fig. 10.2).

Therefore, the decrease of posterior tipping known generally in case of frozen shoulder should be verified again through an in-depth study. Also, the medial rotation of the scapula in the affected shoulder was decreased compared with the contralateral scapula in the same degree of glenohumeral angle. However, it is not obvious whether the cause of decreased medial rotation is due to the imbalance of scapular thoracic muscular activity or scapular stiffness itself. The scapula stiffness is another important factor that interferes with the natural shoulder kinematics in frozen shoulder. This concept was first suggested by Hamada et al. What they found in patient with frozen shoulder with passive mobilization test was a decreased range of motion in medial rotation, posterior tilt, and downward rotation. They considered scapular stiffness and muscular tightness as major factors rather than capsular contracture in frozen shoulder.

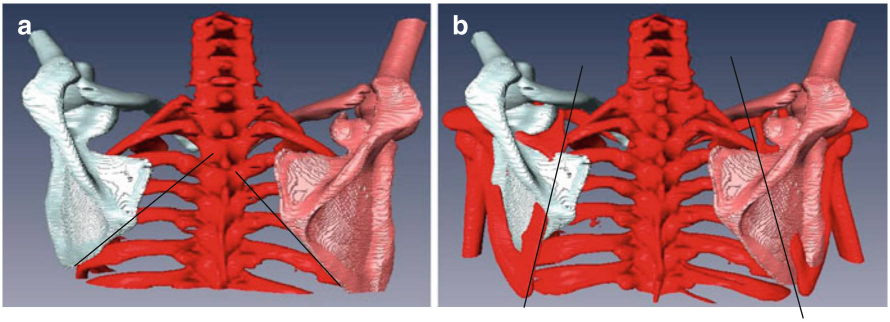

Fig. 10.2 A 3D abduction image of representative case in frozen shoulder (**a**) The left scapula (affected side) showing an increase in upward rotation. (**b**) Overlap image between two different positions indicating a decrease in medical rotation of scapula at abduction

10.7 Summary and Conclusion

Pathomechanical cascade in frozen shoulder has yet to be fully understood. Although a few studies have pointed out abnormal scapular location according to glenohumeral abduction using electromagnetic tracking device in frozen shoulder patient, that method has an innate limitation in evaluating detailed motions except upward and downward rotation [24]. Likewise, EMG study focusing on periscapular muscle activity did not provide an integral insight into changing the way of activity in both scapulohumeral and scapulothoracic muscle groups [10]. Thus, abnormal biomechanics in frozen shoulder needs to be further investigated.

According to our result and speculation based on our 3D kinematics, early abutment of humeral head against acromion which in turn causes failed obligatory external rotation was speculated to be an initiator of pathologic cascade in pathogenic mechanics of frozen shoulder. The change in position of humeral head causes another change in the axis of rotation of both the humerus and scapula, resulting in unpredictable periscapular muscular action.

In conclusion, pathomechanics of frozen shoulder characterized by glenohumeral joint limitation has a complicated pathogenesis made with the contracture of glenohumeral capsule, the periscapular stiffness, and the imbalance of strength of muscles surrounding the scapula.

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11 Rotator Interval and Stiffness

Catherine A. Logan, Petar Golijanin, Daniel J. Gross, Rachel M. Frank, and Matthew T. Provencher

11.1 Introduction

Despite a significant increase in the number of shoulder instability studies, the role of the rotator interval (RI) and the outcomes associated with various closure techniques remains controversial. The RI is a triangular space located between the subscapularis (SSc) and supraspinatus (SS) tendons in the anterosuperior region of the shoulder. It contains the superior glenohumeral (SGHL) and coracohumeral (CHL) ligaments, the long head of the biceps tendon (LHB), and joint capsule. Nobuhara and Ikeda [20] performed a clinical study on 101 patients and concluded that a deficient RI is present in patients with posterior instability and frozen shoulder. Biceps tendon stability largely depends on structures that are within RI, such as the SS and SSc tendons, SGHL, and CHL [10]. Multiple studies have shown that the structures within the RI aid in resisting posterior and inferior glenohumeral translation as well as maintain negative intra-articular pressure [29]. As imaging techniques improve, measuring the distance between the boundaries of the RI continues to play an essential role in diagnosis and clinical decision-making. Maintenance of optimal external rotation (ER) postoperatively while restoring stability to the shoulder is a primary goal, and the roles of surgical positioning and technique remain a source of

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discussion [7]. Stiffness of the shoulder almost always involves pathologic changes with the RI such as thickening, synovitis, tissue injury, and capsulitis.

11.2 Anatomy

Neer [19] first described the RI in 1979 as an anterosuperior space between the SS and SSc tendons. Its superior, inferior, and medial borders are the superior edge of the SSc, the anterior edge of the SS, and the base of coracoid, respectively [29]. The complexity of the RI lies in the relative density of its structural components; the CHL, SGHL, LHB and shoulder capsule all within a small area.

The CHL originates from the base of the coracoid and inserts on both greater and lesser tuberosity over the bicipital grove. From its origin, it splits into two distinct bands. One band inserts on the greater tuberosity and mergers into the SS tendon, while the other band inserts on lesser tuberosity and merges with the SSc tendon and transverse humeral ligament. Due to the intimate association between the CHL and the SS and SSc tendons, there is continued debate within the literature as to whether the CHL is truly a separate entity [22] or only a thickening of glenohumeral capsule [10].

The SGHL originates from the labrum that is adjacent supraglenoid tubercle. It is smaller than the CHL, and from its origin it crosses the floor of the RI deep to CHL, inserting on the superior aspect of the lesser tuberosity, a prominence known as the fovea capitis [21].

The LHB originates from the superior glenoid labrum and exits through the apex of the RI. It travels through the bicipital groove and combines with the SGHL, CHL and SSc tendon to make the biceps pulley system. This system is essential for proper biomechanics and helps to retain the LHB in its anatomic position.

11.3 Biomechanics

Similar to the debate regarding the discrete structure of the CHL, some studies have shown that the SGHL is the main glenohumeral stabilizer within the RI [28], while other studies claim that CHL is a more significant stabilizer [2]. Warner et al. showed that the role of the SGHL is more significant as a static restraint to inferior humeral head translation [28, 29]. Furthermore, Cooper et al. showed that the CHL is unlikely to play a significant role as a suspensory structure in its physiologic state [5]. However, Ovesen's [22] study showed that the CHL is the primary restraint to inferior humeral translation. He and his colleagues were radiographically measuring the subacromial (SA) space after sequential transection of the SSc tendon, CHL, and proximal part of the anterior capsule. The largest increase in the SA space was measured after release of the CHL. Moreover, Boardman et al. measured the cross-sectional area and tensile properties of both the CHL and SGHL in order to determine which of the two is the more important static stabilizer. They found not only that the CHL has a greater cross-sectional area, but also larger tensile strength properties, and therefore concluded that the CHL plays a more prominent role in static stabilization of the shoulder.

Harryman and colleagues [10] performed the first extensive cadaveric study of the RI in 1992 in order to analyze the effects of the RI on glenohumeral stability, motion, and translation. After sectioning of the RI, there is a significantly higher risk of posterior and inferior dislocations, while the imbrication of the RI increased the resistance to inferior and posterior glenohumeral translation [10]. Furthermore, it was shown that sectioning of the RI increases the range of glenohumeral flexion, extension, adduction, and external rotation. The RI contributes to stabilization against inferior translation of the humeral head in the adducted shoulder, and posterior translation in the abducted and externally rotated shoulder [10, 29]. Besides extensive motion restraint, the RI plays an important role in the maintenance of negative intra-articular pressure, and when combined with static and dynamic stabilizers, provides a significant contribution to overall shoulder stability [13, 28].

11.4 Pathologies

The shoulder joint is tasked with maintaining a delicate balance between instability and stiffness. The stiff, painful or "frozen shoulder" is not completely understood and may be idiopathic in nature or associated with trauma or periods of immobilization. Those with frozen shoulder commonly complain of pain both at rest and with movement, as well as while sleeping. Motion is restricted both passively and activity. The majority of cases are self-limited; however, a subset of patients is recalcitrant to conservative measures, such as physical therapy and injections, and thus are candidates for surgical intervention.

The rotator interval has been implicated in the vast majority of cases in the painful restricted shoulder. Contracture or scarring of the RI can manifest as a frozen shoulder, as demonstrated by both clinical exam and MRI findings [14, 23]. Limited external rotation of an adducted shoulder or an abducted shoulder is affected by anterosuperior and anteroinferior capsular tightening, respectively. MRI reveals obliteration of the subcoracoid fat plane, as well as anatomic changes of the rotator interval including thickening of the CHL and joint capsule [12, 15, 16]. Patients with a "stiff" or frozen RI will demonstrate loss of ER at the side, decreased flexion, and a variable loss of abduction (Fig. 11.1).

The importance of CHL release in a refractory frozen shoulder suggests its role in the pathoanatomy of this problem [3, 23] . The CHL is one of the primary restraints to external rotation during shoulder adduction, which is of significance clinically in a frozen shoulder [18, 25], and it is also thought to be the main pathologic structure restricting external rotation [10, 18, 23] and forward flexion in a frozen shoulder (Fig. 11.2). Patients will complain of pain, especially anteriosuperior, and have tenderness in anterior shoulder joint. There will be commensurate losses in motion and pain at the extreme limits of motion.

RI lesions can be difficult to evaluate during physical exam since they are often concomitant with anterior, posterior, and multidirectional instability (MDI) [7].

Fig. 11.1 Prior SLAP repair in a 34-year-old male, 1 year out from surgery. The rotator interval (RI) is significantly scarred with resultant loss of ER at the side in adduction. The probe is on the anterior loose suture from prior repair. The cannula is through the scarred RI

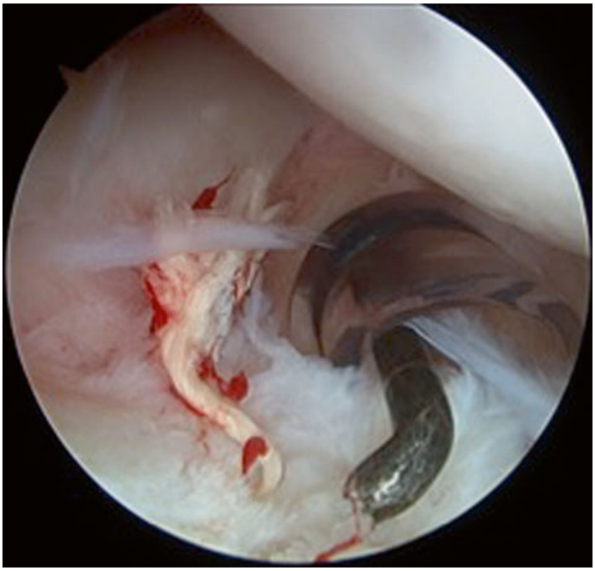

Fig. 11.2 Starting to release the superior aspect of the RI with a basket cutter

During the examination, it is essential to compare the injured shoulder with the opposite side in order to find any asymmetry in appearance, strength, sensation, stability, or ROM. Asymmetrical loss of ER in abduction may reveal overconstraint of the inferior ligaments, while loss of ER at the side may imply overconstraint of the superior capsule, RI, or SSc [7]. While making a surgical plan, it is imperative to test shoulder stability with focus on glenohumeral ligaments in order to determine the exact type of laxity. The sulcus sign test is a strong predictor of RI lesion [22] (Fig. 11.3). Throughout the test, a downward traction may cause inferior

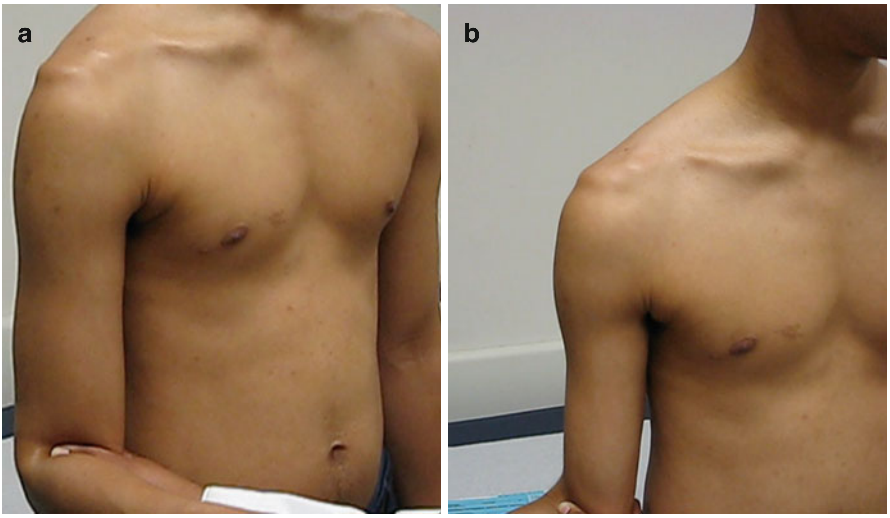

Fig. 11.3 (**a**, **b**) Sulcus sign test being performed. Downward traction on the arm causes a sulcus sign in internal rotation (**a**), and presence of the sign in external rotation is a predictable indication for an RI lesion

subluxation of the humeral head, and if the sign does not disappear with external rotation, the patient most likely has an RI lesion. In cases where the RI is intact, the sulcus sign will disappear due to proper function of the CHL [20].

11.5 Treatment

Different imaging modalities such as computed tomography (CT), MRI, and MRA can be used to diagnose RI pathology [7]. Plain radiographs are usually taken to assess any abnormalities within the joint such as osteoarthritis, glenoid bone loss, or humeral head deficiency. If there is any concern with regard to bony defects a three-dimensional CT reconstruction should be ordered. MRA is the most sensitive of all imaging studies, but if it is not properly performed, it can be extremely difficult to interpret the resulting image [7]. MRI in the oblique, coronal, and axial planes can be useful in assessing the structures within the anterosuperior capsule. In the coronal view, the RI can be seen superiorly from the SSc and inferiorly from the SS, while in the axial plane, the anterior coracohumeral ligament and SGHL can be seen anteriorly from the RI region. These views can be useful to assess thickening of CHL [4].

Initial management strategies are conservative, and the majority of patients with a frozen shoulder do well with symptomatic treatment, activity modification, and physical therapy [24]. Early on, in the acute inflammatory phase, aggressive physical therapy and exercise may be counterproductive, aggravating the patient's pain. During this phase, analgesia and activity modification prove more useful [24]. As the patient's pain eases, gentle physical therapist assisted range of motion and patient self-guided exercises are prescribed. A specific four-direction shoulder stretching exercise program has been successful [9].

While the literature reveals mixed reviews, corticosteroid injections are often used in parallel to physical therapy to assist with symptomatic control [9], but whether the pain relief is long term is unknown [11]. Refractory cases of frozen shoulder may necessitate manipulation under anesthesia (MUA) or surgical soft-tissue release. MUA may be done alone, or combined with an arthroscopic soft-tissue release procedure. It is unclear whether manipulation decreases the natural history of a frozen shoulder [1, 26]. Robinson et al. reported that arthroscopic release compared with manipulation in refractory cases has been shown to attain similar range of motion outcomes, but greater pain relief and functional improvement occurred in the arthroscopic release group [24].

The sequence of arthroscopic release and manipulation procedures is debated. MUA prior to arthroscopic capsular release has the disadvantage of uncontrolled tearing of tissues, increase in bleeding, and difficulty with subsequent arthroscopic release. Arthroscopy as the first step has the benefit of better visualization without a post-manipulation hemarthrosis [7] and is the recommended first step in surgical treatment.

The patient may be positioned in the lateral or beach chair position. Once acceptable visualization is established, a thick, robust capsule is usually confirmed. Synovitis and scar tissue within the RI may be debrided with a 3.5–4.0-mm oscillating shaver and electrocautery. Initial release consists of contractures within the RI, including the CHL, anterior capsule, SGHL and possibly the MGHL, and the subscapularis bursa (Fig. 11.4). If present, release of the adhesions between the conjoint tendon and the subscapularis is performed; however, care is taken not to release or cut the SSc (Fig. 11.5). Continuing anteroinferiorly, the anterior capsule and anterior band of the inferior glenohumeral ligament (IGHL) are released. The

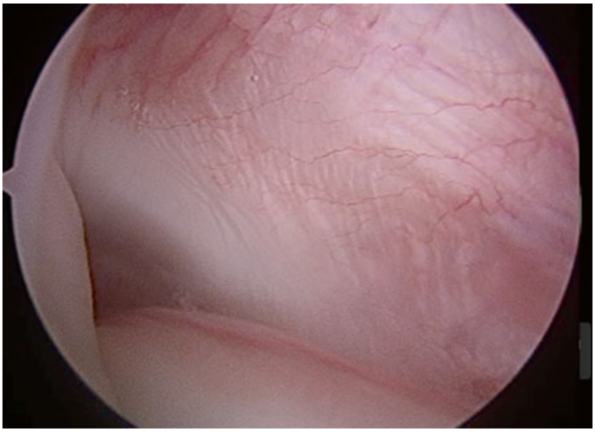

Fig. 11.4 RI contractures are part of the initial release of the RI that will contribute to resolving stiffness

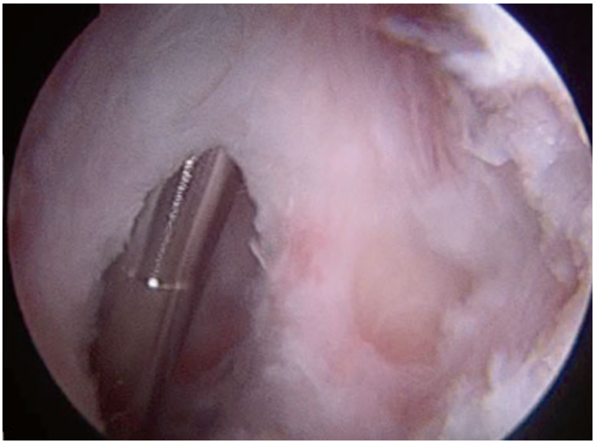

Fig. 11.5 The RI release is shown here, starting medially and extending laterally. Care must be taken not to cut the subscapularis tendon that is located inferiorly

release is then extended across the axillary pouch lateral to the labrum to allow for improved forward flexion, with caution to avoid the axillary nerve. Further intervention may be performed to release the posterior band of the IGHL and posterior capsule; however, the extent of release is influenced by clinical exam and surgeon preference [24, 27].

