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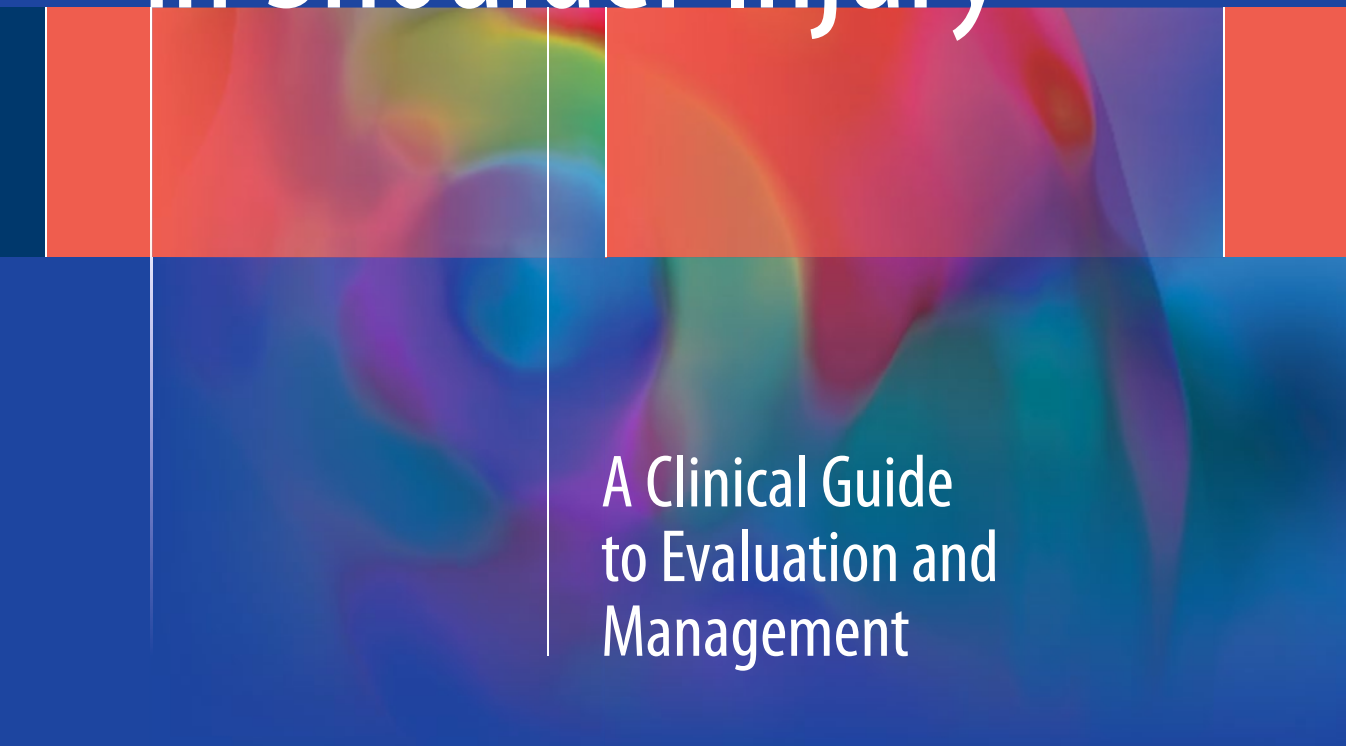
Disorders of the Scapula and Their Role in Shoulder Injury



A Clinical Guide to Evaluation and Management

Springer

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Foreword I

Why This Book Should Be Written (A Shoulder Surgeon's Viewpoint)

As the pathoanatomy of the shoulder comes into greater clarity, the dynamic role that the scapula plays in that complex relationship is also becoming apparent. However, until recently, the scapula may have been incompletely considered [1]. It is quickly becoming obvious that the scapula must be thoroughly studied to effectively optimize treatment of shoulder injuries of all natures. For a practicing orthopedic surgeon, this relationship is important to consider when seeing patients in clinic for outpatient treatment, in planning operative interventions for shoulder injuries, and as an integral part of the recovery process. Indeed, the senior author feels that correcting scapular positioning and tracking is the key to the long-term success of any operative intervention involving the shoulder. Scapular dysfunction is present in some form in all patients with shoulder pathology, and it can alter the accuracy of the examination, imaging, and outcome of both operative and non-operative treatment if not recognized and managed appropriately. Thus, the accurate recognition, diagnosis, and treatment of scapular disorders are an imperative for any surgeon wishing to successfully treat shoulder injuries [1].

Recognition

Scapular dyskinesis is determined during the clinical exam, thus requiring that practicing clinicians be able to recognize the abnormality as well as be informed of the appropriate treatment [2]. The scope of this book aims to address that need, making it an invaluable tool. Scapular dyskinesis can be caused by both internal factors (intrinsic muscle weakness or neurovascular injury) and external factors (acromioclavicular and/or glenohumeral joint injury or soft tissue injuries). A surgeon must be able to recognize the cause of the scapular disorder to successfully treat the injury [2]. It is important to appreciate that not only can scapular dysfunction be caused by shoulder injury but also that intrinsic scapular pathology can lead to greater shoulder pathology [3]. Due to this intricate balance between scapular and shoulder pathology, a thorough understanding of the scapular disorder can often lead to a more complete understanding of the shoulder injury and will guide appropriate treatment, both operative and non-operative. The future chapters

of this book aim to gather this information and make it readily available to the practicing orthopedic surgeon.

There are several major categories of shoulder pathology with associated scapular disorder, and these will be the focus of later chapters with much more in-depth discussion. However, a brief overview of these specific injuries will demonstrate the importance of understanding the scapular-shoulder relationship to the successful treatments of the injuries.

Rotator Cuff Issues

Increased upward rotation of the scapula has been demonstrated in anatomical studies of patients with rotator cuff tears. While it is unclear if this displacement of the scapula is a cause or effect related to the rotator cuff tear, it certainly has been shown to decrease patient's functional abilities [1, 3]. Scapular protraction has also been shown to decrease the maximal rotator cuff strength, creating the appearance of muscle weakness when there may truly be no weakness [2, 3]. Thus, rehabilitation of the scapular dyskinesia should be an integral part of the patient's initial therapy. In patients with rotator cuff pathology and scapular dyskinesis, shoulder therapy initially focuses on correction of the scapular disorder before focusing on the rotator cuff rehabilitation [3]. Proper scapular function allows for accurate assessment of the actual strength and integrity of the rotator cuff and the appropriate treatment for the rotator cuff injury. Without appropriate rehabilitation and treatment of the scapular disorder, the true nature of the rotator cuff injury can be difficult to ascertain and may lead to improper treatment.

AC and Clavicle Issues

In patients with AC separations, the recognition of severe scapular dyskinesis likely dictates the success of non-operative management. Failure to correct the downsloping, dysfunctional scapula and thereby decreasing the deformity of the dislocated AC joint is the major determining factor as to whether the patient will require operative treatment of the AC separation versus conservation management [1–3]. The clavicle connects the scapula to the axial skeleton and provides an anchor about which the scapula can move and rotate. Disassociation from the clavicle can cause significant functional impairments to the scapula, including the loss of rotator cuff strength and shoulder impingement [2, 3]. In the cases of AC separation with resultant scapular dyskinesis, patients who fail early therapy programs will likely require surgical fixation of the AC separation at both the AC and CC ligaments to improve the scapular dyskinesis [3]. Patients who do not develop scapular dyskinesis after an AC separation will usually go on to good outcomes with non-operative treatment [1]. Thus, unlike rotator cuffs where the development of scapular dyskinesis has an unknown etiology, scapular disorders in AC separation can dictate surgical versus nonsurgical treatment, demanding that the surgeon be

able to recognize and understand the relationship between the scapular injury and the shoulder injury.

As seen with AC separations, clavicle fractures also can alter the relationship between the axial skeleton and the scapula via loss of the strut function of the clavicle. Again, this can lead to improper protraction of the scapula, which in turn alters the biomechanics of the glenohumeral joint, potentially leading to rotator cuff weakness, loss of motion, and impingement [2]. These changes have been shown to significantly affect a patient's subjective function scores. Fractures with as little as 1.5 cm of shortening can cause significant scapular dysfunction [3]. Thus again, scapular dysfunction is the result of shoulder injury but propagates the injury to altering the kinematics of the shoulder joint. Patients with clavicle fractures and scapular dyskinesis therefore should be considered for surgical fixation aimed at restoring length, alignment, and rotation to the clavicle [3]. The ability to identify scapular dyskinesis in the setting of a scapular fracture leads the surgeon to consider the potential need for surgical fixation over conservative management.

Instability Issues

Slap injuries: Scapular dyskinesis is also important to recognize with diagnosing superior labral tears. The initial scapular dyskinesis of internal rotation and anterior tilt places stress upon the anterior ligaments of the shoulder [1–3]. This pathologic stress creates an impingement of the labrum and contributes to the development of superior labral tears [3]. Thus, as the scapular disorder contributed to the development of the shoulder pathology, it is vital that the dyskinesis be corrected with therapy. Additionally, correction of scapular dysfunction if recognized early can prevent the subsequent development of superior labral tears. Therefore, a surgeon that can accurately identify scapular dyskinesis without concurrent shoulder pathology can potentially prevent the development of those injuries by placing the patient in appropriate therapy [1].

Throwing athletes: The entire scapular dysfunction issue in association with SLAP tears is most commonly seen in the overhead throwing athlete. The vast majority of throwers with SLAP tears do not require surgery but simply a scapular reposition to correct their imbalance and return to play. The malposition of the scapula in these overhead athletes results in an exacerbation of internal impingement, an artificial increase in GIRD, contracture of the pec minor and symptoms of subacromial impingement, and rotator cuff tendonitis, confusing the management of these athletes.

MDI: Symptomatic patients with multidirectional instability always have severe scapular dyskinesis, which restricts the ability of the rotator cuff musculature to keep the humeral head in the glenoid and increases subluxation, impingement, and tendonitis. The severe dyskinesis produces a biomechanical malalignment of the glenoid. The altered glenoid positioning allows the humeral head to be more prone to dislocation [1–3]. Recognizing the presence of scapular dyskinesis in MDI allows therapy to appropriately target the correct muscle groups. With appropriate strengthening and stabilization of

the scapula through therapy, the glenoid can be brought into appropriate alignment and decrease to risk of recurrent dislocations. By recognizing the presence of multiple factors that are presented in MDI, including scapular pathology, the orthopedist is able to appropriately prescribe the therapy that will be the most successful for the individual patient. Indeed, failure to initially restore the scapula in these patients makes therapy not only ineffective but also painful.

Anterior and posterior instability: The scapula also plays an important role in unidirectional glenohumeral instability. Scapular dyskinesis can both create and potentiate abnormal shoulder biomechanics that are present in glenohumeral instability [4]. Scapular dyskinesis in shoulder instability can result from many factors seen with the instability, including decreased muscle activity coordination, joint pain due to musculoskeletal injury leading to altered kinematics, and muscle weakness or fatigue [4]. Patients with instability secondary to a traumatic injury, often with structural lesions, will often have dyskinesis that cannot be corrected until the anatomic pathology is corrected. In patients with inability secondary to microtrauma or a chronic labral injury, dyskinesis is often present secondary to muscle weakness, and rehabilitation of the dyskinesis can often lead to treatment of the instability [3]. Specifically in posterior instability, winging of the scapula allows for subluxation of the humeral head [5]. Voluntary posterior dislocators thus must recreate this dyskinesis to allow for dislocation. Thus, in unidirectional instability, it is important to understand the etiology of the instability to understand the appropriate treatment of the concurrent dyskinesis.

As shown in the brief examples of shoulder injuries often associated with scapular pathology, the importance of recognizing scapular dyskinesis becomes apparent. However, the treating surgeon needs to not only become intimate with the diagnosis of scapular pathology but also understand the relationships between the present shoulder injury and scapular dyskinesis. In some cases, the scapular disorder may be the cause of the shoulder injury, the reverse may be true, or, in other situations, the causality may not be able to be established. In cases where the shoulder injury leads to the development of scapular dysfunction, the presence of dyskinesis on exam may dictate treatment. Thus, it is important for the orthopedic surgeon to understand these relationships and their role in treating the shoulder injury as a whole. Treating either the shoulder injury without the scapular dysfunction or vice versa can lead to poor outcomes regardless of the level of surgical execution or appropriateness of therapy. These two injuries must be considered together and their causal relationships are important. This book will serve the important function of highlighting those relationships while also providing insight into the appropriate treatments for these injuries, thus making it a valuable resource. We would highly recommend this text to all health-care professionals who manage disorders of the shoulder. As a surgeon, I can honestly say that the philosophy of Dr. Kibler has allowed me to obtain better results in both the operative and non-operative management of my patients.

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Felix H. Savoie III, MD
Emily Wild, MD

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Foreword II

Treating musculoskeletal and sports injuries requires understanding how integrated segments work together to serve a specific biomechanical process. Throwing a baseball requires transferring foot-ground reaction forces up through the spine to release the ball from a hand that has been gripping it in a very precise way. The timing and firing of all these muscles, across all these joints, is essential to optimal function. Understanding the intricate working of all of these segments is basic to understanding the normal physiology and biomechanics as well as the pathophysiology of sports injuries. The scapula is a perfect example of the impact of a very strategically placed bone and its implications with many, if not most, upper limb sporting activities. Finally, there is a book that addresses every aspect of the scapula and its many implications in sports rehabilitation.

As a physiatrist and non-operative sports physician who treats many patients with spine, shoulder, and upper limb injuries, understanding the functions, movements, and interactions of the scapula with the rest of the musculoskeletal system is critical. When I began training over 30 years ago, almost all of the focus at the shoulder was on the glenohumeral joint and the impact of the rotator cuff muscles on maintaining a narrow instantaneous center of rotation [1–3]. Then I recall a paper by Ben Kibler in 1998 that described the importance of scapular positioning on the glenoid to be a stable socket for the rotating humerus as the instant center of rotation of the shoulder. Suddenly, there was more to shoulder motion than simply the rotator cuff muscles [4]. Later papers described how scapular dyskinesis, something that had seen me many times although I had not quite seen it, was an obvious clinical manifestation of a nonoptimal functioning scapula that would have profound effects on the rotator cuff muscles [5]. It becomes more clear to me that the scapula, with its three bony articulations (clavicle, humerus, and thoracic spine) and 18 muscular origins and insertions, asserts great influence on many sports-specific activities such as the throwing motion and the tennis serve. Significant force generation, transfer, and attenuation are achieved through well-coordinated movements around and with the scapula. The location of the scapula in the upper quarter of the body, which is at a distance from much of the major shoulder pathology we treat (i.e., rotator cuff disease, labral tears, etc.), makes it easy to overlook as an important part in the causation of upper limb injuries. Since that time, it has further occurred to be the importance of how the scapula interacts with the cervical and thoracic spine (which it sits on) to contribute to upper limb- and upper spine-related injuries.

From a practical standpoint, every assessment I make of a patient with any upper limb- or upper spine-related problem includes assessment of scapular position and scapular movement. Fortunately, the scapula is relatively easily palpable and generally very easy to inspect and observe. Just looking at the relationship of the cervical spine, thoracic spine, scapular position, as well as arm and hand positions at rest provides a great deal of information regarding what structures are being loaded excessively or being put on tension.

Beyond inspection of the scapula, quantitative measurements can also be made of the position of the scapula as it rests on the spine. Kibler's article from 1998 also described the scapular slide measurements which are also easily done and provide a good assessment of scapula position both statically and dynamically [4]. After years of using the scapular slide as a clinical tool, it appears to be very common finding in patients with many shoulder pathologies, in particular rotator cuff impingement. As symptoms resolve with proper rehabilitation, the scapular slide often improves too.

Still other clinical tests using the scapula are a regular part of my physical examination of upper limb problems. Kibler [4] devised the scapular assistance test to determine whether impingement is due to a lack of active acromial elevation. A significant impingement sign is a painful arc between 60 and 130° upon actively or passively elevating an arm, indicating that something in the subacromial space is being compressed and irritated. Muscle testing of a single cuff muscle may appear weak due to pain along with clicking and symptoms of impingement. The scapular assistance test may normalize these impingement signs indicating that dysfunction of the scapula is related to the impingement syndrome. The test consists of pushing the inferior medial border of the scapula laterally and upward while stabilizing the upper medial border of the scapula to simulate the serratus anterior/lower trapezius muscle portion of the elevation force couple, as the patient elevates the arm in the scapular plane. If the impingement is related to these muscles being inhibited, the impingement symptoms will diminish or be abolished. This test is an essential one in testing patients with impingement symptoms.

Similarly, the scapular retraction/repositioning test (SRT) [6, 7] is important and useful when assessing rotator cuff impingement. The scapular retraction/repositioning test has been described by Kibler in 2006 and Tate in 2008. If a positive impingement test is identified, it can then be repeated with the scapula manually repositioned using the SRT. The SRT is performed by grasping the scapula with the fingers contacting the acromioclavicular joint anteriorly and the palm and thenar eminence contacting the spine of the scapula posteriorly, with the forearm obliquely angled toward the inferior angle of the scapula for additional support on the medial border. In this manner, the examiner's hand and forearm apply a moderate force to the scapula to encourage scapular retraction (scapular retraction test) or posterior tilting and external rotation (scapular repositioning test) and to approximate the scapula to a mid-position on the thorax. The scapular repositioning test has demonstrated reliability, and while performing this test, it has been shown that subjects are capable of demonstrating increased rotator cuff strength and report less pain by providing a stable base [6, 7].

Understanding the scapula is especially important when trying to provide patients with appropriate rehabilitation programs for neck, shoulder, and arm

pains [8]. If the scapula is not positioning properly (i.e., excessive protraction), shoulder elevation in flexion and abduction is more difficult. Furthermore, with excessive scapular protraction, pectoralis and scalene muscles end up in tightened, shortened positions. Middle and lower trapezius muscles are put on stretch. The resulting forward head position forces the upper trapezius muscles to overwork to keep the 8–10-lb head (with contents) from falling forward. This positioning of the scapula on the thoracic spine, with head-forward positions, seems to be at the heart of many, if not most, cervical spine disorders. Again, it seems odd that to address many cervical spine problems, we actually start with the scapula and how we position it properly.

I find *Disorders of the Scapula and Their Role in Shoulder Injury* to be a one-of-a-kind book that looks at a key anatomic structure in sports medicine and gives you every piece of up-to-date knowledge on the subject that will enhance your ability to diagnose and treat sports and musculoskeletal injuries of the upper limbs and spine. The information is well organized and presented in clinically relevant and applicable order. I use the concepts in this book on a daily basis in evaluating and treating my patients with musculoskeletal injuries. It is a must-have for sports physicians, surgical and nonsurgical.

Now York, NY Joel Press, MD

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Preface

The scapula is a fascinating bone, mainly because of the wide variety of roles it plays in facilitating and optimizing shoulder and arm function in almost every human activity. Because of its location on the posterior shoulder, its overlying subcutaneous and muscular tissue, and its large mobility, it has been underappreciated and underevaluated in most thought processes regarding shoulder function and injury. However, scholarship is now demonstrating the multiple key roles of the scapula that facilitate shoulder function and has started highlighting the roles that altered scapular static position and dynamic motion may have in many types of shoulder pathology and injury.

The scapula is a welcome partner when it works well to increase muscle strength, to move the acromion out of the way of the moving arm, to work as a stable base for arm motion, and to create optimal mechanics for strength and power. Its effects are manifested throughout the entire arm and hand. However, it is a difficult adversary when it is not working well, decreasing demonstrated shoulder strength, creating or increasing joint instability, and causing increased pain with use. Its deleterious effects are also seen throughout the shoulder, arm, and hand. It can be difficult to clinically examine, and treatment protocols can be quite complicated. This often requires precise and comprehensive evaluation of all the factors that may be contributing to the dysfunction. There has been a general lack of medical education regarding the scapula, which complicated efforts to provide adequate clinical care for patients with these problems.

I was no different. I had minimal knowledge about the scapula after my medical school and residency training. My personal involvement with the scapula started about 30 years ago, when I first noticed scapular winging in a patient with "impingement" that did not respond to traditional treatment. The manual repositioning of the scapula immediately changed her symptoms and set me on a journey to learn more about this bone. The journey has been drawn out, with many starts and stops and with some dead ends. It started with trying to understand the basic motions and functions of the scapula, both in two-dimensional and three-dimensional functions. Then it required development of some types of evaluation and description of the motions. Finally, it required clinical correlation: What roles did these motions play in shoulder function and injury, and what are the best treatment protocols? It also required the development of a network of like-minded individuals who shared this interest and had research and clinical capabilities that could advance the knowledge base and the clinical application.

These efforts resulted in a series of "scapular summits," consensus meetings that brought together the individuals, organized the knowledge, highlighted the

future directions for future research and application, and created a larger network of interested individuals. These meetings, and the consensus statements published from the proceedings, stimulated a larger body of knowledge, most of which is captured in this book. I am deeply indebted to those, such as Phil McClure, Paula Ludewig, Ann Cools, and Tim Uhl, who have been in this from the beginning and who formed the core of the knowledge base. Others, such as Jed Kuhn, Robin Cromwell, Dave Ebaugh, Lori Michener, and Marty Kelley, have made valuable contributions to enlarging the knowledge base. I owe a special debt to Aaron Sciascia, who has played pivotal roles in developing the scapular database, the clinical treatment and rehabilitation protocols, and the structure of this book.

This book is the result of a long process of discovery and implementation, which has taken up a large portion of my professional life. I am indebted to my partners in the Shoulder Center of Kentucky, Drs. David Dome, Pete Hester, Trevor Wilkes, and Brent Morris, who have carried a lot of the clinical burden which has allowed this type of investigation; they have also contributed chapters to the book.

This amount of dedicated effort also impacts my personal life. My solid foundation and best counsel has always been Betty Kibler, my wife of 47 years. Her contributions to everything in my life go way beyond things that are seen and known, and I will always look to her for help, guidance, and wisdom.

The scapula is one part of the amazing created machine we call the human body. It is so wonderfully made, and its parts, even though individual in anatomy, work so perfectly together in function that there is clearly a purposeful and intentional Creator, God. I am thankful that He has given me some insight into the miraculous workings of a part of this creation, and I hope to continue to work to understand the mysteries and beauty of the human body and to be able to help people with injuries and dysfunction.

Last but certainly not least, I wish to express my thanks and gratitude to all the patients who have been evaluated and treated for scapular problems as this process of discovery and improvement has occurred over the years. Because of the relative lack of knowledge, they frequently did not have access to timely treatment, with the resulting frustration and problems with function. Their persistence in seeking treatment and their willingness to participate in developing a deeper understanding of scapular dyskinesis were large factors in encouraging us to keep trying to improve the knowledge and care. I can truly say that they are partners in this process.

It is time this book is written. There is enough basic science knowledge to form a solid foundation of function and dysfunction, enough clinical experience to develop a reliable evaluation protocol, enough rehabilitation knowledge to set up successful rehabilitation protocols, and enough clinical knowledge to make valid correlations between scapula function/dysfunction and various shoulder injuries. The authors of the chapters are well versed in their subjects, most of them being pioneers in development of the knowledge within their chapters. This book is not the end of the process of understanding the scapula but will serve as an excellent start for the process.

Read it and enjoy.

Lexington, KY W. Ben Kibler, MD

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Part I

The Basics

Anatomy of the Scapula

1

Trevor Wilkes, W. Ben Kibler, and Aaron D. Sciascia

Introduction

Ideal scapular function reflects its complex anatomy and in turn is foundational for all shoulder function. The scapula plays a multitude of roles. Anatomically, it is the "G" of the glenohumeral (GH) joint and the "A" of the acromioclavicular (AC) joint. Physiologically, it is the "S" of scapulohumeral rhythm (SHR), the coupled and coordinated movement between the scapula and arm that allows the arm to be placed in the optimum position and motion to accomplish tasks. Biomechanically, it provides a stable base for muscle activation, a moving platform to maintain ball-and-socket kinematics, and an efficient link between the core, which develops force, and the arm, which delivers the force. Critical to these roles is normal scapular motion.

To comprehend the complex biomechanics of the scapula, it is critical to have a deep knowledge of the anatomy. It is not surprising that all types of shoulder pathology demonstrate altered motion. Frequently, assessment of scapular muscular attachments, innervation, motion, and position can provide key information on treatment options and guide rehabilitation. This chapter will concisely address pertinent aspects of anatomy of the scapula as it pertains to normal scapular function and clinical implications.

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Scapula: Anatomy

The bony anatomy is predicated on the developmental advantages of mobility, such as prehension and overhead use. This is reflected in several primary changes noted through time in the hominid scapula. First, the acromion has broadened and lateralized to allow mechanical advantage for the deltoid muscle [1]. The coracoid process (meaning "like a crow's beak") enlarged in a manner theorized to assist in the prevention of anterior dislocation at 90° of abduction [2, 3]. Finally, broadening and alteration in the force vector of the infraspinatus and teres minor are postulated to increase both external rotation strength and humeral head depression [4].

The scapula is a large flat bone which forms from a collection of mesenchymal cells [5]. It shows signs of ossification by the fifth week of embryologic development [5]. The scapula follows a predictable course in descending from the paracervical

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region to the thorax. Failure of this process leads to Sprengel's deformity [6]. By the seventh week, the scapula has descended to its final position, and the glenoid is easily identified.

The scapula is primarily formed through intramembranous ossification. The body and spine are ossified at birth and subsequently follow an expected pattern. However, there are several notable exceptions with clinical implications. The coracoid forms from two centers of ossification and is generally united by age 15. Rarely, a third ossification center at the tip can persist and present confusion with a fracture [7]. The glenoid also forms from two separate ossification centers, one at the base of the coracoid and another with a horseshoe contour inferiorly [7]. These are usually fused by 15 years of age as well. Finally, os acromiale may be noted in up to 8% of the population and is the result of two or three ossification centers which arise in puberty and fail to unite by the expected age of 22 [8]. The variable failures of fusion may result in the following abnormalities, from anterior to posterior, pre-acromion, meso-acromion (most common), meta-acromion, and basi-acromion [1, 8].

Grossly, the scapula is a thin sheet of bone which serves as a critical site of muscle attachment. The blood supply is primarily through a network of periosteal vessels which take origin from muscular insertions. Thickening of the bone is notable at the lateral border and superior and inferior angles. Ventral concavity creates a smooth articulating surface against the ribs. Small oblique ridges exist ventrally for the tendinous insertions of the subscapularis [5]. Similarly, small fibrous septa are present dorsally to attach and separate the infraspinatus, teres minor, and teres major. The dorsal surface is traversed by the scapular spine which divides two concavities, the supraspinatus and infraspinatus fossae. The medial two thirds of these fossae give rise to the supraspinatus and infraspinatus muscles. The spine contains two important notches. The suprascapular notch at the base of the coracoid contains the suprascapular nerve, and compression at this location will affect both the supraspinatus and infraspinatus muscles [3, 9]. Second, the spinoglenoid notch is present at the lateral border of the spine [3]. Various causes can lead to compression of the suprascapular nerve here as well, producing isolated atrophy of the infraspinatus.

Anatomic interest in the scapula is frequently directed at the coracoid, acromion, or glenoid. The name coracoid derives from the Greek word korakodes meaning "like a crow's beak" [3]. The bent shape resembles a finger pointed toward the glenoid. From the Greek word "akros" for point, the acromion is often referred to as the point of the shoulder. The morphology of the acromion is among the most studied in the body. Considerable cadaveric research has been directed at the relative frequency and postulated causes of the types 1 through 3 acromion, as described by Bigliani [1]. However, the relationship between acromial shape and "impingement syndrome" or rotator cuff tear has not borne out in literature. Similarly, the glenoid has been the subject of intensive study in an effort to define bony anatomy in shoulder instability [7, 10–13]. Average values for size include a height of 35 mm and width of 25 mm, but considerable variability exists. Comparison to the contralateral side may be required to precisely define bone loss. Glenoid version may also range widely. Retroversion, up to 6°, is most common, as seen in 75% of the population, but anteversion up to 2° is reported [14–18].