If performed, gentle manipulation is executed in a controlled manner after the controlled arthroscopic debridement, beginning with passive forward flexion to rupture the inferior capsule, followed by external rotation in glenohumeral adduction, then abduction, followed by internal rotation with the arm abducted. Care should be taken to avoid fracture of the humerus and manipulation should not be performed in an osteoporotic patient [6, 17]. A postoperative course of physical therapy is crucial to preserve motion gains, with daily therapy for 2 weeks, followed by a supervised progression of home exercises. Adequate analgesia is critical to promote participation in rehabilitation, and may be administered by an indwelling interscalene catheter postoperatively [24].

In rare cases, open release of contracture has been performed. Open release is indicated in previous failed attempts at arthroscopic release and frozen shoulder secondary to fracture or previous surgery [8].

11.6 Discussion

The RI has been linked to the pathoanatomy of frozen shoulder, with both clinical exam and MRI evidence illuminating its involvement in this painful problem. The CHL has an especially important role in the pathology, limiting both external rotation and forward flexion of the involved shoulder.

The frozen shoulder, while quite painful, is often self-limited in its natural history. Conservative modalities of activity modification and physical therapy are often the only treatment methods needed, although corticosteroid injections may be used in conjunction to assist in pain management. When conservative measures prove unsuccessful, MUA with or without an arthroscopic soft-tissue release may be considered. Post-manipulation gains in shoulder range of motion and functional activity seem to be sustained long term as reported by Farrell et al. [6], while combined manipulation and arthroscopic release have also produced favorable results [8].

Postoperative physical therapy and self-guided home exercises are a critical aspect of the patient's overall outcome. Interscalene catheters for pain control postoperatively may be used to ensure the patient's ability to participate in formal and self-guided exercise therapies.

Conclusion

The RI remains a topic of importance, and its specific role in frozen shoulder continues to be an area of need for further investigation. The RI's role in the pathology of frozen shoulder is better understood secondary to advances within basic science, imaging, and clinical outcome investigations. With regard to management, the majority of cases may be treated conservatively; however, for those cases that fail conservative therapies, MUA, arthroscopic soft tissue release, or both may be considered. Additional comparative clinical studies are needed to better define the effectiveness of our available treatment modalities.

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Part IV

Examinations

Pathophysiology of Frozen Shoulders: Histology and Laboratory Tests

12

Eiji Itoi and Yoshihiro Hagiwara

12.1 Histology

The pathophysiology of frozen shoulder has been thought to be a combination of synovial inflammation and capsular fibrosis [17, 19], and similarity of Dupuytren's disease was reported [3]. When a patient with frozen shoulder cannot regain satisfactory range of motion after prolonged conservative treatment, manipulation under anesthesia and/or arthroscopic capsular release would be the treatment of choice. Both of these procedures interrupt the continuity of the joint capsule, which results in an increase in the range of motion. This tells us that the joint capsule is the structure that mostly contributes to the restriction of motion. The joint capsule, therefore, seems to be one of the main pathologies of frozen shoulder. During surgery, one could see inflamed synovia with neoangiogenesis (related to inflammation), and thickened and stiffened joint capsule (related to fibrosis) [23], but the pathogenesis of frozen shoulder is still unclear.

Many studies reported the microscopic pathology of frozen shoulder. Both synovial and capsular tissues were analyzed but it is difficult to distinguish the two except for the subacromial space or synovial hyperplasia in the joint space. These two tissues might affect each other, resulting in capsular stiffness. The sampling stages of frozen shoulder also affect the results, which may lead to different results and interpretations.

Collagen density of the joint capsule increased and it became stiffer measured by scanning acoustic microscope in frozen shoulder [6]. The number of cells [3, 6] and structural collagens, such as collagen types I [6, 13] and III [6, 20], significantly increased in frozen shoulder (Figs. 12.1 and 12.2). Transformation from fibroblasts

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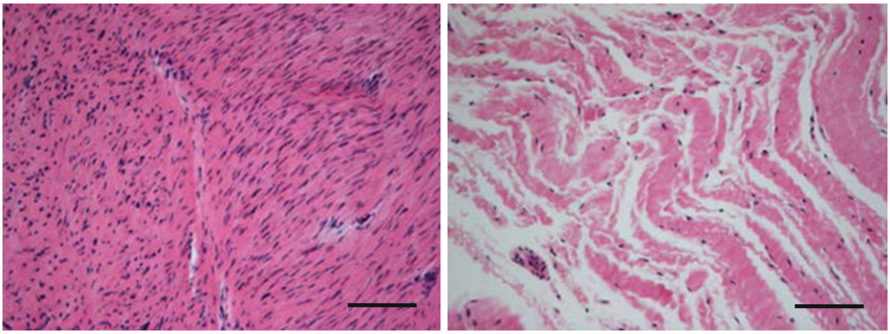

Fig. 12.1 H-E staining. The number of cells is increased and the collagen bundles are more densely packed with less space in between in the capsule of frozen shoulder. Scale bar = 100 μm

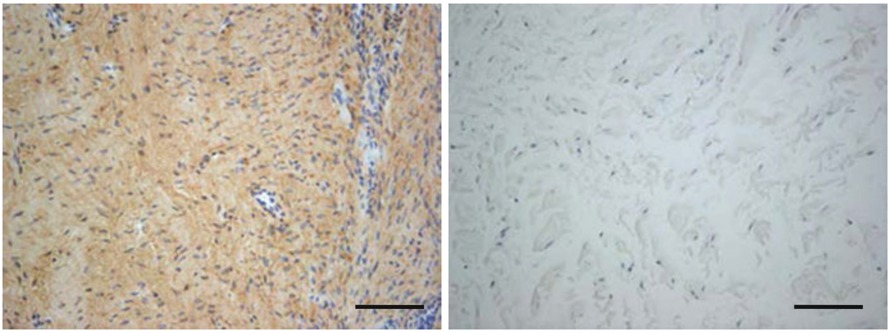

Fig. 12.2 Immunohistochemistry. Strong staining of type I collagen is observed in the capsule of frozen shoulder. Scale bar = 100 μm

to myofibroblasts is a key to understanding the pathology of frozen shoulder. Myofibroblasts increased in frozen shoulder [3], but others could not prove their existence in the joint capsule [6, 8]. A loss of fibril order and a twisting of collagen fibrils, which led to an up to fourfold diameter of the collagenous fibrils, were observed in frozen shoulder by transmission electron microscope examination [12]. There was low correlation between the intensity of the collagen type I expression by immunohistochemistry and the tissue elasticity measured by scanning acoustic microscope in frozen shoulder [6]. There remain other factors related to "capsular stiffness" such as abnormal cross-linking of collagens by advanced glycation end products [1].

Cytokines and growth factors related to fibrosis and inflammation increased in the joint capsule from frozen shoulder. Transforming growth factor beta (TGF-β), platelet-derived growth factor (PDGF), hepatocyte growth factor (HGF), interleukin-1-beta (IL1-β), IL-6, tumor necrosis factor-alpha (TNF-α), vimentin, secreted protein, acidic, cysteine-rich (SPARC, osteonectin), cyclooxygenase (COX) 1, COX-2, and intercellular adhesion molecule-1 (ICAM-1, CD54) increased in frozen shoulder [6, 8, 11, 14, 15, 20]. No up-regulation of matrix metalloproteinasis-1 (MMP-1), MMP-2, MMP-3, MMP-9, and MMP-14 was reported [5, 6]. Tissue inhibitor of metalloproteinase-1 (TIMP-1) increased but not for TIMP-2 and TIMP-3 in frozen shoulder [6]. Besides increased vascularity [6, 8, 24], an angiogenic factor of cystein-rich, angiogenic inducer, 61 (CYR61) increased in the joint capsule from frozen shoulder [6]. A decrease of blood supply from anterior humeral circumflex artery may affect an increase of angiogenic factors and hypervascularity related to a poor posture, frequently seen in patients with frozen shoulder [7]. Neurogenesis [24] and increased expression of neuronal proteins, such as substance P and calcitonin gene-related peptide (CGRP), were observed in frozen shoulder [6].

Same trends in the joint capsule were observed in synovial tissues. Related to fibrosis and inflammation, TNF-α, IL1-α, IL-1β, IL-6, TGF-β, PDGF, fibroblast growth factor (FGF), COX-2, and MMP-3 increased in the synovial tissues from frozen shoulder [10, 15, 20]. For angiogenesis, vascular endothelial growth factor (VEGF) increased in frozen shoulder [9, 11, 21].

Besides fibrosis and inflammation, chondrogenesis is one of the main pathologies of frozen shoulder [6, 18]. Although alcian blue staining intensity increased, there were no chondrocyte-like cells in frozen shoulder (Fig. 12.3). Gene expressions of aggrecan (ACAN), type II collagen (COL2A1), and type X collagen (COL10A1) increased in frozen shoulder [6]. From DNA microarray analysis, some genes related to fibrosis such as collagen type I (COL1A1) and collagen type XIII (COL13A1), chondrogenesis such as FBJ murine osteosarcoma viral oncogen homolog (FOS), ACAN, FOSB, and COL10A1, angiogenesis such as CYR61 were up-regulated in frozen shoulder [6].

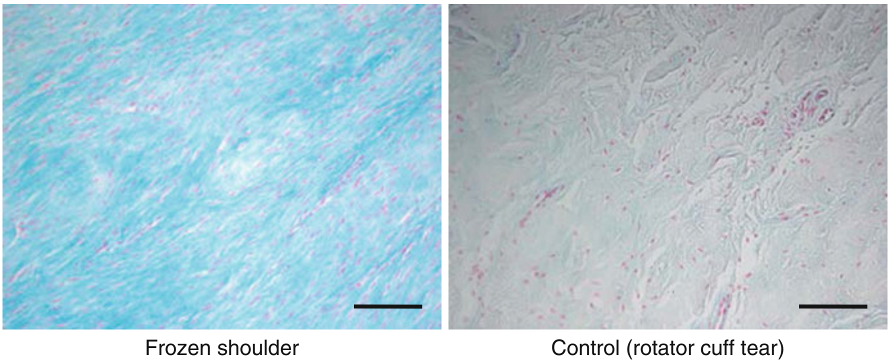

Fig. 12.3 Alcian blue staining. Proteoglycan, a major component of cartilage, is richly observed in the capsule of frozen shoulder (blue staining portion). Scale bar = 100 μm

12.2 Laboratory Tests

There is no specific blood test for frozen shoulder. The serum triglyceride and cholesterol levels were significantly elevated in frozen shoulder [4]. For type 2 diabetic patients, the blood sugar, both fasting and 2 h after breakfast, HbA1c and serum triglyceride levels significantly increased in patients with frozen shoulder compared to those without it [22]. Serum IgA and lymphocyte transformation to phytohemagglutinin significantly reduced [2]. Serum levels of ICAM-1 [14], TIMP-1, TIMP-2, and TGF-β1 were significantly higher, but MMP-1 and MMP-2 levels were significantly lower in frozen shoulder [16]. After intensive stretching, serum levels of MMP-1 and MMP-2 increased but TIMP-1 and TIMP-2 decreased compared with supervised neglect in frozen shoulder [16].

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Clinical Symptoms and Physical Examinations

13

Ronald L. Diercks and Tom Clement Ludvigsen

13.1 Introduction

Frozen shoulder is first described in the nineteenth century [17, 18]. Since then more descriptions and anecdotic notes have been written about this entity. It is not a rare condition, as the prevalence of frozen shoulder is 2–5 % in the population.

One of the main discussions throughout the last century is that there is no consensus about the definition and classification of frozen shoulder. This influences the numbers concerning incidence and prevalence. An expert workshop was held by the Health and Safety Executive (HSE) and the University of Birmingham to determine a set of diagnostic criteria for frozen shoulder using a Delphi technique [19]. Among the experts were rheumatologists, orthopedic surgeons, occupational medicine specialists, epidemiologists, physical therapists, ergonomists, and clinical psychologists. They came up with the following definition: frozen shoulder is a condition characterized by current or past pain in the upper arm, with global restriction of glenohumeral movement in a capsular pattern. The tendency is to label any patient with a stiff, painful shoulder as a case of frozen shoulder. Frozen shoulder is a specific condition that has a natural history of spontaneous resolution and requires a management pathway that is completely different from other shoulder conditions leading to restricted movement. Specific criteria are a history of unilateral pain in the deltoid area and equal restriction of active and passive glenohumeral movement in a capsular pattern (external rotation > abduction > internal rotation). In a review of

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the definitions used for frozen shoulder, adhesive capsulitis, and painful stiff shoulder in 21 RCTs, Schellingerhout [20] concluded there was unanimous agreement that restricted movement should be present, but there was no consensus regarding the sort, the amount, and the direction of restriction or the presence of shoulder pain at night. Both Schellingerhout and Harrington [11] noted that pain could be present. Zuckerman [22] surveyed members of the American Shoulder and Elbow surgeons (ASES) and proposed to them a definition and a classification of frozen shoulder. The following definition was accepted: frozen shoulder is a condition characterized by functional restriction of both active and passive shoulder motion for which radiographs of the glenohumeral joint are essentially unremarkable except for the possible presence of osteopenia or calcific tendonitis. Frozen shoulder can be divided in primary (idiopathic) and a secondary form. Eighty-two percent of the members (strongly) agreed with this definition. Eighty-six percent (strongly) agreed the division into primary and secondary types, but only 66 % (strongly) agreed that secondary frozen shoulder should be divided into intrinsic, extrinsic, and systemic types.

13.2 Patient History and Symptoms

The diagnosis of "frozen shoulder" is based upon the history of an insidious onset [3–8, 12, 13, 22]. Pain starts often at night and is sometimes so severe that it prevents the patient from sleeping on the affected side. Gradually pain increases and is present all the time. At clinical examination the typical finding is a global reduction of range of motion, by definition in two or more planes, and equal in passive and active examination [2, 14, 15]. Three phases of have been described. The first, or freezing phase, is distinguished mainly by pain and gradually increasing stiffness and lasts for 2–9 months. In the second, or frozen phase, lasting from 4 to 12 months, pain is less severe and there is minor discomfort in the shoulder, but stiffness is substantial. In the third thawing phase, function is gradually restored and pain is resolved. This can take a further 5–26 months. Some patients may regain full use of their shoulder within 12–18 months, whereas others may have persistent symptoms for several more months. Opposed to that, the diagnosis of "stiff shoulder" is based on a history of joint-related disease or trauma. An important differential aspect is that in "frozen shoulder" there is no anatomical abnormality, no imaging abnormality, and no systemic abnormality or disease that can directly explain these typical signs and symptoms [1, 9, 21].

13.3 Physical Examinations (see also Chap. 14)

In patients with shouder stiffness essential measurements of ROM are forward flexion, external rotation with the arm at the side, and internal rotation in the standing position. In addition, in order to eliminate the effect of pain during the measurement, measuring the external rotation with the patient in the supine position can be helpful through stabilization of the scapula [10]. In “frozen shoulder” in the early phase, loss of external rotation with the arm along the side is typical, the pain is located at the insertional area of the deltoid on the proximal part of the humerus, and there is often some tenderness at palpation of the coracohumeral ligament. Important feature is that the reduction in range of motion is fixed and not influenced by pain. This means that even in a fully anesthesized patient the range of motion will be similarly reduced. Strength is unaffected when tested within the pain-free range. During the evolution from phase I to phase II, pain diminishes, but stiffness persists. As a guideline, if the range of motion measured is less than 100° in forward flexion, less than 10° in external rotation, and less than L5 level in internal rotation, we can define this as “frozen shoulder.”

In patients with “stiff shoulders,” the range of motion is typically reduced in the directions affected by the underlying abnormalities. Often there is a difference in ROM between active and passive examination. A prerequisite is that there are imaging abnormalities coinciding with the decreased range of motion.

When first symptoms appear, differentiation between other causes of shoulder pain is difficult [16] in this phase, pain is the principal problem, stiffness is not yet evident. Differentiation between pain-induced reduction in ROM and true “capsular” reduction can be difficult. In the early presentation of degenerative disease of the joint or the rotator cuff, the same primary symptoms may be prominent.

Frozen shoulder is characterized by functional restriction of both active and passive shoulder motion for which imaging of the glenohumeral joint is essentially unremarkable.

The UEW advices as a guideline: a range of motion of less than 100° in forward flexion, less than 10° in external rotation, and less than L5 level in internal rotation is indicative.

In stiff shoulders with less limited ROM or non-global ROM, X-ray and also MRI are indicated to rule out other pathologies.