The function of the scapula is dependent on the complex recruitment patterns of the 18 muscular attachments [19]. These muscles can generally be categorized as axioscapular, scapulohumeral, and muscles of the upper arm (coracobrachialis, biceps brachii, and triceps brachii) [20, 21].

The axioscapular muscles serve to anchor the scapula for its role as the foundation of the

shoulder. In addition, they guide the scapula through the requisite degrees of freedom. These muscles include the serratus anterior, levator scapulae, pectoralis minor, rhomboids, and trapezius. The trapezius is the largest and most superficial axioscapular muscle. The expansive muscle originates from the occiput, nuchal ligament, and spinous processes of C7 through T12 [20]. The upper trapezius inserts across the distal third of the clavicle and acromion. The middle trapezius inserts across the scapular spine and the lower portion at the base of the spine. The broad muscle allows complex function in scapular retraction, elevation, and posterior tilting based upon the recruitment pattern. Frequently, the upper and lower trapeziuses are associated separately.

Motor innervation is through cranial nerve 11, the spinal accessory nerve [3]. The rhomboids are divided into the major and minor portions. The rhomboid minor originates from the spinous processes of C7 and T1 and inserts at the medial scapular border at the base of the spine [20]. The rhomboid major begins from T2 to T5 and inserts along the posterior aspect of the medial border from the base of the spine caudally to the inferior angle. This orientation allows an important role in scapular retraction. The dorsal scapular nerve (C5) provides innervation. The serratus anterior is comprised of three divisions taking origin from the anterolateral aspect of the first to ninth ribs. Innervation of the serratus is provided by the long thoracic nerve. The serratus produces protraction and upward rotation of the scapula with arm elevation while providing a critical stabilization function against excessive internal rotation throughout nearly all positions of arm forward flexion and elevation. The levator scapulae is intimately associated with the serratus and serves a role to elevate and upwardly rotate the scapula. The levator originates from the transverse processes of C1 through C3 and at times C4. Insertion is found upon the superior angle. Innervation comes from the deep branches of C3 and C4. The pectoralis minor is often overlooked in its role in scapular position. The muscle originates from the second to fifth ribs and courses superolaterally to insert upon the coracoid. Chronic tightness can contribute to protracted, anteriorly tilted scapular positioning [22–24].

The scapulohumeral muscles produce glenohumeral motion and are composed of the deltoid, supraspinatus, infraspinatus, subscapularis, teres minor, and teres major. The deltoid originates broadly across the acromion and scapular spine while inserting on the deltoid tubercle of the humerus. This structure allows it to power elevation in multiple planes. As previously noted the supraspinatus and infraspinatus originate from the medial two thirds of their respective fossae while inserting in a complex arrangement on the greater tuberosity. The subscapularis originates from the anterior aspect of the scapula and attaches on the lesser tuberosity. The teres minor takes origin from the middle section of the lateral scapula and is innervated by the posterior branch of the axillary nerve. The teres major emerges from a more inferior position on the lateral scapula and shares a common tendinous insertion with the latissimus dorsi on the medial aspect of the bicipital groove. It shares innervation from the subscapular nerve and functions in internal rotation, adduction, and extension of the humerus.

Two major bursas exist persistently around the scapula. The infraserratus bursa resides between the serratus anterior and the chest wall. The supraserratus bursa occupies space between the subscapularis and serratus anterior. In addition, several minor bursas may be present at the superomedial border, the inferior angle, or the medial base of the spine. Bursa may become persistently inflamed through overuse and subtle abnormalities in mechanics.

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Mechanics of the Scapula in Shoulder Function and Dysfunction

2

Paula M. Ludewig and Rebekah L. Lawrence

Abbreviations

AC	Acromioclavicular
CA	Coracoacromial
SC	Sternoclavicular

Introduction

The purpose of the shoulder is often described as to allow the positioning of the hand across a broad range of motion or functional workspace. As such, a great deal of mobility is required making the glenohumeral joint the most mobile individual joint in the human body. In serving as the proximal (glenoid) component of the glenohumeral joint, the scapula plays a critical role in maximizing the range of possible positioning of the hand while still maintaining the integrity of the glenohumeral joint. This chapter reviews the current state of knowledge regarding the normal positions and motions of the scapula during upper extremity motion and overviews how scapular motion abnormalities may contribute to shoulder pain and dysfunction. With these goals, it is important to recognize that our knowledge base continues to evolve as research advances our understanding of shoulder function and dysfunction.

Overview of Component Joint Motions and Scapular Function

While not a true joint by definition, the overall scapular positioning and motion on the thorax (i.e., scapulothoracic position and motion) is often described clinically and in the scientific literature rather than the sternoclavicular (SC) and acromioclavicular (AC) joint positions and motions from which scapulothoracic kinematics originate. This is in part due to the greater ease in visualizing and measuring scapulothoracic positions and motions. This chapter will provide the traditional scapulothoracic descriptions and also the specific contributions of the SC and AC joints when known. As linked segments, it is not possible to produce scapular motion on the thorax without motion at either, or most commonly, both of the

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SC and AC joints [1, 2]. The surface of the thoracic rib cage provides an additional constraint to the possible positioning and motions of the scapula. The combined thoracic, clavicular, and scapular segments and their associated articulations are often referred to as the shoulder girdle. As such, scapulothoracic motion is in reality motion of the combined shoulder girdle complex.

Overall, the ability to move and reposition the scapula on the thorax is important to several aspects of shoulder function. As already noted, scapulothoracic complex motion is critical to maximize overall range of motion to position the hand while still maintaining the humeral head in the glenoid "socket" [3]. Figure 2.1 visually illustrates the approximate functional workspace without scapular motion, as well as the additional range of motion available through combined scapular and humeral motion. Further, because of the large range of motion of the glenohumeral joint, the deltoid is unique among muscles anatomically crossing only a single joint in that active insufficiency may occur as the humerus elevates relative to the thorax [4]. Moving the proximal scapular attachment of the deltoid through scapular motion (in particular upward rotation) maintains a more moderate length-tension relationship. Better maintaining the length-tension relationship allows for higher deltoid force and subsequently power for a given humerothoracic elevation angle. Finally, scapular motion and positioning is believed critical to minimizing excess stress to musculature and joint structures (e.g., rotator cuff, labrum, biceps long head, acromioclavicular and glenohumeral capsule and ligaments, coracoacromial ligament, acromial undersurface, etc.) while still maintaining a stable and functional glenohumeral joint. The chapter will conclude with further discussion of the implications to tissue and joint stress in the presence of abnormal scapular motions and positions, or "dyskinesis."

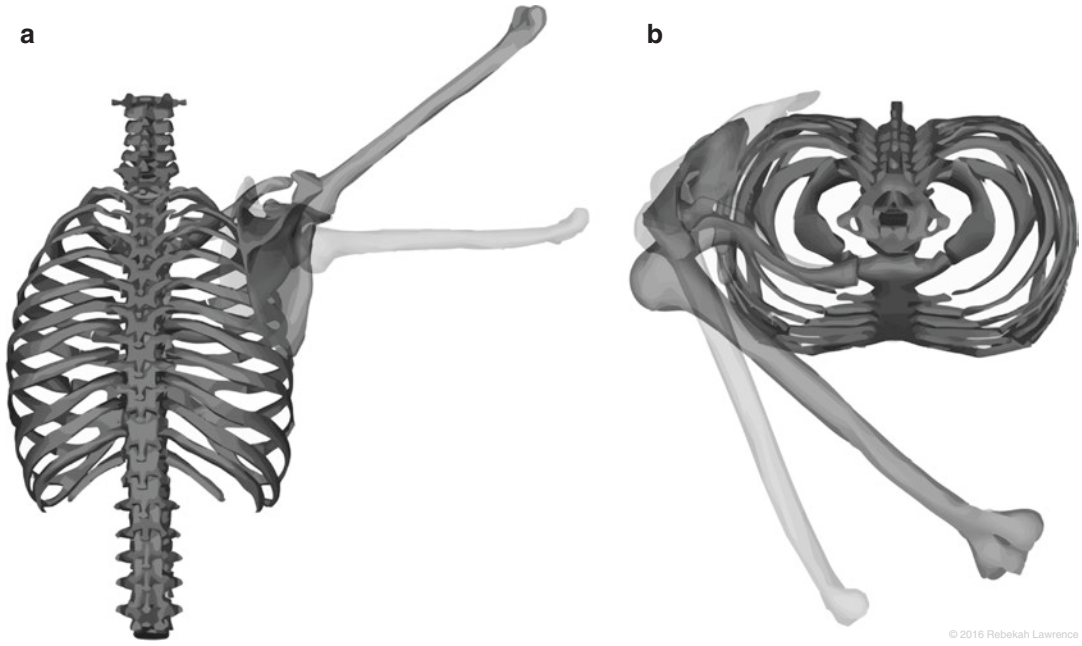

Fig. 2.1 The contribution of scapular motion to overall shoulder motion during (**a**) scapular plane abduction and (**b**) horizontal adduction. Transparent bones illustrate the expected range of motion without scapulothoracic contribution. Opaque bones illustrate the range of motion with both glenohumeral and scapulothoracic contribution. (Reproduced with permission of Rebekah L. Lawrence)

Sternoclavicular Joint Position and Motion

The SC joint consists of the clavicle moving relative to the manubrium of the sternum and contains an intermediate disc (Fig. 2.2a–c). Internationally, a number of different naming conventions exist; however, we will describe the three rotational motions of the SC joint as elevation/depression, protraction/retraction, and anterior/posterior long-axis rotation. In addition to the osteokinematic rotational motions, small amounts of translations can occur three- dimensionally at the joint. These translatory motions and associated arthrokinematic joint motions are described elsewhere [5] and are not the focus of this chapter.

Protraction and retraction of the SC joint (Fig. 2.2a) occur about an approximately vertical axis. Protraction brings the distal clavicle anterior, while retraction brings the distal clavicle posterior. The initial position of the clavicle relative to the thorax frontal or coronal plane in relaxed standing is approximately 20° of retraction [6, 7]. This retraction can be appreciated during physical exam by noting with palpation a more posterior position of the AC joint relative to the SC joint. Total possible motion of the clavicle into protraction and retraction is also not well characterized in the research literature but is believed to be approximately 20° of protraction and approximately 30° of retraction from its initial retracted position [6, 8].

Elevation and depression of the SC joint (Fig. 2.2b) occur about an approximately anterior/posterior axis. Elevation raises the distal clavicle superiorly relative to its rest position, while depression lowers the distal clavicle. The initial position of the clavicle relative to a thoracic transverse plane in a relaxed standing posture is typically slight elevation (10° or less) [6, 7]. This slight elevation can be appreciated during a physical exam by noting with palpation a more superior position of the AC joint relative to the SC joint. From the initial position of slight elevation, minimal depression of the clavicle can occur (10–15°) [8] due to the physical constraint of the rib cage immediately below the clavicle. Total possible motion of the clavicle into elevation is not well characterized in the research literature but has been described as 45° of elevation from the initial position [8]. As will be described later, much less clavicle elevation occurs during functional arm elevation.

Anterior and posterior rotations of the SC joint (Fig. 2.2c) occur about the long axis of the clavicle. Anterior rotation brings the conoid tuberosity of the clavicle posteriorly, while posterior rotation brings this process anteriorly. No anatomical standard currently exists to define the initial axial rotation position of the clavicle, and as such, its position during relaxed standing is typically defined as zero degrees rotation. Total possible motion of the clavicle into anterior rotation is minimal due to the constraint of the first rib. Total possible motion of the clavicle into posterior

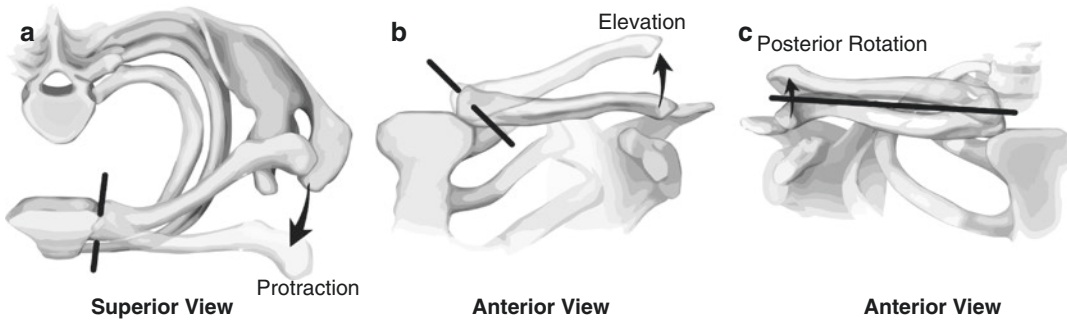

Fig. 2.2 Motions of the sternoclavicular joint: (**a**) protraction/retraction about a superiorly directed axis; (**b**) elevation/depression about an anteriorly directed axis; (**c**) anterior/posterior rotation about the long axis. (Adapted from Ludewig et al. Motion of the shoulder complex during multiplanar humeral elevation. J Bone Joint Surg 2009)

rotation is described as 50° [2], corresponding to this rotation being the primary motion of the SC joint during arm elevation [2, 6].

Acromioclavicular Joint Position and Motion

The AC joint allows for relative motion between the distal clavicle and the anteromedial acromion process of the scapula and often includes an intermediate disc. This is commonly described as the more distal scapula moving relative to the clavicle (Fig. 2.3a–c). As with sternoclavicular joint motion, a variety of naming conventions exist. We will use upward/downward rotation, internal/external rotation, and anterior/posterior tilting to describe the three angular rotations at the AC joint. The reader is again referred elsewhere for descriptions of AC joint translations and arthrokinematic motions [5].

Acromioclavicular joint internal and external rotations are also commonly referred to as protraction and retraction, respectively. However, we prefer the internal/external rotation convention to distinguish SC and AC transverse plane rotations. Acromioclavicular internal/external rotation are described about an approximately vertical axis at the AC joint (Fig. 2.3a). Internal rotation will orient the glenoid anteriorly, while external rotation will orient the glenoid posteriorly. The initial position of AC joint internal/external rotation during relaxed standing is best appreciated from a superior transverse plane view of the scapula and clavicle (Fig. 2.4). The initial position of the AC joint is slightly less than 60° of internal rotation [6, 9] considering the alignment of the scapular axis (pointing from the root of the spine of the scapula to the posterior AC joint) relative to the clavicular long axis (Fig. 2.4). Total possible motion of the AC joint into internal/external rotation has received little investigation [8]. However, the range of motion available is dependent on the amount of SC joint retraction because of the intermediate constraint of the thoracic rib cage. For example, when the clavicle is in a more retracted position, the transverse plane angle between the scapula and clavicle will be reduced. Subsequently, acromioclavicular joint internal rotation will be limited by contact of the anterolateral scapular border with the thorax, while AC joint external rotation will be limited by contact of the anterior vertebral border of the scapula with the thorax. In contrast, when SC joint retraction is reduced, the transverse plane angle between the scapula and clavicle will increase as the scapula slides along the curved thoracic constraint with SC protraction. When the scapula is more laterally positioned on the thorax, it is likely the available internal and external rotation motion at the AC joint increases due to lesser constraint from the rounded thorax.

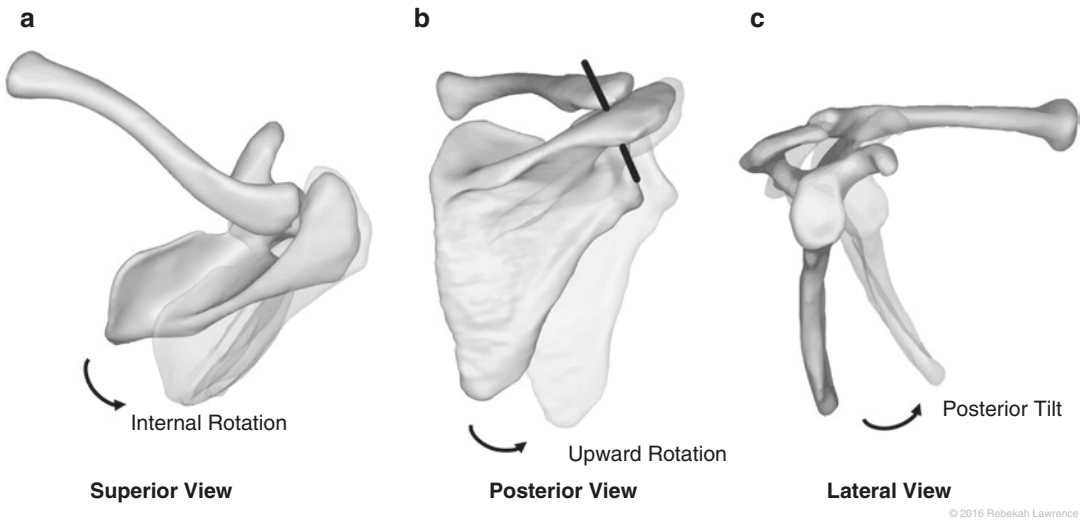

Fig. 2.3 Motions of the acromioclavicular joint: (**a**) internal/external rotation; (**b**) upward/downward rotation about an axis perpendicular to the plane of the scapula; (**c**) anterior/posterior tilt. (Reproduced with permission of Rebekah L. Lawrence)

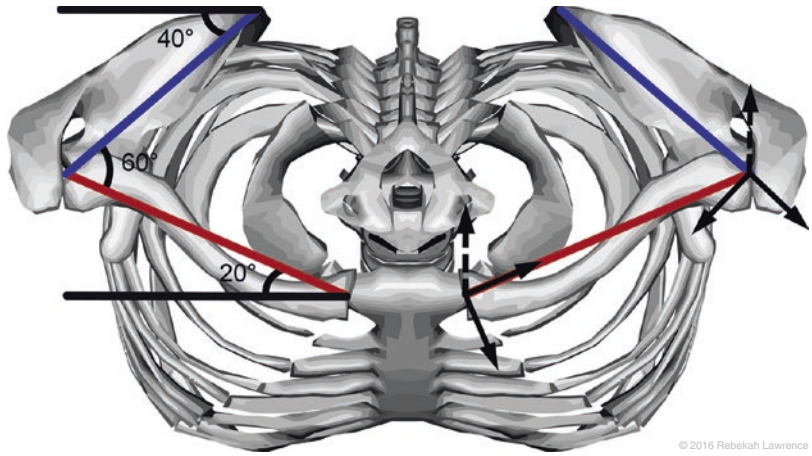

Fig. 2.4 The acromioclavicular and sternoclavicular joint axes including the scapular lateral axes (*blue*), clavicular long axes (*red*), and trunk coronal plane axis (*black*). The oblique orientation of the scapular axes relative to the clavicular axes defines the indirect coupling relationship between sternoclavicular joint motion and scapulothoracic joint motion. In a relaxed standing posture, the clavicle is typically retracted 20° and the scapula internally rotated about 40° relative to the coronal or frontal plane of the trunk. Therefore, the acromioclavicular joint typically demonstrates about a 60° internal rotation angle of the scapula relative to the clavicle long axis (Reproduced with permission of Rebekah L. Lawrence)

Acromioclavicular upward and downward rotations are described about an oblique anterior/posterior axis perpendicular to the plane of the body of the scapula (Fig. 2.3b). Upward rotation will orient the glenoid upward, and downward rotation will orient it downward (Fig. 2.3b). The initial position of AC joint upward rotation during relaxed standing is less than 5° [6], considering the alignment of the scapular axis (pointing from the root of the spine of the scapula to the posterior AC joint) relative to the clavicle long axis. Total possible motions of the AC joint have not been recently described in the research literature [8]. However, at least 20° of upward rotation is known to be possible due to the upward rotation measured during arm elevation in asymptomatic subjects [6], making upward rotation one of the primary motions of the AC joint.

Acromioclavicular anterior and posterior tilts are described about an oblique lateral AC axis passing through the joint (Fig. 2.3c). The motions are defined relative to the acromion process such that anterior tilt will bring the anterior acromion inferior and forward, and posterior tilt will bring the anterior acromion superior and back. Notably, the inferior angle of the scapula moves in the opposite direction during these motions (e.g., anterior tilt results in posterior motion of the inferior angle of the scapula) and is therefore often a source of confusion when defining this motion. The initial position of the AC joint in relaxed standing is about 10° or less of anterior tilt [6]. Total possible motion of the AC joint into anterior and posterior tilt has also not been recently described in the research literature [8]; however, at least 20° of posterior tilt is known to be possible due to the posterior tilt measured during arm elevation in asymptomatic subjects [6]. This magnitude of motion makes posterior tilt a primary motion of the AC joint in addition to upward rotation.

Scapulothoracic Position and Motion

The position and motion of the scapula are often described relative to the cardinal planes of the trunk. Although scapulothoracic motion is a direct consequence of SC and AC joint motion, it is still frequently described in the literature, and the trunk provides a useful clinical reference frame. The scapular axes are aligned the same whether describing the position and motion of the scapula relative to the trunk or to the clavicle. Subsequently, we use the same angular naming conventions (upward/downward rotation, internal/external rotation, anterior/posterior tilting) as at the AC joint (Fig. 2.3a–c), but note that the cardinal

planes of the trunk replace the clavicle as the proximal reference.

"Translations" are often also described for the scapula on the thorax. Because the scapula cannot move on the thorax without motion at either the SC or AC joint or both, and because both of these joints only allow very limited translation, it is important to recognize the origination of scapulothoracic "translations" is actually through rotations of the SC joint. Lateral and medial "translation" of the scapula on the thorax occurs through protraction and retraction at the SC joint, respectively (Fig. 2.5a, b). Other terms for lateral and medial "translation" of the scapula on the thorax include scapulothoracic abduction and adduction or scapulothoracic protraction and retraction. Because of the potential for confusion of the source of these "translations," as well as the overlapping protraction/retraction terminology with angular rotation terminology, we prefer to describe these "translations" by describing the underlying SC joint rotations directly. Elevation and depression of the scapula on the thorax occur through rotational elevation and depression of the clavicle at the SC joint (Fig. 2.5c, d). Finally, it is important to remember the additional con-

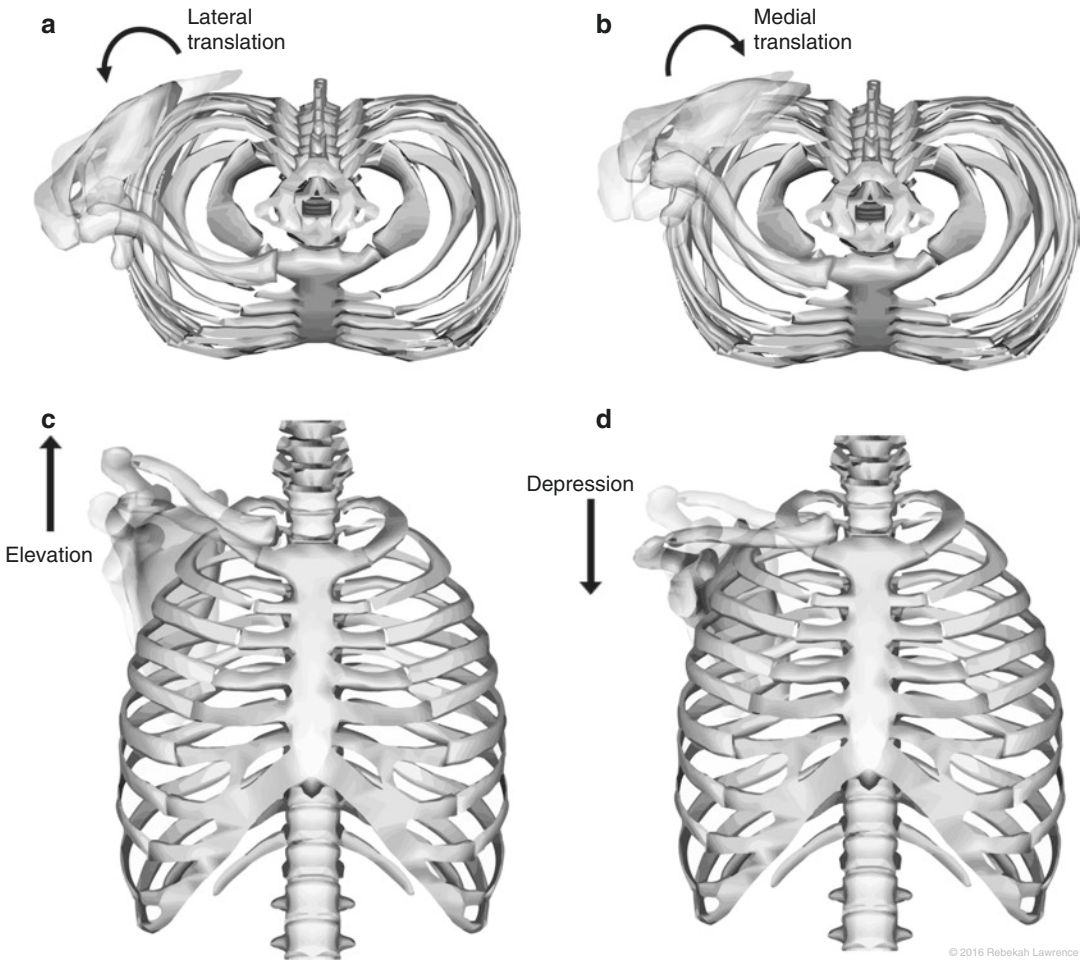

Fig. 2.5 Scapular "translations": (**a**) scapular lateral "translation" or lateral motion of the scapula on the thorax produced through sternoclavicular joint protraction; (**b**) scapular medial "translation" produced through sternoclavicular joint retraction; (**c**) scapular elevation or superior motion of the scapula on the thorax produced through sternoclavicular joint elevation; and (**d**) scapular depression produced through sternoclavicular joint depression. (Reproduced with permission of Rebekah L. Lawrence)

straint provided by the curved thoracic rib cage. As these "translations" of the scapula on the thorax occur through SC joint protraction/retraction or elevation/depression, there will be necessary angular adjustments at the AC joint in order to adapt the scapula to the curved thoracic surface.

Overall upward and downward rotation of the scapula on the thorax is described about an oblique anterior/posterior axis perpendicular to the plane of the body of the scapula. As at the AC joint, upward rotation will orient the glenoid upward, and downward rotation will orient it downward (Fig. 2.3b). The initial position of the scapula on the thorax during relaxed standing is approximately 5° of upward rotation relative to the transverse plane of the trunk [6], considering the alignment of a scapular axis from the root of the spine of the scapula to the posterior aspect of the AC joint. Total possible motion of the scapula on the thorax resulting from combined SC and AC joint motions is typically reported as 60° or greater [2], making upward rotation the primary overall motion of the scapula on the thorax.

It is important to realize with all of these descriptions of joint positions and motions, the reported values are dependent on the anatomical landmarks chosen for rotational axis definitions, as well as the sample of subjects tested in a research investigation. This is particularly important to realize for describing upward rotation of the scapula. With our descriptions of the axis of rotation of the scapula from the root of the spine of the scapula to the posterior AC joint, the initial position of the scapula in relaxed standing will typically be upwardly rotated, even if the vertebral border of the scapula appears to be in a vertical position (Fig. 2.6). This is because the alignment of the vertebral border of the scapula is not perpendicular to the axis pointing from the root of the scapular spine to the AC joint (Fig. 2.6). Descriptions of scapular upward rotation based on an axis aligned with the vertebral border of the scapula will result in lower values, including typically defining the rest position of the scapula on the thorax in relaxed standing as 0° (5).

Internal and external rotations of the scapula on the thorax are also commonly referred to as protraction and retraction. As noted previously, we prefer the internal/external rotation convention to distinguish SC and AC transverse plane rotations and avoid confusion with "translatory" scapulothoracic motions. Internal and external rotations of the scapula on the thorax are described about an approximately vertical axis as at the AC joint; internal rotation will orient the glenoid anteriorly, while external rotation will orient the glenoid posteriorly (Fig. 2.3a). The initial position of the scapula on the thorax during relaxed standing is approximately 30–40° anterior relative to the frontal or coronal plane of the trunk (Fig. 2.4) [6, 7]. Total possible motion of the scapula on the thorax into internal/external rotation has not been directly investigated. However, as a combination of SC protraction/retraction and AC protraction/retraction, total available motion can be substantial. This can be illustrated during cross-body adduction when maximum SC protraction occurs with maximum AC internal rotation (Fig. 2.1b).