Specific findings like diabetes and thyroid disease, have also been described regarding pathology found in frozen shoulder that indicate further general diagnostic procedures.

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14 Imaging of Stiff Shoulders

Hiroyuki Sugaya

14.1 Introduction

The term "frozen shoulder" was coined by Codman in 1934 [1]; then, other authors subdivided this into primary, secondary, and tertiary (postsurgical or post-traumatic) categories [2]. In the meantime, Zuckerman and Rokito proposed a definition for frozen shoulder incorporating the term "functional restriction of both active and passive shoulder motion" [3]. The definition stipulated that a normal joint space must be present on plain radiographs, and it included a separate subgroup for idiopathic/adhesive capsulitis [3]. Based on the consensus definition of shoulder stiffness by the ISAKOS Upper Extremity Committee, the term "stiff shoulder" should be used to describe the patient who presents with a restricted range of motion. The etiology can be due to primary or secondary causes. The term "frozen shoulder" is used exclusively to describe the primary idiopathic stiff shoulder associated with severe and global motion loss [4–6]. Regarding imaging in stiff shoulders, secondary stiff shoulders present various conditions based on their primary pathology, such as rotator cuff tears, calcific tendinitis, fractures, osteoarthritis, and so on. On the other hand, there is no consensus of opinion that primary frozen shoulders could be related to, for example, cuff pathology. Recent advanced imaging technology and molecular biology have shown that some patients with primary frozen shoulder presumably have trivial changes in the tissues around the shoulder including rotator cuff [7–10]. However, imaging in primary stiff shoulders is not yet described enough. Therefore, in this chapter, imaging of stiff shoulders with various conditions is reviewed and summarized.

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14.2 Imaging of Secondary Stiff Shoulders

14.2.1 Stiff Shoulders After Trauma or Surgery

Shoulder stiffness is frequently associated with major or minor traumatic incidents, such as fractures around the shoulder girdle, rotator cuff tears, and even after whiplash neck injury. The pathology found on imaging in stiff shoulders after trauma or surgery varies according to the primary pathology.

14.2.2 Stiff Shoulders Associated with Diabetes Mellitus

Patients with diabetes mellitus have an increased risk of developing limited joint motion, including shoulder and other joints. Pathomechanically, high circulating glucose levels may accelerate aging of certain proteins by forming and accumulating irreversible cross-links between adjacent protein molecules [11, 12]. Shoulder stiffness in patients with diabetes mellitus reportedly antedates diabetic symptoms [13]. The longer a patient has been receiving insulin, the higher the risk of developing shoulder stiffness, and the greater the resistance to all treatment modalities [14–16]. Although age-related degenerative changes in rotator cuff tendon are reported to be more common in shoulders with diabetes mellitus [17], to date there exist no studies which report associations of rotator cuff pathology with stiff shoulders in patients with diabetes mellitus.

14.2.3 Stiff Shoulders Associated with Other Extrinsic Factors

For many years, clinicians have been keen to associate coronary artery disease with shoulder stiffness. In fact, Ernstene and colleagues reviewed 133 consecutive cases of myocardial infarction and found 17 patients whose original presenting symptom was unrelenting pain in the shoulder region [18]. In the meantime, although if the severity or the duration of pulmonary disorders correlates with shoulder stiffness is unknown, frozen shoulder reportedly occurs more frequently in patients afflicted with emphysema and chronic bronchitis [19]. Regarding neurological conditions, there exists much higher chance to develop frozen shoulders in patients with Parkinson's disease (13 %) compared with age-matched controls (1.7 %) [20]. In addition, others also reported a 25 % incidence of frozen shoulder in patients who had subarachnoid hemorrhage [21]. There is no report of associated rotator cuff pathology or imaging abnormalities in stiff shoulders associated with these extrinsic factors.

14.2.4 Stiff Shoulders Associated with Calcific Tendinitis

Etiology of calcific tendinitis is still not clear [22–24]. Patients with calcific tendinitis demonstrate various symptoms according to phases of the disease. Patients in

Fig. 14.1 Images in patient with calcific tendinitis. X-ray (*left*) and 3D CT (*right*)

inflammatory stage complain refractory pain at night and global motion deficit of the shoulder. Once pain subsides, some patients mainly complain shoulder stiffness without pain, and others complain subacromial impingement and catching of the shoulder without restriction of shoulder motion [25]. Calcific deposit can be easily detected through X-rays and CT images, and this is the typical finding (Fig. 14.1). However, partial-thickness rotator cuff tear is often observed in MRI.

14.2.5 Shoulder Stiffness Associated with Osteoarthritis

Osteoarthritis around the shoulder girdle restricts shoulder motion, and the degree of restriction normally depends on severity of osteoarthritis. In shoulders with glenohumeral osteoarthritis, shoulder motion is mechanically restricted due to capsular fibrosis and to excessive osteophytes and malcongruity of the joint, which is very easily detected by X-rays (Fig. 14.2). Rotator cuff is normally well preserved in primary osteoarthritis on MRI. In shoulders with osteoarthritis in the acromioclavicular joint, forward flexion is often restricted due to less mobility and pain at the joint. In these shoulders, external rotation and internal rotation are normally well preserved (Fig. 14.3).

14.2.6 Shoulder Stiffness in Full-Thickness Rotator Cuff Tears

Shoulder stiffness is frequently associated in patients with symptomatic rotator cuff tears. Recently, Iwamoto and coworkers reported characteristics of shoulder stiffness in patients with full-thickness rotator cuff tears in a Japanese journal

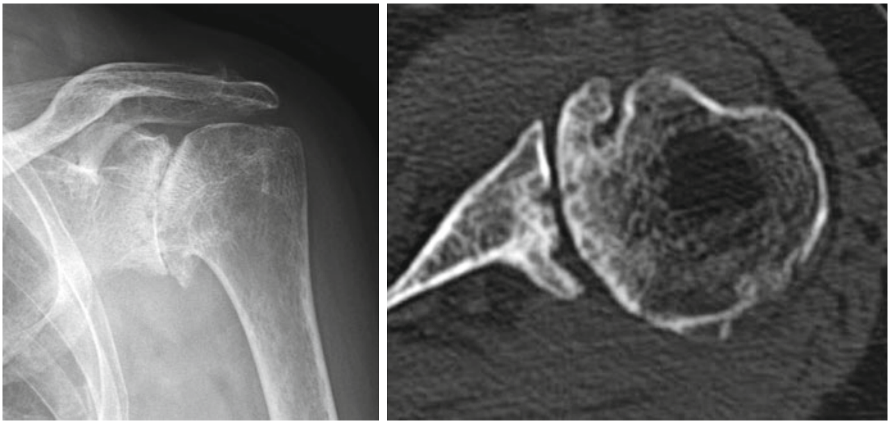


Fig. 14.2 Images in patient with primary glenohumeral osteoarthritis. X-ray (*left*) and an axial image of CT (*right*)

Fig. 14.3 X-ray image in patient with primary osteoarthritis in the acromioclavicular joint

[26]. They investigated patterns and prevalence of shoulder stiffness in 155 patients with unilateral symptomatic full-thickness rotator cuff tear who underwent surgery and compared with 20 patients who underwent arthroscopic pancapsular release for refractory stiff shoulder during the same period. In this article, 89 % of 155 shoulders with full-thickness rotator cuff tears demonstrated passive range of motion deficit in at least one of the following motions which includes

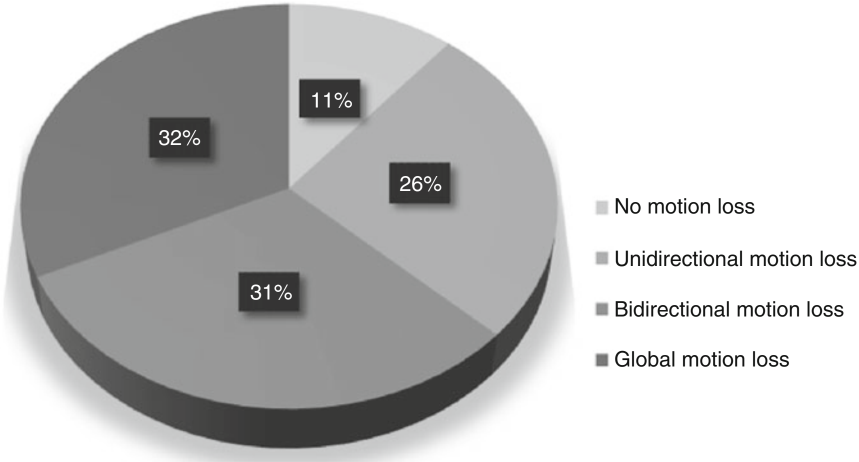


Fig. 14.4 Patterns and prevalence of range of motion loss in shoulders with full-thickness rotator cuff tears. Eighty-nine percent of shoulders demonstrated passive range of motion deficit at least one of the following motion: forward flexion, external rotation at side, and internal rotation preoperatively. Twenty-six percent of shoulders demonstrated unidirectional, 31 % demonstrated bidirectional, and 32 % demonstrated global motion restriction. However, none of the 49 shoulders who had global motion loss demonstrated severe and global loss of motion

forward flexion (FF), external rotation at side (ER), and internal rotation (IR) preoperatively. Among them, unidirectional motion restriction was seen in 41 shoulders (26.5 %) and bidirectional restriction observed in 48 shoulders (31.0 %). The rest of 49 shoulders (31.6 %) demonstrated global motion restriction (Fig. 14.4). On the other hand, all 20 refractory stiff shoulders who underwent capsular release demonstrated severe and global loss of motion, which was less than 100° in FF, 10° in ER, and less than L5 in IR. However, none of the 49 shoulders (31.6 %) with full-thickness rotator cuff tears who demonstrated global motion loss did not meet above criteria (severe and global motion loss). Therefore, they concluded that range of motion deficit in patients with full-thickness rotator cuff tears was relatively mild compared with that in refractory stiff shoulders which requires pancapsular release.

14.3 Imaging of Frozen Shoulders or Adhesive Capsulitis

It has been believed that rotator cuff disease and adhesive capsulitis can typically be distinguished based on a careful history and physical examination [27]. However, since the recent progress of diagnostic imaging revealed high prevalence of rotator cuff lesions in frozen shoulders, several articles reported that the rotator cuff lesions are strongly associated with frozen shoulders or idiopathic adhesive capsulitis [6, 28–30]. Although these conditions may be present concomitantly, it is less likely for patients with idiopathic adhesive capsulitis to have rotator cuff pathology.

Loeffler and colleagues retrospectively reviewed the medical records of 38 consecutive patients who underwent arthroscopic capsular release for adhesive capsulitis [31]. A preoperative MRI diagnosis was compared to the actual status of the rotator cuff during surgery. Although preoperative MRI interpretations predicted an incidence of rotator cuff tears of 57.9 %, operative findings revealed a true incidence of rotator cuff pathology of only 13.2 % ($P < .0001$). They concluded that MRI interpretations in patients with adhesive capsulitis may provide misleading information with a high percentage of false-positive reports of rotator cuff tears [31].

In a very recent study, Ueda and colleagues have investigated rotator cuff lesions in frozen shoulders with global and severe motion loss using a large cohort of patients [32]. A consecutive series of 379 stiff shoulders, in which patients with evident secondary stiff shoulders were excluded, was prospectively investigated using MRI or ultrasonography. Among them, 89 shoulders demonstrated severe and global loss of passive motion based on the criteria in the previous literature [26]. In these shoulders, only 9 % showed a partial-thickness cuff tear; in 91 % an intact rotator cuff was seen. Interestingly, none of the patients demonstrated full-thickness rotator cuff tears.

In this manuscript, they also insisted of importance of ROM measurement methods since many patients complain pain during measurement. Therefore, they recommend to measure external rotation with the patient in the supine position in order to diminish the effect of pain, in addition to standard measurement with the patients in the standing or sitting position [32].

Case Presentations

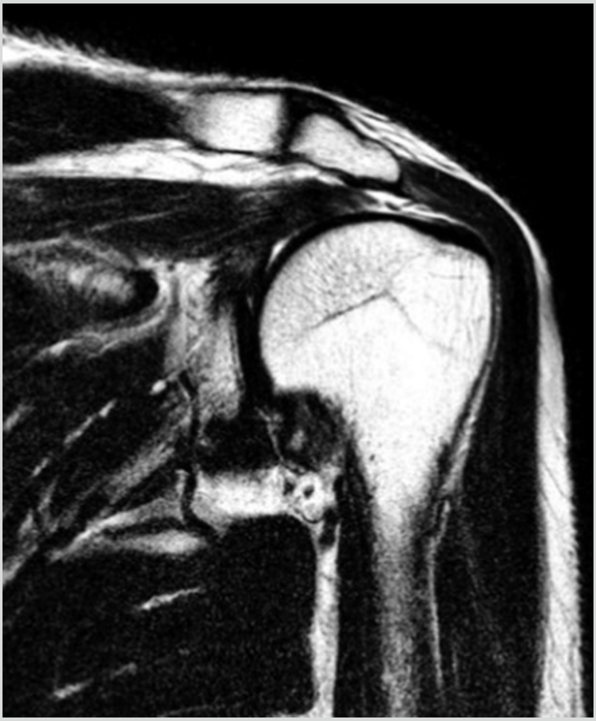
Following are the examples showing how the pain, range of motion, and position of the body are intimately related.

Case 1. A 51-year-old female presented to our institute with left shoulder pain and range of motion deficit. Her external rotation with the arm at the side was −5° in the standing position and 0° in the supine position, with similar pain in both positions. MRI demonstrated intact rotator cuff (Fig. 14.5).

Case 2. A 55-year-old male complained of left shoulder pain and limited range of motion. His external rotation at the side in the standing position was 10° with pain, whereas it increased up to 50° in the supine position where he felt much less pain. MRI demonstrated a full-thickness rotator cuff tear (Fig. 14.6).

Fig. 14.5 MRI on the left shoulder in a 51-year-old female. T2-weighted coronal image demonstrates intact rotator cuff

Fig. 14.6 MRI on the left shoulder in a 55-year-old male. T2-weighted coronal image demonstrates full-thickness rotator cuff tear



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Part V

Conservative Treatment

15 Natural Course of Frozen Shoulder

Maria Valencia and Emilio Calvo

Primary stiff shoulder or “frozen shoulder” has been classically regarded as a self-limiting entity that should be managed conservatively. Considering the theoretical benign prognosis of this condition, there is still controversy on several characteristics of the disease that once identified, could help to recommend additional therapies in a selected subgroup of patients in order to reduce the impact of the disease on the patient. For instance, the presence of certain factors, like the duration of the symptoms, could be useful to establish a prognosis and, thus, select an individualized specific treatment option. Traditionally, Reeves [8] described three clinical phases in the evolution of idiopathic frozen shoulder. The first one, the “freezing” phase, develops within the first 3–9 months and presents with diffuse pain and progressive stiffness. The patient may attribute the symptoms to a trivial injury. Pain usually precedes the restriction in motion, but not necessarily. The second one, the “frozen” phase, is characterized by diminished pain and an established stiffness. It typically lasts for 4–12 months. The last phase is called the resolution or “thawing” phase. Pain and stiffness start to gradually improve along a period of time that can last from 12 to 24 months. Although new classifications have been proposed, all of them stress the fact that the process is continuous and that the three phases are highly variable and may overlap. The duration of the condition is from 1 to 3.5 years with a mean of 30 months. It is not unusual that the opposite shoulder is affected subsequently between 6 months and 7 years after initial onset of symptoms of the first shoulder [8].

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The degree of recovery is variable and depends on the cause of the frozen shoulder as well as other factors that remain controversial [10]. Studies regarding the natural course of frozen shoulder are very scant. Russell et al. [9] have pointed out that including a “nontreatment” group in a comparative study could incur in conflict with ethical restrictions. This is one of the reasons why it is difficult to assess what the evolution without any kind of treatment would be. The other important difficulty is the delay in recognition and diagnosis in the early phases. It has been published that the mean duration of shoulder pain prior to the initial evaluation is approximately 9 months [6]. Moreover, most of the studies regarding the natural history of frozen shoulder do not specify if any kind of physical therapy, oral treatment, or injections were performed during the follow-up.

Codman [2] originally described a self-limited course of frozen shoulder with a treatment that consisted of supervised neglect with analgesia, physical therapy, or steroid injection. Other studies have confirmed this self-limiting course and have reported rates of recovery in more than 90 % of patients [3, 5, 10]. However, residual pain and restriction in motion are not uncommon. Binder et al. [1] reported a series of 40 patients prospectively followed for a mean of 44 months. Although 18 patients (45 %) had residual symptoms in terms of pain at final follow-up, only one was severe. However, 16 patients (40 %) had not reached a complete range of motion when compared to the age- and gender-matched control group. Five patients presented with severe restriction in range of motion and four had developed the same symptoms in the uninvolved shoulder. They found three prognostic factors of worse outcome: dominant arm involvement, manual labor, and mobilization therapy. Men showed more restriction than women at final follow-up, but this difference was not statistically significant. Traumatic onset and duration of symptoms at presentation were not factors related to outcome. Reeves [8] published a long-term follow-up study with similar results. At 5–10-years follow-up, 3 of 49 patients presented with severe disability and 22 with mild disability. Some other authors, on the other hand, have obtained better results, as Grey et al. [4], in which series 24 of 25 patients with untreated frozen shoulder achieved complete recovery.

More recent studies have also reported variable results. Griggs et al. reported that most patients could be treated successfully with a specific 4-direction shoulder stretching exercise program that would start during the second phase of the disease [5]. They considered it a failure when the patient was unsatisfied after 3 months of treatment. They divided the patients into two groups: those who finally needed a surgery and those who did not. Previous treatments with physical therapy and “workers’ compensation” claim or litigation were the only variables associated with failure or eventual manipulation or capsular release. Age, menopause, duration of symptoms, dominancy, trivial trauma, medical comorbidities, initial degree of pain, range of motion or Simple Shoulder Test score were not related to the indication to pursue a surgery. In their study, the mean time for the patient to decide to undergo a surgical procedure was 6 months (range, 3–13 months) after the initial evaluation and only five patients of 71 (7 %) conformed to this group.

Hand et al. [6] demonstrated that 41 % of the patients had mild to moderate symptoms at 7 years and 6 % had severe ongoing symptoms with pain and functional loss. The mean time to recover full shoulder function was 4.4 years (52.3 months). They suggest that symptoms improve in the first 3 years from onset and that this improvement then ceases. Although in their series they include a variety of treatment options, what seems clear is that those patients with the most severe symptoms during the first 6 months have the worst long-term prognosis. Differently to Binder et al. [1], they did not find any relation between the dominant arm and a worse prognosis, neither with insidious onset as referred by Codman [2] nor having had a minor traumatic event. The authors suggest that diabetic patients might have a worse prognosis but Levine et al. did not find any differences in rate of success of nonoperative treatment when referred to this group of patients [7].

Vastamäki et al. have recently reported the results of idiopathic frozen shoulder by comparing two groups: one consisting in observation only (nontreatment group) and the other consisting of conservative management [10]. The duration of the symptoms was similar in both groups (15 and 20 months, respectively). At final follow-up of 2 years, the range of motion had improved to the contralateral level in 94 % in the untreated group and 91 % in the nonoperative group. Fifty-one percent of patients in the untreated group and 44 % in the nonoperative group were totally pain free. The Constant Murley score averaged 83 (86 %) in the untreated group and 81 (77 %) in the nonoperative group. Similarly, Diercks and Stevens stated also that supervised neglect yields better outcome than intense physical therapy and passive stretching [3]. In their study, within 2 years, 89 % in the supervised neglect group reached a Constant score of 80 or higher and 64 % of them have reached this level within 12 months. They achieved a better range of motion through a shorter and less painful process.

In conclusion, while in most cases frozen shoulder will resolve spontaneously, it usually takes a long period of time that may severely impact on patients' quality of life. Although prognostic factors still remain controversial, symptomatic onset and progression during the first 6 months seem to determine a worse outcome as well as being involved in a litigation or worker's compensation claim. Age at the time of presentation could influence in our treatment algorithm, as younger patients can be more demanding according to their activity expectations. It is not still clear whether diabetic patients have a worse prognosis and will need a quicker indication for surgery.

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Physical Therapy

16

Francisco J. Vergara and Ronald L. Diercks

16.1 Physical Therapy and Its Efficacy

A systematic review was made on the effects of physical therapy on frozen shoulder. There are several papers discussing the effect of physical therapy in the treatment of frozen shoulder. Some publications maintain that there are no differences in the final results, because the disease is self-limiting [1, 2]. A meta-analysis, by Green et al. reviews 26 therapies for diverse shoulder diseases, of which only four discuss frozen shoulder. They conclude that there is no evidence that physical therapy alone could have a significant effect on the end result of frozen shoulder. This study also showed evidence of laser therapy being beneficial in the short term [3]. On the other hand, Levine [4], reports 90 % improvement with oral anti-inflammatory medications and/or steroid injections, when combined with physical therapy. It is suggested that invasive procedures should not be performed without first attempting 6 months of conservative treatment [5].

Although different physical therapy programs have been studied [6–8], there is no consensus with respect to the frequency, intensity, and duration of physical therapy in frozen shoulder. Diercks evaluated the effect of intensive physical rehabilitation treatment, including passive stretching versus supportive therapy and exercises within pain limits in 77 patients. They observed significantly earlier recovery of function and less pain in the group treated supportively at 12 months, compared to

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the group that was treated with passive manipulation beyond the painless range [9]. There is no further evidence supporting passive capsular and muscle stretching, soft tissue manipulation, and joint mobilization techniques.

The effectiveness of adding scapula-thoracic exercises to the specific exercises for the glenohumeral joint has been researched: scapula-thoracic exercises that include scapular stabilization should be added to achieve the goals of pain relief, increase in range of motion, and recovery of scapula-humeral rhythm [8, 10, 11]

A large number of studies have been performed on the various techniques of physical therapy, manual therapy, mobilization techniques, and stretching techniques [7, 8, 12–17]. Small differences in outcomes are reported, but in most cases either the inclusion criteria are not strictly defined or the results are operator-dependent.

Despite these findings, we can say that manual therapy techniques can complement conventional treatments for frozen shoulder. A recent study establishes the use of Mulligan techniques based upon painfree repositioning of the joint by applying glides to it, achieving analgesic effects as well as an increase in the range of motion that last beyond the maneuver, thus allowing improvement in patient functionality [12]. In this study, the authors compare these techniques to stretching exercises. They conclude that although both were effective for restoration of range of motion and reduction in pain, Mulligan techniques achieve greater benefits.

Another manual therapy method, the Maitland technique which involves indirect manipulative techniques of the joint was used by Deshmukh et al. [13]. In their study, the authors use myofascial release techniques and Maitland technique for joint mobilization. Both methods achieve significant improvements in outcome. However, they show that by performing myofascial release techniques before doing the articular release techniques, significant differences are obtained in pain scores, range of movement, and functionality. Thus they achieved better results when treating the soft tissues prior to joint mobilization. In a study by Suri et al. [14] muscle energy techniques were added to the Maitland mobilization techniques. Their results differ according to the expected goals. If an increase of range of movement is the desired goal, Maitland techniques will be more effective; on the other hand, for analgesic effects better results are achieved using muscle energy techniques.

Another study compared between Maitland mid-range and final-range mobilizations and Mulligan mobilizations. The range of motion increased with both techniques. The Maitland method achieved a higher maximal range of motion. The Mulligan achieved more improvements in scapula-humeral rhythm [15].

In 2007, Johnson et al. showed that performing a posterior glide combined with mobilizations, stretching, ultrasound, and exercise is an effective therapeutic intervention to treat external rotation deficits. These external rotation deficits are frequently present in patients with frozen shoulder. When performing the same maneuver together with an anterior glide, no increases in the shoulder external rotation range were achieved [16]. On the other hand, Joshi et al. in 2013 also obtained significant differences in terms of range of motion improvement and decrease in pain when anterior and posterior glides were combined with an exercise program and ultrasound [17]. The difference with this study illustrates that improvements are greater when anterior glide is performed when expecting better external rotation and decrease in pain. Vermeulen in 2006 found that at 12 weeks, improvements in range of motion and function were found in patients treated with the three mobilization techniques, especially with high grade of mobilization [18].

In general, and according to the existing evidence [6–8], a physical therapy program to treat frozen shoulder should include: exercises to increase both active and passive range of movement, capsular and muscle stretching, soft tissue manipulation, joint mobilization techniques, patient education about the disease, and a program for home exercises. It is critical that the patient is able to maintain or improve the goals achieved during the physical therapy sessions. The manipulations can be slowly progressed within the painfree zone, to avoid contractures and reflex spasms which will lead to greater stiffness and inflammation. The therapist must inform the patient that the mobilizations can cause pain. The patient must warn the therapist when reaching the pain limit. Due to the limitation of movement of the glenohumeral joint, there must be recognition that the scapula-thoracic joint can become dyskinetic. This results in an increased upper rotation and elevation of the scapula, generating a scapula-humeral rhythm disorder [10]. Therefore, adding scapula-thoracic exercises to the specific exercises for the glenohumeral joint has been recommended; scapula-thoracic exercises including scapular stabilization [11] work to achieve the goals of decrease in pain and increase in range of movement, and thus help to recover scapula-humeral rhythm [8].

General Goals of Physical Therapy

1. Decrease pain
2. Restore scapular mobility and position of the glenohumeral joint
3. Restore range of motion of the glenohumeral joint
4. Stabilize peri-articular muscles
5. Return the patient back to his/her activities of daily living
6. Educate the patient about the disease

It is important that during the exercise program, the therapist acts to ease the normal movement pattern, avoiding the presence of adaptive pathologic patterns [10].

We advice that exercises should begin with low and stable positions, progressing to higher levels. To protect the joint complex stability, the initial therapy consists of passive motion, progressing to active-assisted, then, finally reaching to active exercises, in all cases with and without resistance. Exercises should begin with a closed kinematic chain and later progress to an open kinetic chain.

Table 16.1 Revision of studies of physical therapy for frozen shoulder

Study and date	Type of study	Number of participants	Symptoms duration	Interventions and number of treatments	Results measurements	Follow-up	Findings
Doner et al. 2013 [12]	Randomized controlled study	40	$\geq$3 months	Moist hot towels (MHT), transcutaneous electric nerve stimulation (TENS), passive stretching vs. MHT, TENS, Mulligan techniques Number not described	Pain ROM (range of movement) Function Satisfaction	3 months	Both techniques (passive stretching and Mulligan techniques) were effective in pain reduction, and range of movement and function improvements. The group treated with Mulligan techniques was superior when comparing improvements achieved in terms of pain, range of movement, function and patient satisfaction
Johnson et al. 2007 [16]	Clinical randomized study	20	Not described	Ultrasound (US), anterior and posterior joint mobilization (direction according to group), upper limb ergometric exercise 6 sessions	Pain ROM external rotation	Not described	Both groups showed significant improvements in pain decrease Posterior versus anterior mobilization was more effective to achieve increases of external rotation in subjects with adhesive capsulitis

Celik D. 2010 [8]	Randomized study	39	Not described	TENS, cold pack, NSAIDs, articular range exercises, scapulothoracic exercises (group 2) 30 sessions (6 semanas)	Pain Function ROM	Not described	Both groups showed improvements in function, range of movement and pain decrease at the end of 6 and 12 weeks Improvements in range of movement achieved at 12 weeks were significantly greater in group 2 (scapulothoracic exercises) Scapulothoracic exercises contributed to decreased pain and range of movement improvements, when performed together with glenohumeral exercises, in patients with adhesive capsulitis
Deshmukh et al. 2014 [13]	Not described	30	Not described	Maitland mobilization, exercises (control group) vs. Maitland mobilization, miofascial release, exercises (experimental group) 9 sessions (3 weeks)	Pain Function ROM	Not described	Significant improvements in pain relief, function, and range of movement were achieved in both groups. When comparing results between groups, significant differences exist in terms of pain, function, and range of movement, when miofascial release is performed before Maitland manipulation

(continued)

Table 16.1 (continued)

Study and date	Type of study	Number of participants	Symptoms duration	Interventions and number of treatments	Results measurements	Follow-up	Findings
Suri et al. 2013 [14]	Comparative study	30	Not described	CHC, active exercises, Maitland mobilization (group 1) vs. CHC, Muscle energy techniques (group 2) 12 sessions (2 semanas)	Pain ROM	Not described	Results show that both techniques are useful to treat adhesive capsulitis, Maitland mobilization being more effective in active and passive movement increments, while muscle energy techniques were more effective in pain relief
Vermeulen et al. 2006 [18]	Randomized controlled study	100	≥3 months	High degree mobilization techniques vs. low degree mobilization techniques 24 sessions (12 weeks)	Function Active and passive ROM	12 months	By the end of 12 weeks, improvements in range of motion and function were found in patients treated with the three mobilization techniques. When comparing the effectiveness of the three mobilization techniques, the end of range and movement mobilization techniques achieved greater benefits in movement and function than mid-range mobilization After 3 weeks of mobilization techniques with movement, improvements in scapulohumeral movement strategies are achieved

Joshi et al. 2013 [17]	Not described	30	Not described	US, exercises, anterior sliding vs. US, exercises, posterior sliding 6 sessions (2 weeks)	Pain ROM external rotation	Not described	The authors found that combining anterior sliding with US and exercises was superior for the treatment of external rotation deficits in patients with adhesive capsulitis. Due to this, they conclude that this technique is very effective for pain decrease and shoulder external rotation range increase in patients with primary adhesive capsulitis
Yang et al. 2007 [15]	Not described	28	3 months	Mid-range mobilization End of range mobilization Mobilization with movement exercises 12 weeks	Function ROM	12 weeks	By the end of 12 weeks, improvements in range of motion and function were found in patients treated with the three mobilization techniques. When comparing the effectiveness of the three mobilization techniques, the end of range and movement mobilization techniques achieved greater benefits in movement and function than mid-range mobilization After 3 weeks of mobilization techniques with movement, improvements in scapulohumeral movement strategies are achieved

(continued)

Table 16.1 (continued)

Study and date	Type of study	Number of participants	Symptoms duration	Interventions and number of treatments	Results measurements	Follow-up	Findings
Diercks et al. 2004 [9]	Prospective cohort study	77	≥3 months	Support therapy and supervised neglect vs. passive mobilization and passive stretching Number not described	Function	2 years	During follow-up, the authors reported significant differences between both groups. For this study, the supervised neglect treatment was superior in the benefits compared to passive mobilization and passive stretching
Shishir et al. 2013[19]	Not described	36	4 weeks–6 months	Home exercises Minimum 5 sessions per day	ROM	2 years	Patients with a home exercise program achieved good results at the end of the 2 years follow-up The study concludes that an invasive home exercise program protocol is effective in achieving early improvements in pain decrease and disability in patients with adhesive capsulitis

16.2 Practical Advices

Within the recommendations and according to the disease phase of the patient we can see that in the freezing phase the main goal will be a decrease in pain, patient education about the disease, and enhancing a good postural behavior. Other authors also recommend the interruption of the inflammatory process [11]. Assisted passive and active movements shall be performed so as to maintain and increase the range of movement of the glenohumeral joint, together with soft tissues manipulation [7]. If there is no pain relief or increase in range of movement in the first 2 weeks, the recommendation is to revaluate so as to find a better way of pain relief and a good response to physical therapy.

During the frozen phase, normally there will be pain decrease together with stiffness increase; Therapeutic intensity can be increased by using joint mobilization techniques combined with capsular and affected muscles stretching [7]. The greatest stiffness occurs in abduction and external rotation (the motion with the longest time-to-recover), then comes the internal rotation. The techniques performed include release of scapular trigger points (infraspinatus, teres minor, latissimus dorsi, pectoralis major, and subscapularis management), scapular, clavicle, and glenohumeral mobilization in postero-inferior direction, in rotation work, scapular stretching and assisted stretching. Exercises that improve scapular stabilization, strengthening under low loads, isometric-concentric rhomboid activation are performed (Figs. 16.1 and 16.2). Proprioceptive and neuromuscular work to enhance postural control is also initiated. Using the current technology, the patient may be instructed to set an alarm every 2 hours to remember postural correction; this has had very good response, for it is usual that the patient forgets the corrections taught in the previous session.

In the thawing phase, exercise intensity advances and range of movement increases so as to achieve the maximum functionality and range of movement in all planes and also to strengthen the peri-articular muscles. The proposed techniques include joint and scapular mobilization, articular distraction, articular glides, and capsular stretching [7]. With respect to capsular stretching, it has been shown that prolonged stretching under light loads is more effective than under heavy loads [9].

In addition, the need for a home exercise program is critical. This will maintain the advances achieved and avoid the setbacks during treatment [19].

Fig. 16.1 Passive external rotation and release of the subscapularis muscle

Fig. 16.2 Glenohumeral joint glide while the patient performs active external rotation

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Oral Medication and Intra-articular Injection: Strategies and Results

17

Ronald L. Diercks and Francisco J. Vergara

17.1 Methods

In a literature search performed in January 2014, 33 publications were found, and the abstracts reviewed. Twenty-four were included in this review.

17.2 Results

There are several interventions used to treat frozen shoulder including supervised neglect, nonsteriodal anti-inflammatory drugs, oral steroids, physical therapy, intra-articular corticosteroid injections, manipulation with or without anesthesia, or surgical intervention [4, 5, 8, 10, 12].

NSAIDS are one of the most common interventions in treating frozen shoulder. The evidence for this treatment is lacking [8]. Only one comparative study was found [9] in which treatment with oral analgesics was inferior to hydrocortisone injection in the joint and exercises, hydrocortisone in the bicipital groove and exercises, and heat and exercises. No further studies were found.

> There is evidence that treatment of frozen shoulder with only NSAIDs has no effect on the natural course of frozen shoulder.

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Oral steroids were first used in the 1950s and showed an expedited recovery [5]. Later studies came up with different results. In 2009, a Cochrane review by [4] included five different, small, RCTs comparing oral steroids with placebo, with no treatment, with intra-articular steroids or manipulation under anesthesia and intra-articular steroids with or without oral steroid. Two trials compared oral steroids and placebo treatment. One trial compared oral steroids and no treatment. No conclusions could be drawn from the other two trials due poor quality. In the other three trials different doses of oral steroids were used: 12.5 mg cortisone acetate every 2 days for 4 weeks [2] ($N=32$), 10 mg prednisolone for 4 weeks followed by 5 mg for 2 weeks [3] ($N=40$), 30 mg prednisolone daily [4] ($N=49$). There is "Silver" level evidence that a short course of oral steroids for frozen shoulder may be of significant short-term benefit compared to placebo or no treatment, but the effect is not maintained at 6 weeks. However, Binder and Buchbinder suggested that significant improvement may have continued and increased with a more prolonged course of therapy and/or more gradual withdrawal of treatment.