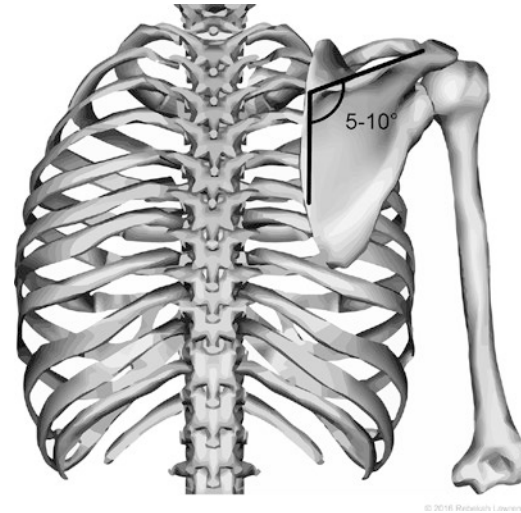

Fig. 2.6 The typical position of the scapula on the thorax during a relaxed standing position. Note the magnitude of upward rotation of the scapula on the thorax described will depend on the axis alignment used. An axis parallel to the medial border of the scapula on the thorax is not directly perpendicular to an axis running from the root of the scapular spine to the posterior acromioclavicular joint. Rather, this axis is at an angle of 95–100° from the medial border axis. As such, a scapular position with the arm relaxed at the side of 0° of upward rotation based on the medial border axis will be described as 5–10° upward rotation based on the scapular spine axis. (Reproduced with permission of Rebekah L. Lawrence)

Anterior and posterior tilts of the scapula on the thorax are described about an oblique lateral axis. As at the AC joint, the motions are described relative to the acromion process such that anterior tilt will bring the acromion superior and forward and posterior tilt will bring the acromion inferiorly and back (Fig. 2.3c). The initial position of the scapula on the thorax in relaxed standing is approximately 5–10° of anterior tilt [6]. Again, the total possible motion of the scapula on the thorax into anterior and posterior tilt has also not been described in the research literature. However, as this composite scapular motion does not receive substantial contribution from the SC joint [6, 9], the total motion possible should be similar to that of the AC joint.

Composite Motions During Planar Elevation and Functional Reaching

To best understand the composite motion of the scapula on the thorax during planar elevation and functional reaching motions, it is necessary to first understand the individual joint motions of the SC and AC joint. Subsequently, we can then review the coupling of the SC and AC joints that occurs with motion of the scapula on the thorax. Most investigations have focused on elevation of the arm into scapular plane abduction (raising the arm in the plane of the scapula, or approximately 40° anterior to the coronal plane of the trunk), flexion, or abduction [6, 7], as well as a study of unrestricted overhead reaching [10]. We will refer to raising the arm in any of these planes in general as humeral elevation. Despite differences in the transverse plane positioning of the arm from which the humerus is then elevated, there is substantial consistency of motion for the SC and AC joints, as well as the scapula on the thorax.

During elevation of the arm in any plane from flexion to abduction, as well as during functional reaching, the SC joint demonstrates characteristic patterns of motion. The primary motion that occurs at the SC joint as the arm is elevated is posterior rotation [2, 6]. About 30° of posterior rotation of the clavicle typically occurs at the SC joint as the arm is raised to 120° of elevation in any plane. Additional posterior rotation will occur to obtain higher degrees of arm elevation. There is no substantive change in the amount or pattern of this motion as the plane of elevation changes from flexion to abduction [6].

Secondarily, the SC joint undergoes retraction during elevation of the arm. About 15° of retraction can be expected to occur in order to reach 120° of elevation during scapular plane abduction. However, the amount of SC retraction will be directly impacted by the plane of arm elevation. For example, to elevate the arm in flexion, the scapula (particularly the glenoid) needs to be oriented more anteriorly in order to maintain congruency with the humerus. In contrast, to elevate the arm in abduction, the scapula needs to be oriented more posteriorly, in line with the humeral motion. To allow this overall change in transverse plane orientation of the scapula, the composite SC and AC transverse plane positions must also change. During flexion, the SC joint will still undergo overall retraction, but there will be a slight reduction in retraction from the initial relaxed standing position, and less overall retraction during the elevation motion [6]. The opposite effect will occur in order to elevate the arm in coronal plane abduction. The SC joint will additionally increase retraction from the initial relaxed standing position and undergo slightly more retraction during the abduction motion [6] in order to optimally align the glenoid with the humeral plane of elevation. Since functional reaching occurs in a plane anterior to the scapular plane but posterior of a flexion plane [10], SC retraction during functional reaching would be expected to be intermediate between that of the respective planar motions.

The final rotation of the SC joint that occurs during elevation of the arm in any plane is clavicle elevation. In healthy shoulder motion, however, this rotation should be small. To elevate the arm to about 120°, less than 10° of SC elevation should occur. Increased SC elevation associated with excess upper trapezius activation is a common movement compensation seen in patients and will be discussed later.

During elevation of the arm in any plane from flexion to abduction, as well as during functional reaching, the AC joint also demonstrates characteristic patterns of motion. The primary motions

that occur at the AC joint as the arm is elevated are upward rotation and posterior tilt [2, 6]. In raising the arm to 120° of elevation in any plane, about 15° of upward rotation and 20° of posterior tilt will occur at the AC joint. Additional posterior tilt will occur to obtain higher degrees of arm elevation. Only subtle changes in the amount or pattern of these motions occur as the plane of elevation changes from flexion to abduction [6].

Historically, it was not believed that substantive motion occurred at the AC joint during functional elevation of the arm. This was largely presumed due to the potential restriction of AC joint upward rotation by tension in the coracoclavicular ligaments. However, we now know substantive motions of the AC joint are normal during functional motion. Rather than acting solely to restrict AC joint rotations, we also now know that a main function of the coracoclavicular ligaments may be to transmit rotations from the scapula to the clavicle [11]. For example, to our knowledge no SC musculature contributes directly to the posterior rotation motion of the clavicle, which is its primary motion. This motion is likely largely produced by tension in the coracoclavicular ligaments when the AC joint is moved into upward rotation and posterior tilt by the torque created by the lower serratus anterior muscle acting on the scapula.

Finally, as elevation of the arm occurs in any plane as described above, the AC joint undergoes internal rotation. The amount of rotation that can be expected to reach 120° of arm elevation during scapular plane abduction is about 10°. Similar to the SC transverse plane rotation of retraction, the amount of AC internal rotation that occurs will be directly impacted by the plane of elevation of the arm. As described previously, in order to accommodate the necessary glenoid orientation for elevation in flexion versus abduction, the AC joint position and motion must change. During flexion, there will be an initial increase in internal rotation from the initial relaxed standing position and more overall internal rotation during the elevation motion. The opposite effect will occur in order to elevate the arm in coronal plane abduction. While it will still internally rotate overall, the AC joint will begin in less internal rotation at the initial relaxed standing position and undergo slightly less internal rotation during the abduction motion [6] in order to optimally align the glenoid with the humeral plane of elevation.

Coupling of SC and AC Joint Motions

One of the most difficult concepts to understand regarding shoulder complex motion is how the individual SC and AC joint rotations combine or "couple" to result in the overall position and motion of the scapula on the thorax [9]. When viewed from above, the long axis of the clavicle and the lateral scapular axis (approximately aligned with the spine of the scapula) are aligned obliquely to each other (Fig. 2.4). In normal relaxed standing, the angle between these two axes (corresponding to AC joint internal rotation as noted above) is typically about 60° (Fig. 2.4). As such, with exception to the vertical axis, motions about any specific SC joint rotation axis will not correspond to motions about any specific AC joint axis and vice versa. However, the SC and AC joint vertical axes are approximately aligned regardless of the AC joint internal rotation angle, and subsequently their respective transverse plane motions are typically easier to interpret [9].

Further, recall as previously described that the AC joint axes are defined consistently with the axes describing scapulothoracic motion. Subsequently, as the AC joint upwardly rotates, posteriorly tilts, and internally rotates during elevation of the arm, these motions will directly couple to similarly named scapulothoracic joint motions if not "offset" in any way by motion at the SC joint. Therefore, if the AC joint were the only joint contributing to scapular motion on the thorax, during normal arm raising to 120° humerothoracic elevation, we would see scapulothoracic upward rotation of about 15°, scapulothoracic posterior tilt of about 20°, and scapulothoracic internal rotation of about 10°. The amount of scapulothoracic internal rotation would also depend on the plane of elevation, as described for the AC joint, with more occurring in flexion and less occurring in coronal plane abduction. As was presumed with SC retraction, the amount of AC internal rotation during functional reaching would be expected to be inter-

mediate in magnitude relative to that seen in flexion versus scapular plane abduction.

Also, recall the three rotations occurring at the SC joint during arm elevation are primarily posterior rotation, secondarily retraction, and finally elevation. Because the SC vertical axis is approximately aligned with the vertical axis for scapular motion on the thorax, if there were no offsetting motion of the AC joint, we would expect similar magnitudes of SC joint retraction and external rotation of the scapula on the thorax during arm elevation. However, we know that the AC joint internally rotates simultaneously as the SC joint retracts during arm elevation [6]. Thus, the transverse plane rotations of the SC and AC joint tend to offset one another in terms of overall scapular motion on the thorax. During scapular plane abduction, the net result is that very little change in scapulothoracic internal rotation alignment occurs. This is because the SC joint retraction that would cause scapulothoracic external rotation is offset by the AC joint internal rotation that would cause net scapulothoracic internal rotation. In later ranges of scapular plane abduction, the larger amount of SC retraction that occurs (approximately 15° overall) is more than the AC joint internal rotation that occurs (approximately 10° overall). This results in a net scapulothoracic motion of external rotation [6]. In contrast, during flexion, SC joint retraction is reduced and AC joint internal rotation is increased, and the net result is a limited amount of scapulothoracic internal rotation [6]. This scapulothoracic internal rotation helps to position the glenoid more anterior in better congruency with the forward-flexing humerus. During coronal plane abduction, SC joint retraction is increased and AC joint internal rotation is decreased, and the net result is a limited increase in scapulothoracic external rotation [6]. This scapulothoracic external rotation helps to position the glenoid more lateral during abduction in better congruency with the laterally abducting humerus.

In order to directly couple the remaining two SC single axis rotations of posterior rotation and elevation to single axis rotations of the scapula on the thorax, consider two hypothetical situations where the SC joint axes are directly aligned with the scapulothoracic joint axes (Fig. 2.7a, b). If the clavicle long axis were aligned with the scapular oblique lateral axis, such that the AC joint internal rotation angle was 0° (Fig. 2.7a), SC joint posterior rotation would directly couple with scapulothoracic posterior tilt, and SC joint elevation would directly couple with scapulothoracic upward rotation [9]. Alternatively, if the clavicle long axis were aligned perpendicular to the scapular oblique lateral axis such that the AC joint internal rotation angle was 90° (Fig. 2.7b), SC joint

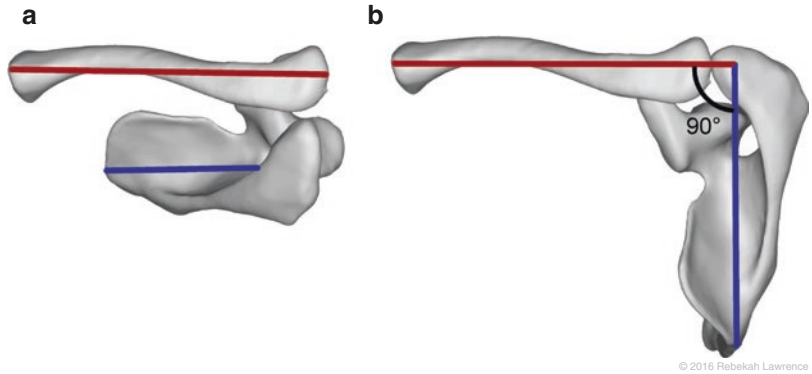

Fig. 2.7 Two hypothetical alignment scenarios between the clavicular and scapular lateral axes for understanding how individual sternoclavicular and acromioclavicular joint rotations couple to result in the overall position and motion of the scapula on the thorax: (**a**) axes parallel; (**b**) axes perpendicular. Parallel axis alignment would couple sternoclavicular elevation to scapulothoracic upward rotation and sternoclavicular posterior rotation to scapulothoracic posterior tilting. Perpendicular axis alignment would couple sternoclavicular elevation to scapulothoracic anterior tilting and sternoclavicular posterior rotation to scapulothoracic upward rotation. (Reproduced with permission of Rebekah L. Lawrence)

posterior rotation would directly couple with scapulothoracic upward rotation and SC joint elevation would directly couple with scapulothoracic anterior tilt (note that anterior tilt is not a desired motion, as will be discussed later in the section on scapular dyskinesis) [9]. We know of course that neither of these two hypothetical alignments occurs. Rather, the AC joint internal rotation angle is about 60° (Fig. 2.4), which is two-thirds of the way to being aligned with the second hypothetical scenario of a 90° internal rotation alignment. Subsequently, SC rotations couple in a complex way with scapulothoracic motion. Approximately two-thirds of SC joint posterior rotation will couple with scapulothoracic upward rotation (90° coupling relationship), and approximately one-third of SC joint posterior rotation will couple with scapulothoracic posterior rotation (0° coupling relationship) [9]. Similarly, approximately two-thirds of SC joint elevation will couple with scapulothoracic anterior tilt (90° coupling relationship), and approximately one-third of SC joint elevation will couple with scapulothoracic upward rotation (0° coupling relationship) [9].