There is evidence that oral corticosteroid use decreases pain and has a positive effect on ROM in frozen shoulder in the short term (6 weeks).

A longer course of oral corticosteroids could have a longer lasting effect on pain and ROM in frozen shoulder.

Intra-articular corticosteroid injection is a common intervention in treating frozen shoulder. There are different techniques, anatomical sites, and dosages for these injections. Favejee reviewed three high-quality RCTs comparing steroid injections with placebo. All suggested that intra-articular injections were significantly superior to placebo [8].

A Cochrane review [5] compared 13 RCTs to determine the efficacy and safety of corticosteroid injections in the treatment of adults with frozen shoulder. The number, site, and dosage of injections varied widely between studies. They were unable to pool the results of any trials comparing intra-articular steroid alone or in combination with subacromial steroid injection to placebo or no treatment because of the wide differences in the study designs, interventions, and outcome measurements.

Based upon the findings of these studies they had difficulty to draw any firm conclusions about the value of intra-articular steroid injection for frozen shoulder. There is a suggestion that intra-articular steroid injection may be beneficial in the short term for frozen shoulder but the effect may be small and not well maintained.

In 2005, the British Journal of General Practice published a systematic review to determine the utility of multiple intra-articular corticosteroid injections [14]. Nine RCTs were included from which four were rated as high quality. They concluded that multiple injections were beneficial until 16 weeks from the date of the first injection. Up to three injections were beneficial, with limited evidence that four to six injections were beneficial. No evidence was found to support giving more than six injections. Yoon [16] published the results of an RCT comparing different doses of corticosteroid injections in a single injection: 40 mg triamcinolone acetonide, 20 mg triamcinolone acetonide, or placebo. They concluded there was no difference in outcome between lower and higher doses of corticosteroids. Oh [13] concluded in an RCT that glenohumeral steroid injection was not superior to a subacromial injection.

Intra-articular corticosteroid injections can be performed blind (landmark-guided) and with image guidance. Soh [15] reviewed two RCTs comparing ultrasound-guided versus blind injections. There was a significantly greater improvement after image-guided corticosteroid injections compared to blind injections. This result should be interpreted with some caution due the limited number of participants.

The last systematic review was done by Griesser et al [9]. It included eight level I-II evidence-based RCTs with a total of 406 participants. The study quality was found poor in seven of eight studies. They hypothesized that intra-articular corticosteroid injections would result in greater improvements in terms of pain and passive range of motion in the short term but that these short-term improvements would equalize with time, resulting in equivalent outcomes for all treatment groups in the long term. This hypothesis was confirmed although they emphasize the poor methodological characteristics of the studies that were analyzed (Table 17.1).

There is evidence that intra-articular steroids have a short-time effect on pain. Repeated injections can be beneficial, with a maximum of three.

The effectiveness of an image-guided intra-articular injection is more predictable than that of a “blind” injection.

Table 17.1 Evidence Tables

Reference	Level of evidence	Study type	No. of patients	Inclusion criteria	Intervention	control	Follow-up	Outcomes	Results	Remarks
Dehghan et al. [6]	3	RCT	57	Frozen shoulder and diabetes mellitus	NSAID while the latter group were undergone intra-articular corticosteroid injection		24 weeks	Both intra-articular corticosteroid and NSAID are effective in treatment of adhesive capsulitis and there is no significant difference between efficacies of these two treatment modalities in diabetic patients		
Arroll and Goodyear-Smith [1]		Meta-analysis	7 RCTs	Shoulder pain	Corticosteroids vs. placebo and three for corticosteroids vs. nonsteroidal anti-inflammatory drugs			Subacromial injections of corticosteroids are effective for improvement for rotator cuff tendonitis up to a 9-month period. They are also probably more effective than NSAID medication. Higher doses may be better than lower doses for subacromial corticosteroid injection for rotator cuff. tendonitis		

Buchbinder et al. [5]	1	Cochrane review	13 RCTs				Intra-articular injection for adhesive capsulitis may be beneficial although their effect may be small and not well-maintained	
Buchbinder et al. [4]	1	Cochrane review	5 trials180				There is "Silver" level evidence that oral steroids provides significant short-term benefits in pain, range of movement of the shoulder and function in adhesive capsulitis but the effect may not be maintained beyond 6 weeks	
Favejee et al. [8]	1	Systematic review	5 cohrane reviews and 18 RCT's	The study included patients with frozen shoulder; (2) the disorder was not caused by an acute trauma or systemic disease; (3) an intervention for treating frozen shoulder was evaluated; (4) results on pain, function or recovery were reported			Strong evidence for the effectiveness of steroid injections	Most of the included studies reported short-term results, whereas symptoms of frozen shoulder may last up to 4 years

(continued)

Table 17.1 (continued)

Reference	Level of evidence	Study type	No. of patients	Inclusion criteria	Intervention	control	Follow-up	Outcomes	Results	Remarks
Soh et al. [15]	2	Meta-analysis	2 RCTs					Image-guided (ultrasound) injections had statistically significant greater improvement in shoulder pain and function at 6 weeks after injection. Image-guided (ultrasound) corticosteroid injections potentially offer a significantly greater clinical improvement over blind (landmark-guided) injections in adults with shoulder pain		
Shah and Lewi [14]	1	Systematic review	9 RCTs					Multiple injections were beneficial until 16 weeks from the date of the first injection. Up to three injections were beneficial, with limited evidence that four to six injections were beneficial. No evidence was found to support giving more than six injections		

Oh et al. [13]	3	RCT	71	Primary frozen shoulder	Glenohumeral injection	Subacromial injection	12 weeks	The GH steroid injection was not superior to an SA injection for patients with primary frozen shoulder even though injection at the GH joint led to earlier pain relief compared with the SA injection	
Yoon et al. [16]	3	RCT	53	Primary adhesive capsulitis in the freezing stage	Ultrasound-guided intra-articular injections with 40 mg triamcinolone acetonide (high-dose group, $n=20$), 20 mg triamcinolone acetonide (low-dose group, $n=20$), or placebo ($n=13$)			No significant differences between the high- and low-dose corticosteroid groups, indicating the preferred use of a low dose in the initial stage	

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Joint Distension

18

Francisco J. Vergara and Ronald L. Diercks

The clinical use of capsular distension was first described by Andren and Lundberg in 1965 [1]. The results have been controversial, especially due to a published systematic review performed by Buchbinder et al. in 2008; with a “silver” level of evidence. This report concludes that joint distension with steroids and saline improves pain relief, range of motion, and functionality of the glenohumeral joint, but a superiority of this therapy compared to other therapeutic options cannot be proven [2]. In 2004, the same authors published a controlled and randomized study comparing joint distension with saline and steroids, with a total volume of 30–90 ml versus a placebo group in which only a standard 7 ml arthrography and no dilation was performed. They showed improvement in pain relief, function and range of motion at 3 and 6 weeks compared to the control group. However at 12 weeks follow-up, only the functional improvement was maintained, as assessed by PET score [3]. In 2007, they published a study about the efficacy and cost-effectiveness of physical therapy after capsular hydro-dilation with steroids. At 6 months, in both groups, significant improvement in pain relief control, function, and quality of life were achieved. In the group with physical therapy, the active range of motion was greater [4].

In 2008, Tveita published a controlled and randomized study comparing intra-articular steroid injection versus saline plus steroids in a schedule of 3 doses twice a week. He found no significant differences between both groups. Assessment was performed at 6 weeks, knowing that steroid infiltration by itself produces

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improvement in frozen phase. The volumes used were 10 and 20 cc, which are very low dilation volumes [5].

Castro performed a study by assessing joint dilation with a 40–80 ml volume of saline to 24 patients with frozen shoulder, followed by intensive physical therapy. He achieved significant improvements in range of motion, constant score, and satisfaction level at 30–60 days [6]. The limitations of this study include: the lack of post-procedure evaluation; failure to separate the two treatment methods, dilation and physical therapy; and absence of a control group. These limitations are found in most of the studies reviewed, with the exception of Buchbinder in 2004.

In a prospective study, Clement showed the effectiveness of joint distension in the short and long term, both in diabetic and non-diabetic patients. This study assessed OSS, VAS, and ROM before the procedure and after, 2 days, 1 month, and 14 months, finding significant differences in the three measured parameters, and not finding differences between diabetics and nondiabetics [7].

To determine if joint distension is equally effective for primary or secondary shoulder stiffness, Watson performed a study assessing hydro-distension plus physical therapy, in patients with frozen shoulder and secondary shoulder stiffness due to a rotator cuff disease. The study found similar improvement in mobility and decrease in pain in both groups after the therapy and even up to 24 months [8].

The study performed by Piotte shows that when performing three arthrographic distensions with steroids at 3-week intervals, significant differences were achieved after the first two, but not with a third procedure [9].

Quraishi published a study comparing hydro-dilation versus manipulation under anesthesia, achieving significant improvements with hydro-dilation in pain management and constant score in patients undergoing joint distension, and similar improvements in range of motion in both groups, when assessed at 6 months. Patients also showed a 94% satisfaction with hydro-dilation versus 81% satisfaction with manipulation under anesthesia, the former being safer and cheaper [10], Another paper showed that manipulation under anesthesia achieves a statistically significant degree of abduction at 16 months, but not so in the severity of pain and the amount of external rotation [11].

The advantage of performing radiologic-guided glenohumeral joint distension in frozen shoulder, the procedure used in most of the published studies, is to observe the moment of capsular tear when the contrast medium reaches the surrounding tissue and, thus, have a control over the volume to be used. On the other hand, this technique allows for radiation exposure and the use of iodide contrast medium, with the potential risks to the patient and, to the operator. Ultrasound-guided procedures represent a safer and more efficient option, as shown by Shin [12] and Oh [13]. This is currently performed by interventional radiologists, and could also be performed by trained orthopedists (Fig. 18.1).

According to our experience, the best moment to perform hydro-dilation would be when recovery of mobility plateaus in physical therapy and in the frozen phase of the disease.

In conclusion, joint distension is a recommended procedure to improve the frozen shoulder during the frozen phase, whether primary or secondary, both in

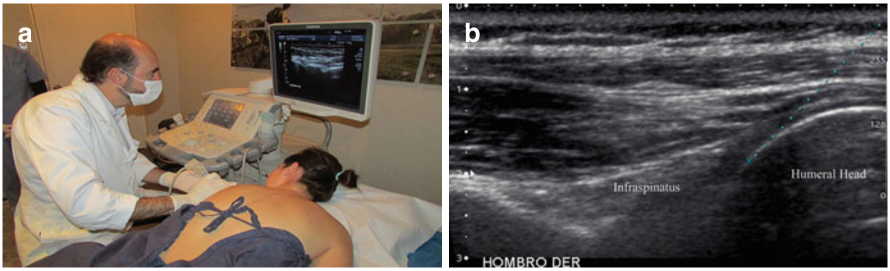

Fig. 18.1 (**a**) Patient's position in ultrasound-guided joint distension (Radiologist Dr. J. Rosales) (**b**) Ultrasound imaging, the needle trans-infraspinatus

diabetic and non-diabetic patients. The use of steroids is not clear. There is some evidence that it may not be necessary, however, most of the studies do use steroids for the procedure. It is recommended for those patients who no longer have improvement in glenohumeral range of motion while in physical therapy. A maximum of two distension procedures have proven to be more effective than a larger number of them.

There is "silver" evidence that joint distension with steroids and saline improves pain relief, range of motion, and functional of the glenohumeral joint. The effect is seen both in diabetic and nondiabetic patients.

The optimal moment for hydro-dilation seems to be when recovery of mobility during rehabilitation plateaus.

Studies advise that a total amount of 40–80 ml volume of physiologic saline should be injected.

There is no evidence that adding steroids has an additive effect.

There is no beneficial effect shown for more than two sessions of joint hydro-dilation.

There is no proven superiority of joint hydro-dilation over other therapeutic options.

Table 18.1 Revision of studies of joint distension for frozen shoulder

Study and date	Type of study	Number of participants	Symptoms duration	Interventions and number of treatments	Measures of results	Follow-up	Findings
Clement et al. 2013 [7]	Prospective study with a long-term cohort	51	>6 weeks	Arthrographic distension as primary intervention Number: not described	Pain Function ROM	14 months	Study includes 12 diabetic patients who achieved similar improvements as nondiabetics. Comparing pre- and post-distension conditions, improvements in function and pain were observed. Average increments in ROM were of 39.3° for flexion, 55.2° for abduction, and 19.5° in external rotation at first month
Watson et al. 2007 [8]	Clinical trial	53	6 weeks	Hydrodilation Physical therapy Number: not described	Function ROM	2 years	In patients with glenohumeral joint contracture, hydrodilation and physical therapy increase functional capacity and ROM in most individuals. These benefits persist in the long term. The greatest changes in external rotation happened 3 days after the procedure for external rotation, between 3 days and 1 week for internal rotation, abduction achieved less improvement, being between the first week and 3 months. It could be observed, however, that mild deficits persist after 2 years

Study and date	Type of study	Number of participants	Symptoms duration	Interventions and number of treatments	Measures of results	Follow-up	Findings
Ng et al. 2012 [11]	Randomized prospective study	28	Not described	Under anesthesia mobilization vs. capsular distension Number: not described	Pain ROM	6 months	At 6 months follow-up; under anesthesia mobilization achieves better increments in shoulder abduction than capsular distension. No significant differences between the study groups were observed in external rotation and pain relief When analyzing capsular distension by itself 6 weeks after the procedure, significant changes were found in the abduction ranges and external rotation
Quraishi et al. 2007 [10]	Prospective controlled trial	36	37, 4–39, 8 weeks	Under anesthesia mobilization vs. hydrodilation	Pain Function ROM Satisfaction	6 months	At 6 months, ROM increased, without significant differences between both groups At the end of follow-up, 94% of patients were satisfied or very satisfied after hydrodilation therapy, compared to an 81% satisfaction of those treated with under-anesthesia mobilization The authors conclude that although most of the patients were successfully treated, the results were significantly greater on those who received hydrodilation compared to under-anesthesia mobilization. Thus, when faced with nonresponding conservative therapies, the authors recommend hydrodilation techniques

(continued)

Table 18.1 (continued)

Study and date	Type of study	Number of participants	Symptoms duration	Interventions and number of treatments	Measures of results	Follow-up	Findings
Piotte et al. 2004 [9]	Not described	15	≥3 months	Arthrographic distension 3 procedures Exercises at home	Pain Strength Function ROM	Not described	Significant increments were observed in all measurements, which were greater after the first arthrographic distension procedure, beneficial effects decreased by the third procedure Study concludes that by performing 2 arthrographic dilation procedures in combination with steroids and a program with exercises at home, function improves thus decreasing disability. Also, the study shows that no further benefits are achieved by adding a third procedure
Tveita et al. 2008 [5]	Randomized trial	76	3 months–2 years	Hydrodilation, steroids vs. steroids 3 injections	Function ROM	6 weeks	Both groups achieved significant increments when comparing basal measurements. The study does not detect important effects when performing 3 hydrodilation procedures plus steroids compared to 3 only steroid injection procedures

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Part VI

Surgical Treatment

Manipulation Under Anesthesia for Frozen Shoulders

19

Eiji Itoi and Hiroshi Minagawa

19.1 Introduction

Frozen shoulder is believed to be a self-limited disease, and most of the patients with frozen shoulder recover from pain and limited range of motion in 1 or 2 years. However, Reeves showed a complete recovery of shoulder range of motion in only 39 % of 41 patients [45]. Shaffer et al. also showed persistent pain and rigidity in 50 % of the patients after 7 years of follow-up [48]. In cases with residual restriction of motion after conservative treatment, manipulation under anesthesia (MUA) may be indicated. MUA was first described by Duplay in 1872 [7]. Manipulation is usually performed under general anesthesia, but local anesthesia or interscalene block is also used as an outpatient treatment. In this chapter, the method of manipulation, including anesthesia, complications, and outcome, is described with recent evidence.

19.2 Anesthesia

General anesthesia has been widely used when performing MUA [12, 15, 17, 31]. For some surgeons who prefer to perform some surgical intervention such as arthroscopic capsular release [5] or mini-open coracohumeral ligament release [8]

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in addition to MUA, general anesthesia may be more preferable. However, considering the risk of general anesthesia and the cost to be hospitalized, local anesthesia or interscalene block has been advocated.

Loyd and Loyd reported a use of local anesthetics with steroid when performing an arthrography [30]. During arthrography, they added a gentle passive manipulation. Two out of 31 patients who did not benefit by the procedure underwent manipulation under general anesthesia. Ekelund and Rydell performed joint distension, followed by injection of steroid and local anesthetics, and manipulation [9]. Van Royen et al. performed arthrography, followed by injection of local anesthetics (10 ml of 0.5 % bupivacaine) and steroid, joint distension using 50 ml of saline, and finally manipulation [53]. Othman et al. used local anesthetics (10 ml of 0.5 % bupivacaine) and steroid in the early part of their series [40]. They later increased the dose up to 20 ml of 0.5 % bupivacaine for better pain control. Khan et al. used a suprascapular nerve block together with the intra-articular injection of local anesthetics and steroid [25]. They concluded that adding the suprascapular nerve block to the intra-articular pain block was simple, safe, cost-effective, and minimally invasive. Local anesthesia into the glenohumeral joint alone may not be enough to achieve pain-free status with sufficient muscle relaxation [30].

Roubal reported the use of interscalene block with nerve stimulator, followed by injection of 30 ml of carbocaine to C5 and C6 roots, which brought sensory and motor paralysis within 30 min [47]. The patient was discharged from the recovery room within 1 h, and went to the physical therapist's office to have manipulation. The same technique was reported by others [4, 41, 42]. Yilmazlar et al. injected a 30 ml of local anesthetics into the interscalene brachial plexus [57]. Following the injection, a 20-G catheter was inserted and kept in place to be used as patient-controlled anesthesia. This was done 30 min before rehabilitation, twice a day, for 15–20 days of hospitalization. More recently, Malhotra reported continuous interscalene nerve block using a catheter and injection pump for 3 days (ropivacaine group and saline group) in a double-masked fashion after manipulation [34]. Both range of motion and pain relief were better achieved in the ropivacaine group. Minagawa reported the use of ultrasonography to perform interscalene block, followed by manipulation (see the first half of Video 19.1) [37]. According to his report, both interscalene block and manipulation were performed by the same surgeon.

19.3 Methods of Manipulation (See the Second Half of Video 19.1)

MUA is performed with the patient in supine position. The scapula is stabilized with one hand. The other hand is recommended to hold the most proximal portion of the upper arm to make the lever arm as short as possible in order to avoid serious complications [29, 40, 44]. First, the arm is gently elevated until the audible and/or palpable crepitus is heard. During this procedure, the anteroinferior to inferior capsule is ruptured parallel to the glenoid rim. Next, the arm is rotated very gently into external rotation while holding the supracondylar level to avoid injury to the elbow ligaments, and then the arm is brought back to adducted position while keeping in external rotation. This causes a tear of the anterior capsule starting from inferior to superior direction. Then, the arm is brought to cross-body adduction to rupture the posterior capsule, followed by internal rotation in flexion to rupture the posteroinferior capsule. Finally, the arm is extended and rotated internally to further release the adhesion around the subacromial bursa. This may be repeated until the range of motion becomes similar to that of the contralateral shoulder. The first step is always elevation to create a tear of the anteroinferior capsule. The next step is either (1) internal rotation to extend the tear to posterior direction, followed by external rotation to extend the tear to anterior direction [10, 29], or (2) the opposite order; external rotation first to extend the tear to anterior direction, followed by internal rotation to extend the tear to posterior direction [9, 13, 51].

Some have proposed a specific technique to avoid complications. Hollis et al. manipulated using Codman's paradox [19]. First, the arm is placed at the side with the arm in internal rotation. Next, the arm is elevated 180° in the sagittal plane. Finally, it was lowered in the coronal plane to achieve full external rotation. Using this technique in 76 patients, they experienced no fractures or other complications. Other investigators used translational manipulation technique [41, 42, 47], in which they applied a linear translational force to the proximal humerus rather than a rotational torque to avoid complications. This translational force is applied in the inferior, posterior, and anterior directions.

19.4 Combined Procedures

Some surgeons prefer to use joint distension in combination with MUA [9, 30, 53]. The reason for this was that they first performed arthrography to make a diagnosis of frozen shoulder. With the needle tip still inside the joint, it might be beneficial for the patients to undergo joint distension, which is known to relieve pain. However, as joint distension was not enough to improve shoulder range of motion, they added MUA after joint distension. Ekelund and Rydell mentioned that they used only MUA before 1982 and the combined treatment after 1982 [9]. Though they did not show any data, they mentioned that the outcome seemed to be better in the combined treatment.

Others recommend the combination of MUA and arthroscopic capsular release [5, 6]. Ogilvie-Harris et al. suggested the use of arthroscopic capsular release before MUA in order to reduce the risk of complications during MUA [39]. On the other hand, those who perform MUA before capsular release believe that it would be much easier to perform arthroscopic capsular release procedure after the manipulation because there is more space in the glenohumeral joint for handling the instruments after the manipulation [5, 6, 52]. De Carli et al. noted that they combined these two procedures together in order to decrease the risk of complications and the risk of recurrence [6].

Eid reported a use of mini-open coracohumeral ligament release combined with MUA [8]. The background of this procedure is that MUA has serious potential complications, whereas arthroscopic capsular release is much safer but requires special instrument, experience, and high cost. Instead of using an arthroscope, he used a mini-open procedure with a 3-cm skin incision to release the coracohumeral ligament without any special instruments, followed by gentle manipulation to achieve satisfactory outcome, while avoiding the risk of complications.

19.5 Timing of MUA

Most authors perform MUA when the symptoms do not improve after a certain period of conservative treatment. Others perform MUA after failed joint distension treatment [20]. Minagawa recommended the use of MUA without any delay once the diagnosis of frozen shoulder is established based on the concept that the sooner the MUA, the shorter the patient's suffering would be [37].

The effect of timing of treatment has had little attention in the literature to date. Kessel et al. reported that a greater degree of improvement was achieved in patients who were symptomatic for more than 6 months before MUA compared with those who had a shorter duration of symptoms. [24] This may indicate that MUA may not be very effective in the freezing phase when the inflammation is strong. Neviaser and Neviaser also reported that any surgical intervention during this phase of inflammation is likely to aggravate the patient's symptoms and restricted range of motion [38]. Flannery et al. reported that 120 patients who underwent early MUA with symptoms for less than 9 months showed significantly better improvement in external rotational range of motion and Oxford Shoulder Score (OSS) compared with 25 patients who underwent late MUA with symptoms for more than 9 months [13]. The average premanipulation range of motion was 69° in flexion and 54° in abduction, suggesting that these patients were in their "frozen phase." A more recent retrospective analysis of 246 patients treated with MUA in their frozen phase revealed that the duration of symptoms and the OSS or the change in OSS had no correlation [51]. In this study, the mean duration of presenting symptoms was 28 weeks, ranging from 6 weeks to 156 weeks. This study tells us that the timing of MUA has nothing to do with the outcome as long as the patients are in their frozen phase.

19.6 Outcome

In general, the outcome of MUA is very satisfactory: normal or near normal range of motion in 75–97 % [1, 15, 46], with little or no pain in 79–97 % [1, 11, 15, 46]. There are two studies with a long-term follow-up of average 15 years [11] and 23 years [54]. Both of these studies showed that MUA led to sustained improvement in shoulder motion and function.

Hill and Bogumill reported that 70 % of patients returned to work within 2.6 months of MUA, much sooner than the natural course of this condition would indicate [18]. It is effective in shortening the course: recovery time was 8–13 weeks [1, 15, 40, 46]. However, Lundberg had an opposite opinion, saying that MUA did not affect the natural history or the time course of disease [32]. A second MUA is reported to be required in 8 % of the cases when the residual pain and/or limited range of motion continue or recur [46]. The risk of recurrence ranges from 5 to 20 %, especially high among those with diabetes mellitus (DM) [16, 22]. A randomized clinical trial revealed that there was no difference between MUA and the home exercises [27]. A recent systematic review revealed that the data from high-quality RCTs of MUA were very limited [35]. In addition, there was considerable variability between the studies in how the procedures were delivered, making comparability between studies difficult (Table 19.1).

Sometimes, intra-articular steroid injection is used together with MUA. Kivimaki and Pohjolainen performed a randomized clinical trial comparing those with MUA alone versus those with MUA + steroid injection [26]. They did not find any significant difference between the groups. They concluded that steroid injection added nothing to the course of recovery after MUA. Hamdan and Al-Essa also compared MUA, MUA + steroid injection, and MUA + joint distension [16]. They found that MUA + joint distension showed better outcome than the other two groups.

19.7 Complications

Complications such as proximal humeral fracture [16, 43], glenoid fracture [33], shoulder dislocation [16, 50], and brachial plexus palsy [3, 36] have been reported. The proximal humeral fracture is reported to occur in 3–5 % of manipulation cases [16, 43]. Magnussen and Taylor used a short lever arm technique to apply force to the proximal humerus to avoid a humeral fracture [33]. They experienced a glenoid fracture instead in this case. They warned that surgeons should be aware of and alert patients to this potential complication even with use of a short lever arm technique. Numerous authors have reported no complications with this procedure [1, 11, 18, 40, 41, 46, 49, 53]. Thomas et al. performed MUA in 246 patients, in which there were no fractures or dislocations, no symptoms of acute rotator cuff tear, no neurological or other iatrogenic injuries [51]. Ibrahim et al. reported one case of vasovagal episode during the procedure among 42 cases [20]. Atoun et al. examined the integrity of the rotator cuff before and after MUA using ultrasonography in 33 patients [2]. They found no rotator cuff tear after MUA, and concluded that MUA had not been associated with rotator cuff tear.

In order to avoid serious complications, specific manipulation techniques to use short lever arm have been proposed [19, 40]. Some authors have recommended the use of translational manipulative force rather than conventional rotational manipulative force to reduce the risk of fractures [41, 42, 47]. Arthroscopic capsular release before manipulation may also decrease the risk of complications [39].

MUA creates a capsular tear, most commonly at the anterior and inferior portions of the capsule [29, 52]. This capsular tear makes it possible to gain an increase in the range of motion. Some additional damages such as SLAP lesions, partial rotator cuff tears, and anterior labral detachments were also reported [29]. Although there is no evidence that these iatrogenic articular lesions are of clinical importance, some prefer to use arthroscopic capsular release [29].

Table 19.1 Evidence table of manipulation versus other treatments (evidence level I and II only)

Patients	*N*	Age, years	Intervention	Study design	F/U, mos	Outcome	Evidence level	Reference	
	4 RCTs			Systematic review		No conclusion on cost-effectiveness	I	Maund et al. [35]	2012
Flexion <140°	24	51	MUA vs. MUA + steroid	RCT	4	Pain, ROM: no effect of adding steroid	II	Kivimaki and Pohjolainen [26]	2001
Increasing pain, stiffness	125	53	MUA + home exercises vs. home exercises	RCT	12	Pain, disability: no difference, flexion: better in MUA @ 3 months, but not @ 6 and 12 months	II	Kivimaki et al. [27]	2007
Flexion <140°, ER <30°		53							
Frozen phase	36	54.5	MUA vs. distension	RCT	2, 6	Pain, Constant score: better in distension, ROM: no difference	II	Quraishi et al. [44]	2007
		55.2							
Freezing phase	53	56.5	MUA vs. distension + steroid	RCT	24	Constant score, VAS, SF-36: no difference	II	Jacobs et al. [21]	2009
		57.0							

RCT randomized clinical trial, *MUA* manipulation under anesthesia, *ROM* range of motion, *ER* external rotation, *VAS* visual analog scale, *SF-36* Short Form-36

19.8 For Secondary Stiff Shoulder

Wang et al. compared idiopathic frozen shoulder versus posttraumatic stiff shoulder versus postsurgical stiff shoulder [55]. They found less improvement in pain and motion in postsurgical stiff shoulders. They also compared MUA for those with and without noninsulin-dependent DM [56]. They found no significant difference between them. Even though DM is a predisposing factor to frozen shoulder, noninsulin-dependent DM alone does not influence both the short- and long-term outcomes of frozen shoulder. Jenkins et al. also compared those with DM (39 shoulders of 36 patients, DM group) and those without (274 shoulders of 256 patients, control group) [23]. They found significant improvement in range of motion and OSS with no difference between the groups at early or late follow-up. In another type of secondary stiff shoulders, Leonidou and Woods performed MUA for secondary stiff shoulder following breast cancer treatment [28]. There were 7 shoulders of 7 patients in the breast cancer group and 274 shoulders of 256 patients in the control group. None of the patients in this study were diabetic. The mean preoperative OSS was 31 for the cancer group and 27 for the control group, improving to 43 for both groups. At the long-term follow-up, 71 % in the breast cancer group were satisfied. Due to recurrence, 42 % in the cancer group and 15 % in the control group had second MUA. They concluded that MUA was effective in patients with secondary stiff shoulder after breast cancer treatment.

19.9 Comparison with Other Treatments

Ogilvie-Harris et al. in their prospective cohort study performed arthroscopy in 40 patients with persistent stiffness and pain for at least 1 year despite conservative treatment: (1) first 20 patients underwent arthroscopy, followed by manipulation, and then arthroscopy; (2) second 20 patients underwent arthroscopic capsular release [39]. At 2–5-year follow-up, no difference in the range of motion, but arthroscopic capsular release group did significantly better in pain and in function: 15/20 (75 %) treated with arthroscopic capsular release had an excellent result compared with 7/18 (39 %) treated with MUA. DM patients did worse initially but there was no difference at final follow-up. A recent systematic review has concluded that the quality of evidence available is low and the data available demonstrate little benefit for a capsular release instead of, or in addition to, an MUA [14]. A high-quality study is required.

Sharma et al. compared MUA and joint distension in 32 patients [49]. They recommended joint distension because it was easy to carry out and gave better results than MUA. Quraishi et al. compared 18 shoulders of 17 patients in the MUA group, 20 shoulders of 19 patients in the joint distension group in their prospective randomized clinical trial [44]. Pain and Constant score were significantly better in joint distension group, but no significant difference in the range of motion. At final follow-up of 6 months, 94 % satisfied in the joint distension group compared with 81 % in the MUA group. Jacobs et al. also performed a prospective randomized clinical trial with 53 patients with frozen shoulder treated by MUA versus steroid injection with joint distension [21]. At 2-year follow-up, there were no significant differences in the outcome (Constant score, visual analog scale, Short Form-36). Therefore, they recommended steroid injection with joint distension as an outpatient treatment, which has the same clinical outcome as MUA.

Kivimaki et al. performed a randomized clinical trial comparing those treated with MUA followed by home exercises and those treated by home exercises alone [27]. There were 65 patients in the MUA group and 60 patients in the home exercises group. They did not find any significant differences at any time point in terms of pain and working ability. Although small differences were observed in the range of motion in favor of MUA, MUA does not add effectiveness to an exercise program performed by the patient. However, the inclusion criteria of this study were those less than 140° of elevation and less than 30° of external rotation. With these criteria, those with very subtle limitation in the range of motion must have been included, who had little benefit from MUA. These wide inclusion criteria might have affected the outcome of this study.

Manipulation under anesthesia was estimated to cost £1,446 (≈ US$2,455) and capsular release £2,204 (≈ US$3,742), both of which included rehabilitation physiotherapy. Arthrographic distension was estimated to cost approximately £114.84 (≈ US$195), depending on the choice of steroid injection [35]. However, given the paucity of economic evidence, it is not possible to make any conclusions regarding the most cost-effectiveness intervention [35].

Conclusions

Manipulation under anesthesia is an effective treatment option for refractory frozen shoulder. It is as effective as but less costly than arthroscopic capsular release. Serious complications such as a fracture should be avoided with use of a short lever arm with slow and gentle motion. High-quality evidence needs to be established.

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20 The Role of Arthroscopic Capsular Release

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20.1 Introduction

"Primary frozen shoulder syndrome" or simply "frozen shoulder" is a fairly common disorder characterized by pain and loss of range of motion [1]. This condition is unique and only occurs at the shoulder [2–4]. The etiology of frozen shoulder remains unknown and most patients receive conservative treatment based on steroid injections and physical therapy, achieving 80% remission rates [3, 5–8]. For refractory cases, most surgeons advocate either joint distension (JD), manipulation under anesthesia (MUA), or arthroscopic capsular release (ACR).

Due to the self-limited features of the frozen shoulder, the ISAKOS Upper Extremity committee recommends conservative measures before proceeding to surgical treatment. These measures should entail well-supervised, 6-month physical therapy program and 1–3 steroid injections. Monthly pain and range of motion assessment is essential. In case of no improvement with physical therapy, a maximum of three cortisone injections should be the last resort prior to ACR.

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Table 20.1 Advantages of the ACR with RF over MUA

1. Precise and controlled release of the capsule
2. Prevention of fracture and rotator cuff injury
3. Releases and capsulotomies performed with an RF* device
(a) Reduces intra-articular bleeding, preventing further adhesions
(b) Delays capsular healing allowing an end range of motion rehabilitation program

*RF radiofrequency

After failing a conservative trial, we can encounter two different scenarios: (1) residual pain with full recovery of shoulder motion, in which the patients are referred for pain management (usually with pregabalin); and (2) persistent shoulder stiffness and loss of motion as the main complaint, where ACR is indicated.

Arthroscopic surgical release can result in immediate relief of symptoms and faster recovery of shoulder function than any other treatment modality. Importantly, clinical improvement with ACR usually lasts many years [9, 10]. Long-term relief of symptoms in patients with frozen shoulder is similar whether they undergo either ACR or MUA; however, MUA may lead to fracture or intra-articular bleeding during forceful rotation, worsening adhesion in the shoulder (Table 20.1) [11, 12].

20.2 Surgical Anatomy and Preoperative Planning

There are four anatomic layers at the anterior shoulder girdle. The capsule with the glenohumeral ligaments and the subscapularis tendon constitute the first and the second layers. The coracohumeral ligament (CHL) forms the third layer, while the coracoid along with the coracoacromial ligament forms the fourth layer.

During the symptomatic phases of frozen shoulder, all layers act in concert to trigger symptomatology, whereas CHL appears to be primarily responsible for the severe decline of range of motion. CHL arises from the coracoid base and ends at the upper part of the bicipital groove (Fig. 20.1). This ligament constitutes the main target for surgical capsular release [13, 14].