As these complex coupling relationships can be difficult to visualize, a numeric example may help to clarify (Fig. 2.8). It is important to note that overall magnitudes in joint motion have been rounded in an effort to simplify the example. The SC and AC motions typically seen during arm elevation should be either additive toward overall scapulothoracic motion or act in ways to offset one another. For example, overall SC and AC joint rotations act additively toward scapulothoracic upward rotation (the primary rotation) and offset one another with regard to scapulothoracic internal/external rotation. Of the approximately 30° SC posterior rotation that occurs during elevation of the arm to 120°, 20° (2/3) of this motion will couple with scapulothoracic upward rotation. In addition, the AC joint upward rotation (typically 15°) will directly couple with scapulothoracic upward rotation. Finally, of the approximately 9° SC elevation that occurs during arm elevation, 3° (1/3) of this motion will couple with scapulothoracic upward rotation. In this scenario, these rotations in combination add to 38° of scapulothoracic upward rotation, which is a realistic value for this motion during scapular plane abduction of the arm [6, 7]. The 30° of SC posterior rotation that is occurring in this scenario would also contribute 10° (1/3) to scapulothoracic posterior tilt. However because the SC joint is also simultaneously elevating approximately 9°, 6° (2/3) of this motion would result in scapulothoracic anterior tilt and would therefore reduce the overall scapulothoracic posterior tilt to 4°. An additional 20° of posterior tilt at the AC joint would result in a combined total scapulothoracic posterior tilt in this scenario of 24°, again a realistic value [6, 7]. Finally, if the SC joint was retracting 15° during this arm elevation scenario, and the AC joint was internally rotating 10°, these transverse plane rotations would offset one another. The net result of this scenario would be scapulothoracic external rotation of 5°. During

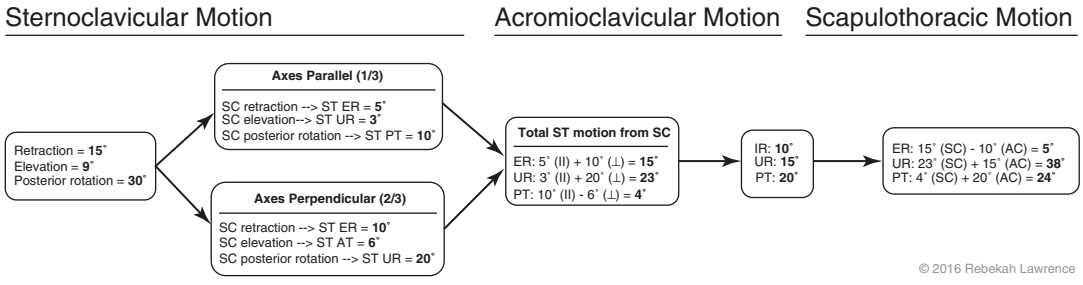

Fig. 2.8 Numeric example demonstrating how sternoclavicular motion is coupled with acromioclavicular motion to produce scapulothoracic motion during elevation of the arm to 120°. *Left box* identifies typical sternoclavicular motion magnitudes during humerothoracic elevation. *Middle boxes* quantify how sternoclavicular motions will couple to scapulothoracic motions based on axis alignment. Further acromioclavicular joint motions are additive to produce end result scapulothoracic joint motion in far *right box*. (Reproduced with permission of Rebekah L. Lawrence)

arm elevation to 120° in any plane ranging from flexion to abduction, the typical scapulothoracic motions described are approximately 40° upward rotation, 20° of posterior tilt, and small, variable amounts of internal or external rotation, depending on the plane and angle of elevation [6, 7].

In summary, it can be seen that during arm elevation, SC posterior rotation and AC upward rotation combine to produce the majority of overall scapulothoracic upward rotation motion that is observed [9]. These primary motions are complemented to a limited amount (<5°) by SC elevation. Sternoclavicular posterior rotation also contributes to scapulothoracic posterior tilt, but this contribution is largely offset by the corresponding SC elevation [9]. Thus, scapulothoracic posterior rotation is predominately produced by AC joint posterior rotation. Finally, SC retraction and AC internal rotation offset one another, resulting in more limited internal or external rotation motions depending on the plane of arm elevation [9].

While these coupling relationships are complex to describe, they are important to understand in optimally diagnosing and treating patients with shoulder pain related to scapular dysfunction. As the actual joint motions are occurring at the underlying SC and AC joint rather than the composite scapulothoracic "joint", muscle actions, ligamentous constraints, and joint reaction forces are influencing the motions at these underlying joints. Diagnostic and treatment approaches that consider these underlying joint component motions and functions have greatest potential to positively advance patient care and preventive approaches.

Abnormal Motions Identified in Patient Populations (Dyskinesis)

Once understanding the normal motions expected at the SC and AC joints during arm elevation, as well as overall scapular motion on the thorax, deviations from these motion patterns are typically considered as abnormal. In the case of the scapula, these abnormalities are frequently termed "scapular dyskinesis" [12]. A wide variety of abnormal motions [13, 14] have been identified in a number of studies of patients with shoulder pain associated with various pathologies. Identified abnormalities have included increased [15, 16, 17] and decreased [18] SC elevation, increased SC retraction [15], decreased SC posterior rotation [18], increased AC upward rotation and posterior tilt [19], increased [15] and decreased [18, 20, 21] scapulothoracic upward rotation, increased [15, 16] and decreased 21 scapulothoracic posterior tilt, and increased scapulothoracic internal rotation [21, 22]. Given the inconsistencies across the literature with regard to the direction of deviations, it is difficult to conclude if alterations observed are causative or compensatory [23]. Further, the small magnitude of changes compared to asymptomatic subjects has caused some to question if these alterations are in fact abnormal or merely an expected range of normal variation 24.

Our premise is that lack of consistent movement deviations across studies relates more to small and varying sample sizes [13, 14], limited precision of measurement techniques, and limited utility of pathoanatomic diagnoses as defining homogenous patient samples [25, 26], rather than a lack of true movement deviations in patient populations. However, research advances and further investigation are needed before definitive conclusions can be reached. Our premise is that scapular position and motion deviations can result in deleterious pathomechanics when present in combination with repetitive movement exposure. Some common clinical presentations of movement deviations are described below.

Increased SC elevation is commonly observed in patients "shrugging" their shoulder in attempt to raise the arm (Fig. 2.9). This deviation results from overuse of the upper trapezius in a compensatory pattern that may occur because of rotator cuff tears (Fig. 2.9), capsular adhesions limiting glenohumeral joint motion, glenohumeral osteoarthritis, or a number of other conditions [15, 16, 17, 27]. Because SC elevation predominately couples with scapulothoracic anterior tilt, this movement deviation is generally considered a negative compensatory strategy as it may further limit the normal posterior tilt of the scapula on the thorax.

Decreased SC elevation can also be observed in some patient populations [18]. This is often a postural deviation whereby the clavicle is not in

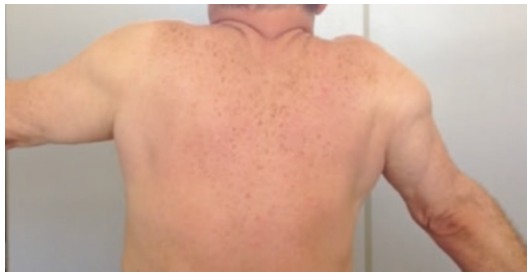

Fig. 2.9 Patient demonstrating bilateral shoulder "shrugging" in attempt to raise his arms overhead. This motion is produced through increased upper trapezius activation and increased sternoclavicular joint elevation. (Reproduced with permission of Paula M. Ludewig)

the typical slight elevation position when the arm is relaxed at the side in standing. This deviation may be associated with reduced scapulothoracic upward rotation and increased scapular internal rotation, which has been described as a "SICK" scapula phenomenon [28].

Increased SC retraction has also been identified in patient populations [15]. This deviation has been identified in combination with increased SC elevation [15], suggesting that increased upper trapezius activation may be contributing to these two deviations in combination.

As SC posterior rotation is difficult to accurately measure by noninvasive means, little investigation of this component motion has occurred in patient populations. One investigation using bone-fixed tracking sensors [18] did identify significant reductions in SC posterior rotation in patients fitting a clinical description of shoulder "impingement." As SC joint posterior rotation couples with scapulothoracic upward rotation, this finding is believe related to the common finding of decreased scapulothoracic upward rotation in patient populations.

Acromioclavicular joint deviations are also difficult to measure accurately by noninvasive means. One study [19] did identify significantly greater AC upward rotation and posterior tilting in patients with AC joint arthritis performing arm elevation. Another single-subject analysis demonstrated increased AC joint motions of upward rotation and posterior tilt in a patient with glenohumeral osteoarthritis [29].

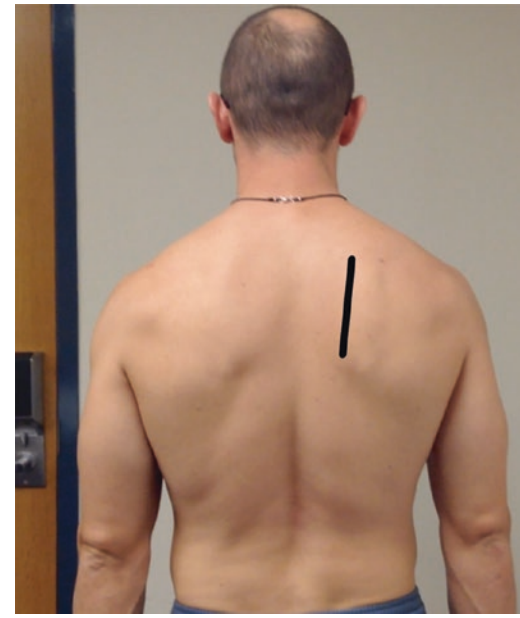

Fig. 2.10 Patient demonstrating decreased scapular upward rotation. Line on right scapular medial border depicts downward slope. (Reproduced with permission of Paula M. Ludewig)

A number of investigations have identified decreased scapulothoracic upward rotation in shoulder pain populations ([13, 14, 18, 20, 21, 27, 30, 31] (Fig. 2.10). Most commonly, these reductions occurred at lower angles of arm elevation [14, 20, 21]. Reduced upward rotation has been frequently presumed to contribute to development of subacromial or internal "impingement" conditions [13], as well as to inferior or multidirectional instability [30–32].

Alternatively, studies have also identified increased scapulothoracic upward rotation in patient populations [15, 33]. This seemingly contradictory finding may relate to increased upward rotation being a compensatory rather than a causative deviation for shoulder pain and pathology. This premise is supported by findings of increased scapulothoracic upward rotation in patients with rotator cuff tears [33], normalization of scapular kinematics after rotator cuff tear surgery [34], and in a study demonstrating increased upward rotation in healthy subjects after a suprascapular nerve block [35]. Increased scapulothoracic upward rotation may also be a compensatory

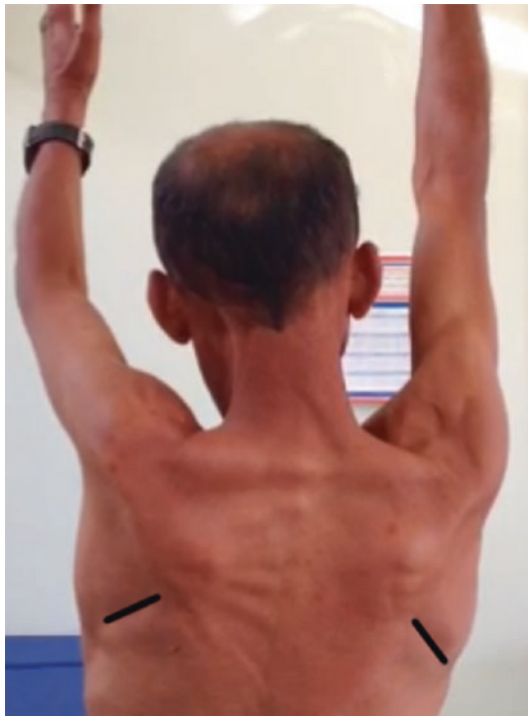

Fig. 2.11 Patient demonstrating increased scapular upward rotation on the left shoulder, secondary to glenohumeral joint soft tissue stiffness. Lines denote scapular medial or vertebral borders bilaterally. (Reproduced with permission of Paula M. Ludewig)

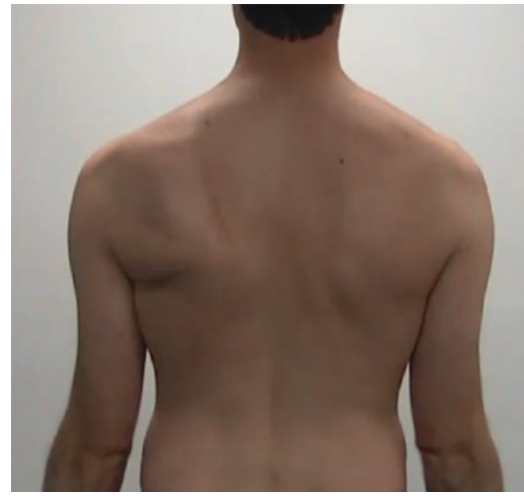

Fig. 2.12 Patient demonstrating increased scapular internal rotation as noted by prominence of scapular medial or vertebral border on the left. The patient is also demonstrating reduced scapular upward rotation as noted by the downward slope of the scapular medial border on the left. (Reproduced with permission of Paula M. Ludewig)

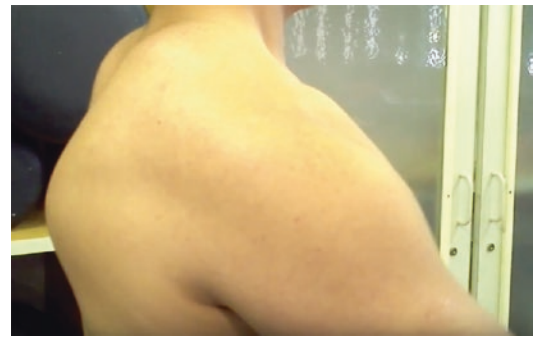

Fig. 2.13 Patient presenting with increased anterior tilt (decreased posterior tilt) on the right shoulder during flexion. (Reproduced with permission of Paula M. Ludewig)

movement for subjects with reduced glenohumeral motion due to osteoarthritis [29, 36] or soft tissue tightness (Fig. 2.11, [36, 37]).

Both increased [15, 16] and decreased scapulothoracic posterior tilt [17, 20, 21, 27] and increased [21, 22, 30, 38] scapulothoracic internal rotation (Fig. 2.12) have also been observed in patient populations. These disparate findings further illustrate the need to better distinguish causative versus compensatory versus inconsequential movement deviations with regard to pain and function.