There is considerable debate regarding the optimal amount of surgical release during ACR. Along with CHL and rotator interval release, some authors advocate releasing other structures including the subscapularis tendon, the inferior and posterior capsule, and the global capsule [15–17] to improve shoulder elevation, internal and external rotations. Pearsall et al. [18] reported that preoperative assessment of motion loss should guide the degree of capsular release.

Although the best timing to proceed with ACR is still controversial, failure of conservative measures and the patients' wishes for a quicker recovery should be taken into consideration during surgical decision-making. We usually defer surgical treatment of concomitant shoulder injuries such as cuff repair or biceps tenodesis until frozen shoulder recovery. Table 20.2 details the main indications for surgical release. Table 20.3 lists evidence-based data about current treatment modalities for frozen shoulder.

Coventry and colleagues described the psychological and neuropathic profile of frozen shoulder patients and coined the term "periarthritic personality". In line with

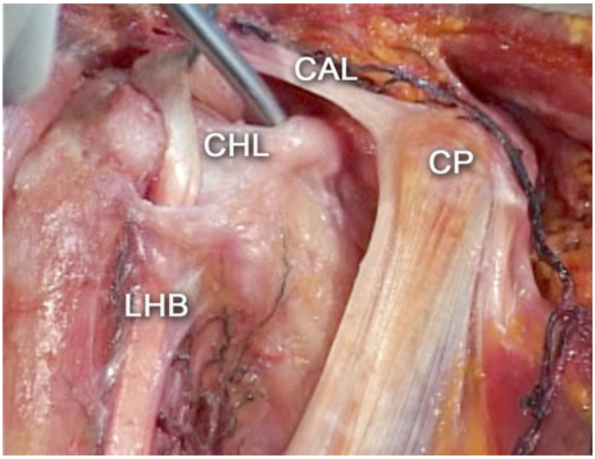

Fig. 20.1 Right shoulder. Anatomic dissection. Rotator interval anatomy. *CP* coracoid process, *CHL* coracohumeral ligament, *CAL* coracoacromial ligament, *LHB* long head of the biceps (Courtesy of Pau Golano, MD. Barcelona, Spain. With permission)

Table 20.2 ISAKOS UEC[a] consent indications for ACR

1. Failure of a well-performed rehabilitation program for a 6-month interval
2. Failure[b] after at least 3 steroid injections in a 6-month interval
3. Failure of less invasive treatments like joint distension or MUA
4. Severe frozen shoulder in patients with diabetes mellitus in whom steroid treatment is contraindicated
5. Patient wishes for a faster recovery

[a]*UEC* upper extremity committee
[b]Failure is defined as VAS for pain over 5/10 and range of motion reduced to less than 50 % of normal motion

this description, it is critical to document (see attached Surgical Technique Video) the assessment of baseline range of motion along with the observed postoperative gain to boost patients' confidence with the surgical procedure and commitment to the rehabilitation process.

20.3 Surgical Technique: Arthroscopic Capsular Release (See the attached Video with the full surgical technique)

Although many surgeons still perform ACR with the patient in the lateral decubitus position, the procedure is best performed with the patient in the beach-chair position under an interscalene block. By diagnostic arthroscopy, any synchronous injuries are first ruled out.

Typically, we observe synovial and capsular reddish at the glenohumeral ligaments, inferior part of the rotator cuff, and at the insertion of the biceps tendon (Fig. 20.2). Typically, there is obliteration of the triangle made by the biceps and subscapularis tendons secondary to proliferative synovitis.

Table 20.3 Clinical evidence regarding treatment

Year	Study size	Treatment comparison	Study design	Outcome	Time	Findings
2006 [19]	100	High-grade mobilization technique ($n=49$) vs. low-grade mobilization technique ($n=51$)	RCT	VAP, SDQ, shoulder rating questionnaire, SF-36	12 M	High superior to low-grade mobilization rehab techniques
2007 [20]	36	MUA ($n=16$) vs. hydrodilation ($n=20$)	RCT, single center	VAP, constant score, ROM	6 M	Hydrodilation superior to MUA
2007 [8]	28	End range motion (ERM) and mobilization with movement (MWM) vs. mid-range of motion (MRM)	RCT	Flex SF and ROM	3 M	ERM and MWM better than MRM
2007 [21]	125	MUA ($n=16$) and home exercises ($n=65$) vs. home exercises alone ($n=60$)	RCT, single center	SDQ, measures of active and passive ROM	12 M	Equivalent
2008 [22]	76	Intra-articular shoulder injections with ($n=39$) vs. without hydrodilation ($n=37$)	RCT, single center	SPADI, measures of active and passive ROM	1.5 M	Equivalent
2009 [5]	53	Intra-articular shoulder injections using steroid with distension ($n=25$) vs. MUA ($n=28$)	RCT, single center	Constant score, VAP, SF36 questionnaire	24 M	Equivalent
2010 [23]	74	ACR with ($n=32$) vs. without ($n=42$) release of inferior and posterior structures	RCT, single center, single-blind	ROM and VAP	28 M	Equivalent
2012 [9]	44	MUA and arthroscopic release ($n+23$) vs. intra-articular steroid injection ($n=21$)	RCT, multicenter	Constant and Murley, ASES, UCLA and SST evaluation scales	12 M	Equivalent but arthroscopic release leads to faster recovery
2012 [24]	45*	Intra-articular steroid injection plus home stretching exercises ($n=23$) vs. home stretching exercises alone ($n=22$)	RCT	VAP, ASES	2 M	Steroid injections lead to better outcomes in patients with diabetes
2012 [25]	70	Intra-articular hyaluronic acid injections plus physical therapy ($n=35$) vs. physical therapy alone ($n=35$)	RCT, single center	SDQ, SPADI, measures of active and passive ROM, SF-36	3 M	Equivalent

2013 [26]	191	Four groups. Steroid injection at subacromial space (Group I, $n=49$), at glenohumeral joint (Group II, $n=48$) Injections at both subacromial space and glenohumeral joint (Group III, $n=47$) or NSAIDS treatment (Group IV, $n=49$)	RCT, single center	VAP, measures of active and passive ROM, SDQ	3 M	Glenohumeral vs. subacromial steroid injections were equivalent and steroid injections better than NSAIDs
2013 [27]	53	High vs. low dose steroid injection vs. lidocaine injection	RCT, triple-blind	VAP, SPADI	3 M	Steroid injection superior to control injection. Equivalent between steroid high and low dose
2013 [28]	68	High (1:30,000, $n=22$) and low dose (1:10,000, =23) bee venom acupuncture vs. sham acupuncture ($n=17$)	RCT, double-blind	VAP, SPADI, measures of active and passive ROM	3 M	Bee venom acupuncture superior to control acupuncture. Equivalent between bee venom arms

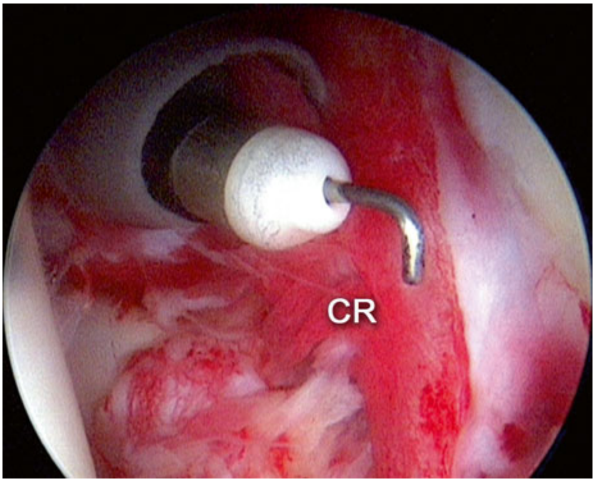

Fig. 20.2 Right shoulder. Arthroscopic view from the posterior portal. *CR* capsular reddish

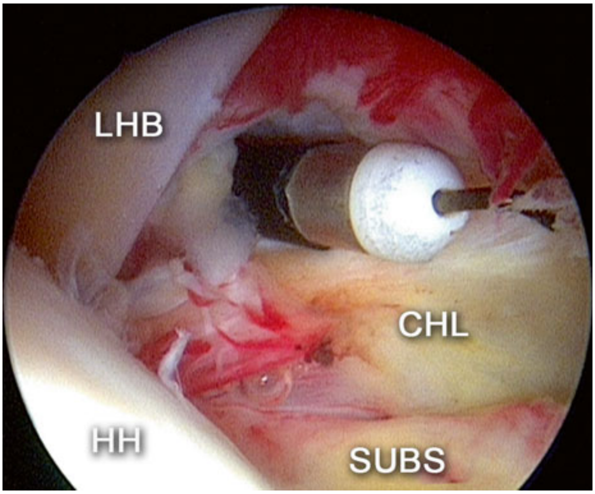

Fig. 20.3 Right shoulder. Arthroscopic view from the posterior portal. *CHL* coracohumeral ligament, *Subs* subscapularis tendon, *HH* humeral head, *LHB* long head of the biceps

With the scope at the posterior portal, the anterolateral portal is established using an outside-in technique just anterior to the biceps. Placing the anterior portal laterally creates an optimal angle to obtain access for the rotator interval release.

The rotator interval release and the anterior and posterior capsulotomies are performed with a radiofrequency device specially designed for coagulation, cut, and tissue vaporization.

First, we proceed with the rotator interval release, fully releasing the CHL (Figs. 20.3 and 20.4). Freeing the anterior structures enables complete external rotation of the shoulder.

We utilize a switching stick to move the scope to the anterior portal. The radiofrequency device is used through the posterior portal to release the posterior capsule and the posterior band of the inferior glenohumeral ligament. Sometimes a gentle

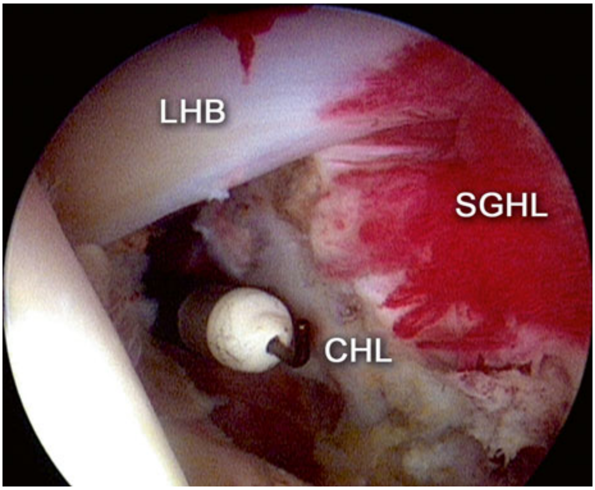

Fig. 20.4 Right shoulder. Arthroscopic view from posterior portal. *SGHL* superior glenohumeral ligament, *CHL* coracohumeral ligament, *SGHL and CHL* previously released by the radiofrequency device, *LHB* long head of the biceps

mobilization is needed to obtain full forward flexion and abduction because the cutting device does not reach a slight amount of the inferior capsule.

In addition, we perform resection of adhesions at the subacromial space and lateral bursae.

The patient obtains full range of motion with 180° of pooled external and internal rotations. Complete recovery of patient's abduction and forward flexion appears immediately after removing the drapes and the improvement is shown to the patient (see attached Surgical Technique Video).

A supervised rehabilitation program should start the same day of surgery with an active end range of motion and mobilization. During the first month after surgery, we implement an oral cortisone protocol (prednisolone 40 mg QD), tapering 10 mg per week.

20.4 Discussion

Arthroscopic capsular release emerges as a valuable tool for the management of patients with frozen shoulder that is refractory to conservative therapy. This procedure allows precise and controlled release of the capsule and ligaments, reducing the traumatic complications of a more forceful manipulation [29]. Although there is an improvement in elevation and abduction with MUA, restricted rotation is frequently a persisting problem. In addition, because bone is weakest during torsion, fracture risk is highest during this part of manipulation. Of note, surgeons' worries about fracture risk may impair MUA effectiveness.

The amount of capsular release required to improve patients' mobility continues to be a matter of debate. Data derived from randomized clinical trials support the concept of only releasing the rotator interval (CHL) and the anterior capsule (superior and middle glenohumeral ligaments together with the anterior band of inferior glenohumeral ligament), while the posterior capsule release is chiefly indicated in cases of glenohumeral internal rotations deficits [23, 30, 31].

In the setting of diabetes mellitus, patients with frozen shoulder frequently cannot receive adequate medical treatment, therefore requiring ACR more commonly. In these individuals, surgical results are less encouraging than in nondiabetic counterparts [11, 32].

Following the abovementioned technique, the patient is awake during ACR and usually tolerates well such a short procedure. In addition, conscious patients at the end of the procedure help considerably during arm movement and range of motion assessment.

Although long-term results are comparable between medical treatment and ACR, surgical therapy achieves faster satisfactory results (6 vs. 12 weeks) [18]. In addition, Le Lievre and colleagues demonstrated that early improvement in pain and range of motion after ACR persists for many years (7 years) [10].

Conclusions

Our conclusion after performing the technique described herein for many years is that ACR is straightforward, technically fast, and safe. Even though a conservative approach remains the first step in the treatment of frozen shoulder, full recovery usually takes many months or years. In this scenario, ACR should be entertained because it can shorten recovery time. In patients with refractory frozen shoulder, ACR remains a valuable option and leads to a quicker and consistent recovery.

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21 Postoperative Management and Rehabilitation

Brian Gilmer, Robert Drenning, Marcia Ready, Maurine Morris, and Dan Guttmann

21.1 Hospital Admission

The potential benefits of a brief hospital admission after capsular release are early aggressive targeted physical therapy, early initiation and compliant use of CPM, and improved postoperative pain control and management. The benefits of improved therapy compliance and pain management are offset by financial cost considerations. Those who advocate hospital admission have recommended a length of stay from 48 to 72 h, but no outcome data are available for these protocols [6, 15, 19].

21.2 Continuous Passive Motion (CPM)

Several authors have advocated the use of CPM; however, there is no evidence to clearly support routine clinical application. In a randomized controlled trial CPM has been shown to provide better pain reduction in the early phase of rehabilitation [5].

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A systematic review confirmed this finding but cited no difference in long-term range of motion or function [10].

21.3 Immobilization

Most authors recommend early discontinuation of sling immobilization. Some have even advocated brief immobilization in 90° of abduction and external rotation [6, 11, 15] Joint Active Systems (JAS) has a unique harness system that allows for a progressive stretch for 30 min two to three times per day. Unfortunately, data are lacking to recommend any particular regimen of immobilization.

21.4 Other Modalities

Other modalities that have been advocated in the treatment of frozen shoulder include ultrasound, acupuncture, bipolar interferential current, transcutaneous electromagnetic stimulation, laser and pulsed electromagnetic field therapy, but few studies are available assessing their efficacy in the postoperative shoulder [11, 17, 19, 15].

21.5 Pain Management

Postoperative pain control is critical for obtaining early ROM and improving rehabilitation compliance. Many options are available which can decrease dependence on opioids including oral or intra-articular steroid injections, interscalene nerve block or catheters, and more recently, liposomal bupivacaine injectable suspensions.

21.6 Outpatient Physical Therapy

Outpatient physical therapy is the mainstay of most postoperative treatment plans. Our protocol has been specifically designed to address the patient undergoing arthroscopic capsular release for both primary and secondary shoulder stiffness. This protocol is intended to give general guidelines for rehabilitation and recovery following surgical intervention for frozen shoulder. It consists of four phases of rehabilitation, including: early motion, active motion, strengthening, and advanced strengthening. It is important to note that phases will overlap to varying degrees. Treatment should always be modified according to each individual patient's needs.

21.6.1 Early Motion Phase

Ideally, treatment should begin within 24 h to minimize scar tissue formation. Goals for the early phase of rehab are to control pain and inflammation, prevent scar tissue and adhesions, and increase shoulder range of motion (ROM).

Initial treatments during this phase should include passive and active-assisted shoulder ROM in all planes. Stretch intensity should be gentle, nothing beyond the threshold of pain [8, 4]. Mobilizations to promote thoracic extension and scapular mobility are important to prevent impingement and restore normal scapulahumeral rhythm [12]. Glenohumeral (GH) joint mobilizations are beneficial in the early phase to restore capsular extensibility [14]. In addition to capsular mobility, it is important to concurrently address soft tissue restrictions of the anterior chest, shoulder, and scapula. Modalities may be used as needed to control pain and inflammation.

During this initial phase, patient education is very important to understand the surgical technique and enhance compliance throughout the rehab process. Patients are given exercises in this phase to address the above goals. These may include, but are not limited to, the following:

- Scapular active range of motion (AROM) to promote muscle activation, postural alignment [18], and kinesthetic awareness.
- Rotator cuff and deltoid isometrics to promote muscle activation and prevent atrophy.
- Pendulums: With the patient standing, the patient performs a lateral bend of the torso to allow the operative arm to abduct away from the body. A gentle circular motion is created by moving the torso. The patient is cued to relax the involved shoulder and utilize momentum created by the trunk. This promotes joint fluid movement, nutrient exchange, and prevents scar tissue formation.
- Supine cane external rotation: With the patient supine and the elbows flexed to 90°, the patient holds a cane in both hands and gradually uses the nonoperative hand to push the operative arm into gentle external rotation. This should be done passively and along the longitudinal axis of the humerus. The goal is to promote anterior capsular/shoulder flexibility and restore external rotation (ER), which is commonly the primary limitation in frozen shoulder [14]
- Table slides: With the patient seated in front of a table, the arm is placed on the surface of the table and the hand is advanced away from the body to achieve the desired stretch. Table slides can be performed in multiple planes and with the thumbs up (if ER mobility allows) to increase the subacromial space and decrease early impingement [2]. The goal is a passive stretch to increase overhead ROM (Fig. 21.1).
- Supine cane flexion: Similar to the supine cane external rotation stretch described above, the nonoperative hand guides the operative arm into forward flexion. The goal is to increase overhead motion with slightly increased muscle activation compared to table slides.