Anecdotally, lack of posterior tilt (or even increased anterior tilt) (Fig. 2.13), increased internal rotation, or decreased upward rotation in some patients may relate to reverse action of the deltoid. As any muscle contracts, it imparts force on both proximal and distal attachment sites. Typically a muscle's distal attachment is to a segment of lesser mass, and subsequently the segment is moved toward the proximal attachment. However, in the case of the deltoid with a proximal attachment to the scapula, the lighter scapula will be pulled into anterior tilt or downward rotation if the scapulothoracic musculature is not adequately activated or is unable to produce enough force. This may be an activation or timing

issue rather than a strength issue. A further clinical observation worth noting is that scapular dyskinesis may be more commonly observed eccentrically [39].

Potential Influence of Abnormal Scapular Motions and Positions on Shoulder Pain and Tissue Pathology

How scapular motion and position relate to potential for shoulder pain and tissue pathology has received limited investigation. Because the glenohumeral joint is where the majority of tissue pathology is observed, the impact of scapulothoracic deviations largely depends on whether the glenohumeral joint is impacted. If scapular dyskinesis is occurring but the humerus is moving synchronously with the scapula, there may be no negative impact. However, if the scapula is dyskinetic and consequently there is increased glenohumeral joint rotational or translational motion, there may be increased stress to glenohumeral joint structures.

One key factor to determining whether scapular dyskinesis is deleterious or not relates to whether it impacts glenohumeral joint stability. The glenohumeral joint is most stable if the net result of the joint contact force is directed into the glenoid at the center of its concavity [40]. If scapular position or dyskinesis alters the net joint resultant force direction, this can contribute to instability, subluxation, or dislocation at the glenohumeral joint. Less extreme cases of "microinstability" can also occur, where scapular dyskinesis may contribute to excessive translations at the glenohumeral joint.

Most extensively, scapular dyskinesis has been theorized to increase risk for subacromial compression and internal or external "impingement." The generalized negative impact of dyskinesis is supported with evidence of tissue pathology development in an animal model [41]. Historically, measures of the acromiohumeral distance have been used to investigate whether abnormal scapular kinematics were negatively impacting the rotator cuff [42, 43]. However, recently it has been clarified that acromiohumeral distance measures need to consider the proximity to actual rotator cuff soft tissue structures [44, 45] in order to best understand potential risk of cuff compression. To date, clear links between subacromial rotator cuff compression and specific scapular dyskinesis *in vivo* have not been established [23, 42, 43]. With regard to internal impingement, Mihata et al. have demonstrated negative implications of reduced scapular upward rotation and increased scapular internal rotation in a cadaver model [46]. Additional investigations linking scapular motion and position alterations and the proximity of the rotator cuff to potential impinging structures, as well as finite element models assessing tissue stress and deformation, are needed. Such studies can further ascertain the clinical consequences of the position and motion alterations identified in patient populations.

In summary, scapular dyskinesis is not necessarily a pathology in and of itself (e.g., as related to a spinal accessory or long thoracic nerve injury) [47]. However, scapular dyskinesis may contribute to abnormal joint stresses and eventually lead to tissue pathology. While much further research is needed, we believe scapular dyskinesis is an impairment of optimal motion and can be a risk factor for shoulder tissue pathology. Everyone who presents with a risk factor will not necessarily develop pathology. In the case of shoulder joint pathology, determining who will go on to develop tissue pathology is likely based on a combination from a number of risk factors. These might include an individual's underlying anatomical structure and alignment, their shoulder motion profile, their tissue resilience to repetitive stress including blood flow and inflammatory responses, genetic factors, and their overall exposure to shoulder positions and motions creating risk. Full understanding of these risk factors and their implications will require substantive ongoing investigation.

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3 Muscle Activation Associated with Scapular Function and Dysfunction

David Ebaugh and Margaret Finley

This chapter will discuss the role of muscles involved with the production and control of scapulothoracic and glenohumeral joint movements. The focus will be on the role of the **primary** muscles and muscle force couples involved with scapulothoracic and glenohumeral movements. The effects of altered muscle activity and muscle inflexibility on scapulothoracic movement will also be presented along with clinical implications for rehabilitation guidelines.

Normal shoulder girdle motion is dependent upon the coordinated interaction of the scapulothoracic and glenohumeral joints. For example, during glenohumeral abduction (frontal plane elevation), the typical scapulothoracic movement pattern includes elevation, retraction, upward rotation, posterior tilt, and external rotation [1]. These scapulothoracic movements are necessary for maintaining optimal alignment between the humeral head and glenoid fossa, optimal size of the subacromial space, ideal length-tension relationship of the rotator cuff muscles, and full-range arm elevation.

Seventeen muscles attach to the scapula. Five muscles, the trapezius (upper, middle, and lower portions), levator scapulae, rhomboids, serratus anterior, and pectoralis minor, are primarily responsible for producing and controlling scapulothoracic movement. The supraspinatus, infraspinatus, subscapularis, teres minor, and deltoid are primarily responsible for producing glenohumeral movement. Little evidence exists regarding the role that other periscapular muscles (latissimus dorsi, pectoralis major, triceps brachii [long head], biceps brachii [short and long head], coracobrachialis, and omohyoid) have in producing or controlling scapulothoracic movement. In the following paragraphs, we will describe the role of the **primary** muscles that are involved with scapulothoracic and glenohumeral movements and how these muscles work together to produce arm elevation.

Translational movements of the scapula on the thorax (elevation, depression, protraction, and retraction) can occur without concomitant glenohumeral movements. Scapular elevation (shoulder shrug) is produced by the upper portion of the trapezius, levator scapulae, and rhomboid muscles [2, 3] (Fig. 3.1a). The attachment of the upper trapezius muscle on the lateral aspect of the clavicle lends it to provide simultaneous

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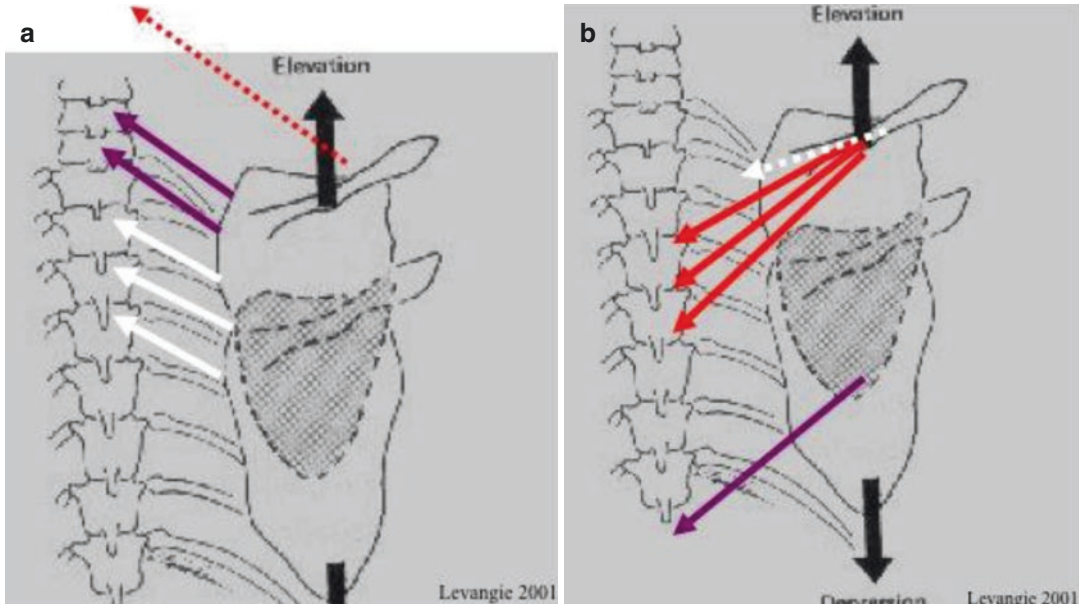

Fig. 3.1 (**a**) Muscles responsible for producing scapular elevation. *Dashed red line* = upper trapezius; *solid purple lines* = levator scapulae; *solid white lines* = rhomboids. (**b**) Muscles responsible for producing scapular depression. *Dashed white line* = pectoralis major and minor; *solid red lines* = lower trapezius; *solid purple line* = latissimus dorsi (From "Joint Structure and Function: A Comprehensive Analysis", 3rd edition by Pamela K. Levangie and Cynthia C. Norkin. ISBN: 0803607105, 9780803607101)

upward rotation of the scapula as it elevates the scapula. Concomitant scapular downward rotation may occur based on the attachment sites of the levator scapula and rhomboid muscles. Depending on the relative activity of the upper trapezius, rhomboids, and levator scapulae muscles, scapular elevation may be accompanied with upward rotation and downward rotation or may occur with the scapula remaining in a relatively neutral upwardly/downwardly rotated position.

The lower trapezius and pectoralis minor muscles, along with the latissimus dorsi and lower portion of the pectoralis major muscles, produce forceful depression of the scapula that is accompanied by scapular downward rotation [4, 5] (Fig. 3.1b). The balance of muscle activity between the anterior muscles (pectoralis minor and major) and posterior muscles (trapezius and latissimus dorsi) will determine whether the scapula protracts, retracts, or remains in a neutral position as the scapula moves into depression.

Scapular protraction is produced by the pectoralis minor, serratus anterior, and pectoralis major muscles [6, 7] (Fig. 3.2a). Based on the relative amount of activity in each of these muscles, other scapular movements (upward/downward rotation, internal/external rotation, and anterior/posterior tilt) may occur simultaneously with scapular protraction. For example, if the pectoralis minor and major muscles dominate the motion then based upon their attachments on the scapula, scapular protraction will be accompanied by scapular downward rotation, internal rotation, and anterior tilt. Conversely, orientation of the serratus anterior muscle allows it to upwardly rotate, externally rotate, and posteriorly tilt the scapula as it protracts the scapula.

Scapular retraction is produced by the trapezius, rhomboids, and latissimus dorsi muscles [2, 3] (Fig. 3.2b). As the rhomboids and latissimus dorsi muscles create scapular retraction, they also produce scapular downward rotation. The ability of the trapezius muscle to upwardly rotate the scapula during retraction can counter this downward rotation, thereby keeping the scapula in a neutral upwardly/downwardly rotated position.

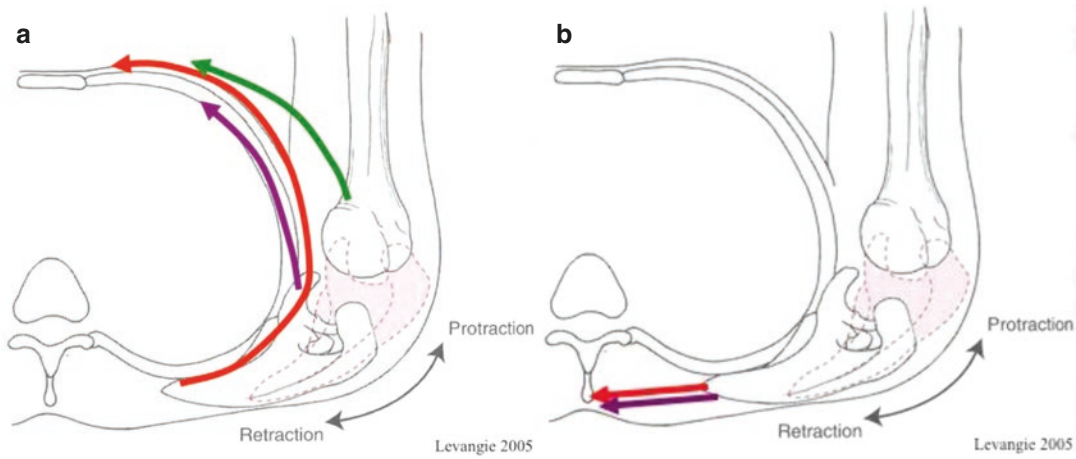

Fig. 3.2 (**a**) Muscles responsible for producing scapular protraction. *Green arrow* = pectoralis major; *red arrow* = serratus anterior; *purple arrow* = pectoralis minor. (**b**) Muscles responsible for producing scapular retraction. *Red arrow* = rhomboids; *purple arrow* = middle trapezius (From "Joint Structure and Function: A Comprehensive Analysis", 4th edition by Pamela K. Levangie and Cynthia C. Norkin. ISBN: 0803611919, 9780803611917)

The deltoid (anterior, middle, and posterior) along with the rotator cuff muscles (subscapularis, supraspinatus, infraspinatus, and teres minor) are the primary muscles that produce glenohumeral movements and provide glenohumeral stability. Glenohumeral elevation in the sagittal plane (flexion) occurs through activation of the anterior and middle portions of the deltoid muscle [8–10] with stabilizing contributions from the infraspinatus, supraspinatus, and subscapularis muscles [11–13]. The anterior and middle portions of the deltoid muscle [10, 14, 15] and the supraspinatus muscle [9, 11, 16] are the primary muscles responsible for producing frontal plane glenohumeral elevation (abduction) with stability being provided by the infraspinatus and subscapularis muscles [11–13, 16–18].

The muscles primarily responsible for producing glenohumeral internal and external rotation vary based upon the angle of arm elevation. The infraspinatus muscle is primarily responsible for producing glenohumeral external rotation in neutral (arm in 0° elevation) and at 90° of arm elevation [10, 18]. The supraspinatus [19] as well as the teres minor and posterior deltoid muscles assist the infraspinatus muscle during these movements [8]. Similarly, glenohumeral internal rotation is produced by the subscapularis muscle when the arm is at the side with additional contributions from the supraspinatus [11], middle deltoid [11], and pectoralis major muscles [8] when the arm is at 90° of elevation.

The anterior deltoid along with the pectoralis major and subscapularis muscles produces glenohumeral horizontal adduction [18, 20]. The posterior deltoid and infraspinatus muscles produce glenohumeral horizontal abduction [18, 20, 21].