21.6.2 Active Motion Phase

When the above goals are being met, the patient can progress to the active phase of rehabilitation. This should begin as pain levels allow and as the patient is able to control compensatory movement patterns through his/her available range of motion.

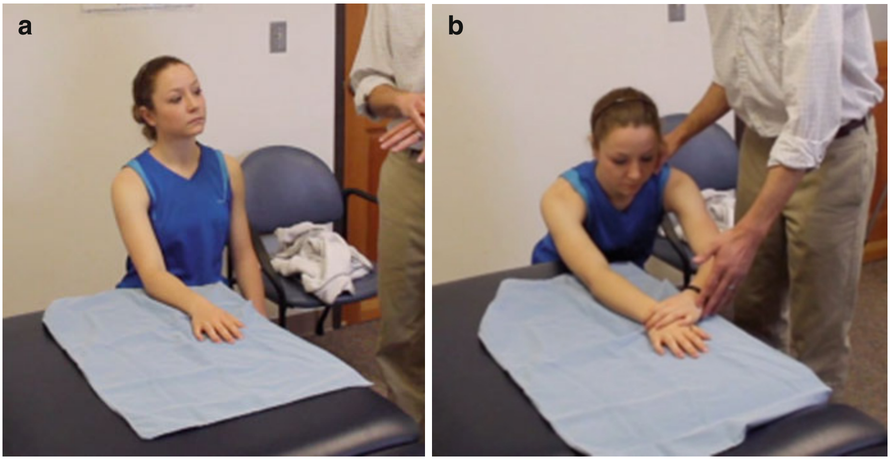

Fig. 21.1 Demonstration of table slides. (**a**) Proper alignment and scapular stabilization are confirmed prior to stretch. (**b**) Therapist guides terminal stretch to avoid impingement and scapular dsykinesis

This ensures that as new passive range of motion is achieved the patient utilizes this new range actively, which promotes functional carryover. Goals of the active phase are to control pain and inflammation, continue to increase passive ROM, prevent scar tissue formation, increase active ROM, and improve functional upper extremity (UE) use with activities of daily living.

Treatment during this phase should continue with early phase treatments as needed to achieve full ROM. In some cases, the surgeon may choose to modify parameters of the patient's home stretching exercises. For example, if pain is decreasing and a firm end-feel is achieved during a stretch, the patient may be instructed to perform low-load long-duration holds at the end range. This increases the total end-range time (TERT) to achieve plastic collagen deformation, which promotes greater gains in capsular mobility [1, 3]. Additional treatments may include rhythmic stabilization drills to restore neuromuscular control and reduce compensations during active movement.

Patients will be given exercises during this phase to achieve the above goals which include, but are not limited to, the following:

- Passive ROM progressions
- Sleeper stretch: To promote posterior capsular mobility it is important that the patient isolate the targeted tissues. If anterior shoulder pain is present, he/she may be creating impingement and the exercise should be stopped. A modified position is often used to target a greater portion of the posterior capsule [9]. Instead of being positioned directly lateral the patient may roll the trunk partly toward a supine position to place the shoulder in the plane of the scapula (Fig. 21.2).

- Doorjamb series: This exercise is a progression from the supine stretch for ER. It is performed standing in an open doorway. With the feet fixed on the ground and the palm against the door frame the torso is rotated until a stretch is achieved. Further progression can be obtained by gradually increasing the angle of arm elevation in the doorway.
- TV-watching stretch: This combined exercise emphasizes both ER and abduction end-range stretch simultaneously. Hands are placed on the forehead and the elbows are allowed to fall into horizontal abduction. When this becomes comfortable the patient progresses to hands behind the neck.

Fig. 21.2 Demonstration of the sleeper stretch. (**a**) Proper starting position is established with therapist assistance. (**b**) Patient utilizes unaffected arm to perform the stretch

- Cane flexion on a 45° incline: Performed the same as the supine cane flexion but the inclination increases the amount of muscle activation in a partially against-gravity position.
- Standing prayer stretch with exercise ball on wall: This exercise mimics a table slide, but in the standing position. Adding the anti-gravity position promotes greater muscle activation in a more functional position.
- Active ROM: This is begun in the scaption plane (full can position), as this will reduce the likelihood of impingement [16]. Again, the patient should be monitored for compensatory movement patterns or scapula dyskinesia.
- Prone shoulder extension, horizontal abduction (thumb up), and forward flexion (thumb up): These exercises are done to promote active GH elevation, as well as scapula stabilization and control. Emphasis should be placed on mid/low trapezius activation, while minimizing excessive upper trap contraction in order to decrease the "shrugging" that is commonly seen in this patient population.
- Side-lying ER and horizontal abduction (without resistance): The goal of this exercise is to increase rotator cuff activation. Placing a towel roll/bolster under the arm increases the subacromial space and improves supraspinatus activation [7].

21.6.3 Strengthening Phase

The third phase of postoperative rehabilitation for frozen shoulder is the strengthening phase. The patient may progress to this phase as pain levels allow, and if active ROM nearly equals passive ROM with good quality movement pattern. Goals are to continue to address passive and active mobility as mentioned above, while also improving the strength and endurance of the shoulder girdle musculature. Any or all of the above treatments should be continued as needed to achieve full shoulder mobility. Additional exercises to promote closed kinetic chain (CKC) and open kinetic chain (OKC) shoulder strength may include, but are not limited to the following:

- Internal and external rotation using elastic resistance band: This is done to increase rotator cuff strength and decrease impingement with AROM.
- Wall push-up: In this exercise the patient is standing and performs a standard push-up against the wall. This position allows for CKC GH stabilization and serratus anterior strengthening to promote good force coupling and minimize deviations with active movement [13].
- Rowing: The patient is cued to avoid excessive GH extension (which causes anterior tipping of the scapula and increases impingement), as well as excessive upper trapezius activation.
- Side-lying ER and horizontal abduction, as well as prone GH extension, horizontal abduction with resistance.

21.6.4 Advanced Strengthening Phase

As the patient gains functional active ROM and is pain-free with the above strengthening program, he/she may progress to the advanced strengthening phase. This may only be appropriate for patients desiring to return to a higher activity level or sport participation. Strength progressions may include, but are not limited to the following:

- Advancing the aforementioned exercises with increasing angles of arm elevation.
- Internal and External rotation at 90° of arm elevation, ensuring adequate scapula stability to isolate GH joint motion.
- Resisted full can exercise: The therapist monitors the patient to ensure compensatory patterns do not return when adding resistance.
- Advanced CKC stabilization exercises: UE step-ups in plank position and pushups on an exercise ballast balls. The therapist ensures that good muscular control is present and the patient does not have scapula winging with increased weight-bearing.
- UE plyometrics: The patient may begin bilaterally and progress to unilaterally or increasing angles of arm elevation.
- Sport-specific drills, including core strength training [20]

Conclusions

A multimodal approach to postoperative pain management and rehabilitation may provide the best and fastest return to function after capsular releases for refractory frozen shoulder. A progressive program of physical therapy predicated on early return of motion is paramount. The full kinetic chain should be considered at all stages of rehabilitation. It is important to tailor postoperative management to a patient's individual needs and accommodate individual goals.

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Frozen Shoulder: Reported Outcomes and Results: What Should We and the Patient Expect?

22

Kevin D. Plancher and Stephanie C. Petterson

22.1 Introduction

Shoulder stiffness is a common problem that affects 2–5% of the population and 10–15% of diabetics [29]. The term "frozen shoulder" has been used to describe the insidious onset of shoulder pain followed by loss of range of motion for no specified reason [4]. The current position of the American Shoulder and Elbow Surgeons (ASES) is that frozen shoulder is "a condition of uncertain etiology characterized by significant restriction of both active and passive shoulder motion that occurs in the absence of a known intrinsic shoulder disorder" [38].

Diabetes, female gender, age 40–60 years, thyroid disease, hyperlipidemia, and prolonged immobilization are several risk factors for developing frozen shoulder [23, 31, 2, 3]. Frozen shoulder is often considered a self-limiting condition resolving within 1–3 years [11]. First-line management for frozen shoulder always includes conservative management. If this approach fails, then surgical intervention may be warranted. There is limited evidence to support the best treatment approach to combat this very challenging condition. The purpose of this chapter is to present evidence to support each of these interventions.

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22.2 Results and Outcomes

22.2.1 Hydrodilatation

Hydrodilatation involves injecting a contrast medium, a local anesthetic, cortisone, and up to 40 ml of sterile saline solution intra-articularly in order to stretch the joint capsule and break up adhesions [7, 1]. A 2008 Cochrane Review presented "silver" level evidence for arthrographic distension with saline and steroids [8]. This review of 196 patients in 5 clinical trials provides evidence for short-term benefits in pain, range of movement, and function in frozen shoulder; however, these studies did not compare the efficacy of capsular distention to other treatment interventions. Buchbinder et al. [7] demonstrated significant improvement in pain and shoulder function 3 months following arthrographic distension with a mixture of saline and steroid [7]. In a prospective study by Quraishi et al. [30], 48 patients with frozen shoulder either underwent hydrodilatation or manipulation under anesthesia. Patient satisfaction and Constant scores were greater in the hydrodilatation group at 6 months; however, range of motion did not differ between groups [30]. Koh et al. [22] assessed a capsule-preserving method of hydraulic distension with saline solution and corticosteroid in patients with frozen shoulder [22]. At the time of the injection, pressures were monitored to avoid capsule ruptures. Results revealed a significant decrease in capsule stiffness and an increase in capsular volume following a series of three injections over a 1-month period. Patients also reported a significant reduction in pain and increases in shoulder range of motion. Elleuch et al. [14] have demonstrated that the benefits of capsular distension followed by an intense rehabilitation program are greater than conservative rehabilitation only and are maintained 12 months following the procedure [14].

Hydrodilatation is a safe, effective, and less invasive treatment approach for the management of frozen shoulder [5, 9]. Capsular rupture does not appear to be key to achieving success; however, the use of a steroid is recommended in conjunction with rehabilitation. Reported range of motion improvement has been shown to be 28° in forward flexion, 42° in abduction, 22° in internal rotation, and 26° in external rotation [9, 34]. We have never had great success with this technique.

22.2.2 Manipulation Under Anesthesia

Manipulation under anesthesia (MUA) involves applying a passive stretch in forward flexion, abduction, and adduction with the scapula stabilized as well as into the extreme ranges of internal and external rotation. Tearing of the capsule may be palpated or even audible by the physician. Closed MUA is typically not indicated in posttraumatic and postsurgical cases of shoulder stiffness due to the increased risk of fracture [19].

Hamdam and Al-Essa prospectively studied 88 patients (98 shoulders) with frozen shoulder that previously failed conservative treatment [18]. Patients received either MUA alone or MUA with one of two types of intra-articular injection (methylprednisolone or a large volume [50–100 cc] of normal saline), followed by physical therapy. Patients who received MUA with an intra-articular normal saline injection had better outcomes at 6–8 months.

Ng et al. [26] reported the outcomes of MUA plus physical therapy in 86 patients with frozen shoulder with a mean duration of symptoms of 13 months [26]. They found a significant improvement in function as measured by the Disabilities of the Arm, Shoulder and Hand (DASH), a significant reduction in VAS pain, as well as improvement in shoulder range of motion (53° in forward flexion, 80° in abduction, and 32° in external rotation) 6 weeks after intervention. Other studies have also demonstrated similar outcomes with longer-term follow-up [35, 15, 18, 25, 28]. Patients with idiopathic or posttraumatic shoulder stiffness have better outcomes compared to patients with postsurgical shoulder stiffness [35].

It is well established that diabetes is a strong risk factor for the development of frozen shoulder; however, the impact of diabetes on intervention remains unclear [2, 6, 16, 33]. Downie et al. [12] compared the outcome of intervention for frozen shoulder in patients with and without diabetes [12]. This retrospective chart review of 148 patients who underwent MUA or arthroscopic release demonstrated similar functional outcomes as measured by DASH and Constant scores. Similar results have been presented by Wang et al., demonstrating no difference in short- or long-term outcomes following MUA in patients with and without noninsulin-dependent diabetes mellitus, [36] though the risk of recurrence is high [20]. On the contrary, the previously mentioned study by Hamdam et al. (2003) reported worse outcomes in diabetic patients under going MUA and intra-articular injection compared to their nondiabetic peers [18] and others have reported no benefit of MUA in patients with diabetes [20].

A 2013 systematic review compared patient outcomes after MUA or arthroscopic capsular release for frozen shoulder [17]; 989 patients in 22 studies were included. At a median follow-up of 35 months (range: 3–189), the differences between the MUA and capsular release groups were minimal for changes in abduction, flexion, and external rotation range of motion and final Constant score. As the quality of evidence is low, it is difficult to provide any strong clinical recommendations based on the existing published literature, and randomized controlled trials are needed to support the use of either modality.

Complications of closed MUA include iatrogenic humeral fractures, glenohumeral dislocation, rotator cuff and labral tears, brachial plexus injuries, and hemorrhagic effusions and hematomas [19, 24]. The senior author encourages the use of closed manipulation in a slow fashion with full cooperation by the anesthetist to ensure complete muscle relaxation prior to commencing. Success rates have been found in the nondiabetic population avoiding arthroscopic capsular release.

22.2.3 Arthroscopic Capsular Release

Arthroscopic capsular release involves releasing scar tissue formation from the underside of the subscapularis tendon through the rotator interval, followed by release of the anterior then inferior and posterior capsule. Release of these structures improves humeral head translation in the inferior and lateral directions [19]. Jerosch [21] described the 360° capsular release, which involves release of the anterior, posterior, superior, and inferior capsule [21]. Abduction range of motion improved from 76° preoperatively to 167° postoperatively. External rotation with the arm in abduction improved from 4° preoperatively to 85° postoperatively. Internal rotation with the arm in abduction improved from 17° preoperatively to 63° postoperatively. While complications of arthroscopic capsular release may include shoulder instability and dislocation in overly aggressive releases, as well as axillary nerve injury when releasing the inferior capsule, there were no complications reported in this series of 28 patients.

Snow et al. [32] compared anterior and inferior release to anterior, inferior, and posterior capsular release in patients with frozen shoulder [32]. They did not find any added benefit of the posterior capsular release; all groups demonstrated significant improvements in function on the Constant score, improved range of motion, and decreased pain. Eighty-nine percent of patients reported that they felt their condition was better or much better following the arthroscopic release.

Similar to findings with MUA, arthroscopic capsular release is most beneficial in patients with idiopathic or posttraumatic shoulder stiffness compared to patients with postsurgical stiffness with low recurrence rates of only 6% [13]. The addition of an arthroscopically-placed, intra-articular pain catheter following arthroscopic release improves pain control and tolerance to postoperative range of motion exercises [37]. Patients receiving bupivacaine via intra-articular pain catheter reported decreased pain to 1.2/10 on VAS. Diabetic patients have similar pain relief from arthroscopic capsular release; however, they score worse on Constant score and display less shoulder abduction and internal rotation range of motion [10].

We recommend a 360° capsular release in patients with idiopathic, posttraumatic, and postsurgical shoulder stiffness who have failed a 6-month course of conservative treatment including supervised physical therapy and subacromial and intra-articular corticosteroid injections. We routinely release the posterior capsule in all cases. In our experience, we have never had any adverse complications including shoulder instability or dislocation or nerve damage. We believe that arthroscopic capsular release is a safe, controlled intervention that does not induce the high forces to the joint as incurred with MUA and therefore poses minimal adverse consequences for the patient with great success.

22.2.4 Coracohumeral Ligament Release

Open capsular release involves excision of the coracohumeral ligament through an incision made from the clavicle to the lateral border of the coracoid with the arm in external rotation. With advancements in shoulder arthroscopy, open releases are rarely performed today. An open or mini-open release is generally performed in conjunction with other procedures such as total shoulder replacement. Release of the coracohumeral ligament allows for improved inferior humeral translation and motion without compromising joint stability.

Omari and Bunker [27] performed open capsular release in 25 patients who failed conservative treatment and MUA [27]. They found on evaluation of the joint that the rotator interval was obliterated and the coracohumeral ligament was a tough contracted band. Improvements in pain, function, and range of motion were seen in the majority of patients following excision of the coracohumeral ligament. Of the two patients who failed to improve, one had insulin-dependent diabetes mellitus and the other had severe bilateral Dupuytren's contractures. Twelve of the 25 patients underwent histological evaluation of the contracted tissue. The tissue consisted of a dense matrix of type III collagen populated with fibroblasts and myofibroblasts.

Conclusion

In summary, frozen shoulder is a difficult condition to treat. When conservative management fails to adequately address symptoms and restore range of motion, surgical intervention is warranted. While MUA may help to improve range of motion and function, it carries the risk of injury due to the forces applied to the joint to break up the capsular adhesions and caution must be taken to avoid further injury. Open capsular release requires extensive dissection to release the tissues and therefore is mostly considered a historical procedure except in cases where other open procedures are performed. Arthroscopic capsular release should be the treatment of choice in recalcitrant cases of frozen shoulder.

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