Arm elevation can occur in many planes including the frontal, sagittal, and scapular plane. Poppen and Walker [22] suggest that scapular plane elevation, defined as 30°–45° anterior to the frontal plane, provides optimal glenohumeral joint congruity which enhances joint stability and maintains an ideal muscle length-tension relationship of the glenohumeral musculature. Thus, most overhead activities are performed in the scapular plane and require a coordinated balance of scapulothoracic and glenohumeral movements [22, 23]. These movements include scapulothoracic upward rotation, external rotation, and posterior tilt, along with glenohumeral elevation and external rotation [2, 24–27].

As the arm is elevated to an overhead position, the primary scapulothoracic motion is upward rotation. The traditionally described muscle force couple responsible for producing this movement consists of the upper and lower portions of the trapezius muscle along with the serratus anterior

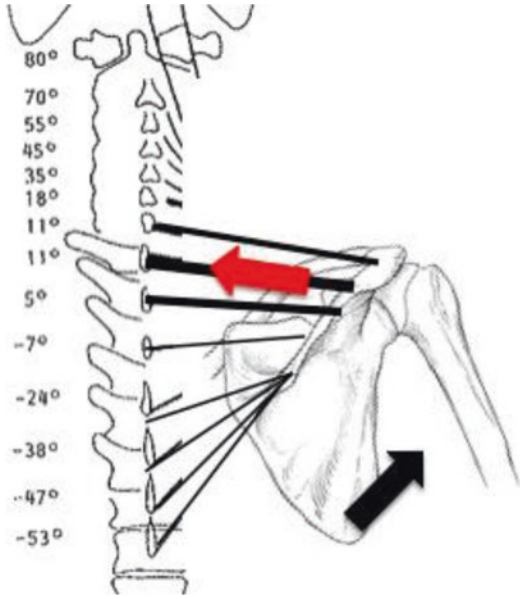

Fig. 3.3 Muscles responsible for producing scapula upward rotation. *Red arrow* = middle trapezius; *black arrow* = lower portion of serratus anterior (From Johnson G, Bogduk N, Nowitzke A, House D. Anatomy and actions of the trapezius muscle. Clin Biomech (Bristol, Avon). 1994;9(1):44–50)

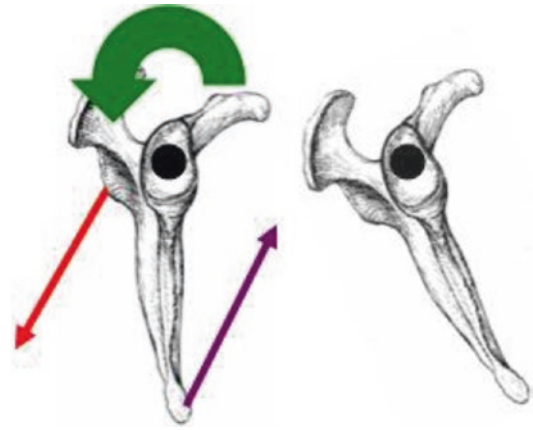

Fig. 3.4 Muscles responsible for producing scapula posterior tilt. *Black dot* = axis of rotation; *green arrow* = posterior tilt motion; *purple arrow* = serratus anterior; *red arrow* = lower trapezius

muscle. Contrary to this, Johnson et al. [3] purport that the middle trapezius muscle works with the serratus anterior muscle in a force couple that produces scapular upward rotation. Based on a cadaveric study, the authors proposed that once the serratus anterior muscle initiated upward rotation, the middle trapezius was optimally aligned to assist with upward rotation [3]. The serratus anterior and middle trapezius muscles then continue to work in a force couple to upwardly rotate the scapula as the arm is raised overhead (Fig. 3.3). The role of the lower trapezius was proposed to be one of scapular stabilization by offsetting scapular elevation and protraction produced by the upper trapezius and serratus anterior muscle, while the upper trapezius muscle was proposed to be one of clavicular and scapular elevation and retraction [3].

At end ranges of scapular plane arm elevation, the scapula posteriorly tilts and externally rotates [27]. The serratus anterior, rhomboids, and trapezius muscles work together as a force couple to produce these motions [14, 26, 28, 29]. It is the extensive attachment on the inferior angle of the scapula that places the lower portion of the serratus anterior muscle in an ideal orientation to produce scapular posterior tilt. The lower trapezius muscle's attachment from the lower thoracic spinous processes to the deltoid tubercle on the scapular spine provides the ability to work with the lower portion of the serratus anterior to produce scapular posterior tilt (Fig. 3.4). Scapular external rotation results from a force couple created by the serratus anterior and rhomboid muscles. These muscle's attachments to the vertebral border of the scapula are aligned to produce scapular external rotation (Fig. 3.5).

The contribution of the glenohumeral joint to overhead arm motions requires a balance of adequate joint mobility and stability. Glenohumeral joint stability relies heavily upon the concavity compression mechanism [30]. The concavity compression mechanism refers to the stabilizing effect of the concave glenoid fossa and compressive forces of the rotator cuff musculature on humeral head translations. Other factors that contribute to this mechanism are glenoid articular cartilage thickness and the glenoid labrum [30].

As the arm is raised, overhead glenohumeral elevation is accomplished through a force couple formed by the deltoid and the rotator cuff muscles (Fig. 3.6). During the initial phases of glenohumeral elevation, the deltoid muscle functions

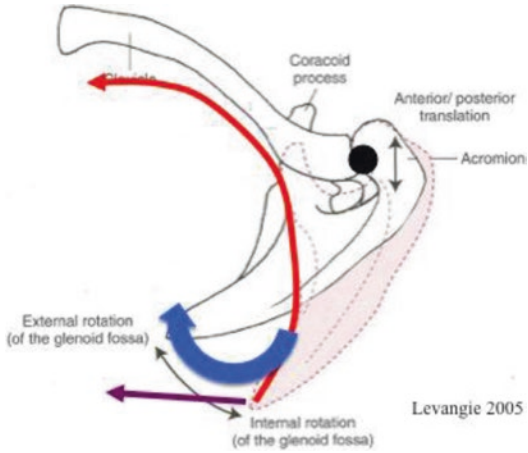

Fig. 3.5 Muscles responsible for producing scapular external rotation. *Black dot* = axis of rotation; *blue arrow* = external rotation motion; *red arrow* = serratus anterior muscle; *purple arrow* = rhomboids (From "Joint Structure and Function: A Comprehensive Analysis", 4th edition by Pamela K. Levangie and Cynthia C. Norkin. ISBN: 0803611919, 9780803611917)

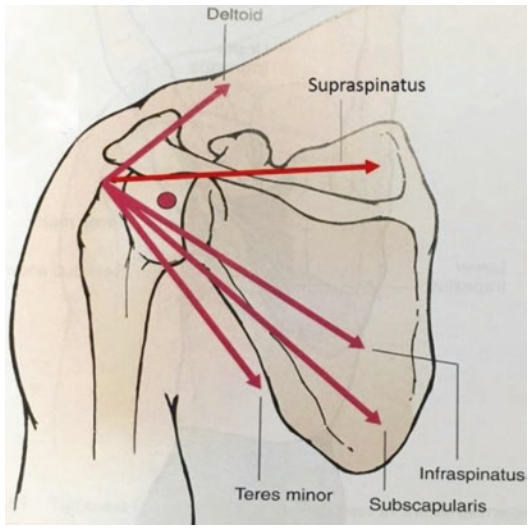

Fig. 3.6 Force couple formed by the deltoid and rotator cuff muscles producing glenohumeral elevation (From "Kinesiology, The Mechanics and Pathomechanics of Human Movement", 3rd edition by Carol A. Oatis. ISBN: 978-1-4511-9156-1)

to elevate the humerus, while the rotator cuff muscles stabilize the glenohumeral joint by compressing the humeral head into the glenoid fossa [15, 16, 31, 32]. Additionally, the supraspinatus muscle assists the deltoid in producing arm elevation, while the infraspinatus and teres minor muscles produce humeral external rotation toward the end range of arm elevation.

Alteration in Muscle Activations and Resultant Motions

Appropriate muscle activity is necessary for producing coordinated scapulothoracic and glenohumeral movements during arm elevation. Neuromuscular lesions such as nerve entrapment, neuritis, or nerve lesions result in altered muscle activation that in turn produces aberrant movements. Specifically, altered scapulothoracic and glenohumeral movements have been documented in impairments of the long thoracic nerve (serratus anterior) [33], spinal accessory nerve (trapezius) [33], dorsal scapular nerve (rhomboids) [34, 35], and suprascapular nerve (supraspinatus, infraspinatus) [36, 37].

Roren et al. [33] investigated scapulothoracic movements during arm elevation in individuals with long thoracic nerve palsy (LTNP, $n = 5$) and spinal accessory nerve palsy (SANP, $n = 4$). Elevation in both the sagittal (flexion) and frontal planes (abduction) resulted in reduced scapulothoracic upward rotation (3.4°–13°), decreased scapulothoracic posterior tilt (3.6°–8.8°), and small reductions in scapulothoracic internal rotation (<3.5°) in those with LTNP [33]. Similarly, in individuals with SANP, scapulothoracic upward rotation was reduced (9.2°–28.6°) with increased scapulothoracic internal rotation (18.5°–20.3°) across both planes. Throughout frontal plane elevation, scapulothoracic posterior tilt was reduced at 2.4°–8.9°; however, at rest and in the initial phases of sagittal plane elevation, posterior tilt increased (2.6°–5.6°) followed by a decrease in the range above 90° of elevation (2.1°) [33]. The aberrant movement patterns observed with these nerve injuries are commonly described in the clinical setting as dynamic scapular winging.

Individuals with electrodiagnostically confirmed dorsal scapular nerve lesions have been shown to have altered resting scapular position as well as aberrant movements [34, 35]. On visual

examination of resting scapular position, the medial scapular border and inferior angle were prominent with the involved scapula located more laterally on the thorax. Individuals presented with increasing scapular winging (medial scapular border lifting off the posterior thoracic wall creating scapulothoracic internal rotation with medial and superior translation) during sagittal and frontal plane arm elevation. Findings of these studies support the importance of the rhomboid muscles in assisting with the production of scapulothoracic retraction and external rotation.

Although neither the supraspinatus nor infraspinatus muscles are primary producers of scapular movements, suprascapular nerve impairment induced by nerve block in healthy adults [36, 37] and in Parsonage-Turner syndrome [38] has been shown to result in aberrant scapulothoracic and glenohumeral movements during scapular plane arm elevation. Suprascapular nerve block resulted in an increase in scapulothoracic upward rotation, reduced glenohumeral elevation during the initial 90° of arm elevation [36, 37], increased scapulothoracic external rotation from 70° to 120° of humerothoracic elevation [36], and superior humeral head translation [37]. In a case of suprascapular neuropathy, Camargo et al. [38] documented increased scapulothoracic upward rotation and internal rotation, as well as decreased scapulothoracic posterior tilt, without a deficit in arm elevation range of motion. These studies support the idea that loss of adequate supraspinatus and infraspinatus muscle activation results in compensatory changes of scapular movements that are believed to be important for elevating the acromion during arm elevation.

Soft Tissue Flexibility

Impaired flexibility of soft tissues associated with the shoulder girdle has been proposed to influence the position and movement of the scapula [39]. The pectoralis minor muscle and posterior rotator cuff musculature/posterior glenohumeral joint capsule have been the focus of recent studies and will be discussed in this section.

Pectoralis minor muscle length is believed to effect the resting position of the scapula on the thorax and scapulothoracic movement [39, 40]. The relationship between resting pectoralis minor muscle length and scapular resting position as well as scapulothoracic movement has been studied in a healthy, young population [41–43]. When standing in a natural relaxed posture, individuals with a shorter resting pectoralis minor muscle length have been shown to have more scapular internal rotation than individuals with a longer resting pectoralis minor muscle length [41]. Additionally, individuals with a shorter resting muscle length also demonstrate reduced scapular upward rotation and scapular posterior tilting during arm elevation [43]. The significance of this information is that these scapulothoracic motion patterns are similar to those reported in individuals with shoulder pain secondary to subacromial impingement, rotator cuff disease, and glenohumeral instability [28, 44].

It should be noted that although studies have investigated associations between resting pectoralis minor muscle length, scapular positioning, shoulder pain, and scapulothoracic movement, pectoralis minor muscle length measures were obtained with participants in a standing or supine position with their arms at their sides [43, 45, 46]. Although this position provides information about the resting length of the pectoralis minor muscle, it does not provide information about whether or not the muscle is tight or shortened. While resting pectoralis minor muscle length provides useful information related to scapular positioning and scapulothoracic movement, determining whether or not the pectoralis minor muscle is tight or shortened could provide other valuable information for clinical decision-making. A tight or shortened pectoralis minor muscle could interfere with normal lengthening of the muscle during overhead arm movements. Based on a modeling study, the pectoralis minor muscle has been shown to elongate up to 67% of its resting length during full overhead arm elevation [14]. Clearly additional studies are needed to determine what constitutes a tight or shortened pectoralis minor muscle and what effect these conditions have on scapulothoracic movement and shoulder function.

Measures of glenohumeral internal rotation and horizontal adduction, as well as ultrasound

measures of posterior glenohumeral joint capsule thickness in overhead athletes, have been used to provide an understanding of the influence of impaired posterior shoulder soft tissue flexibility on scapular positioning and scapulothoracic movement [47–50]. Overhead athletes who present with limited glenohumeral horizontal adduction have been shown to have a more forward scapular position (protraction and anterior tilt) in standing than those with greater amounts of glenohumeral horizontal adduction [48]. Increased amounts of scapulothoracic anterior tilt have been demonstrated in athletes with limited glenohumeral internal rotation deficit (GIRD) during a movement task where participants were asked to move their shoulder from maximum external rotation to maximum internal rotation while maintaining their shoulder in 90° of elevation [47]. Conflicting data exists with regard to the effect of impaired posterior shoulder soft tissue flexibility on scapulothoracic upward rotation. Using measures of GIRD, Thomas et al. [50] demonstrated that athletes with GIRD >15° demonstrated less scapulothoracic upward rotation at 60°, 90°, and 120° of arm elevation. However, in their 2011 study, Thomas et al. [49] reported that athletes with a thicker posterior glenohumeral joint capsule (measured with ultrasound) had more scapulothoracic upward rotation at 60°, 90°, and 120° of arm elevation. These differences may, in part, be explained by different measures of posterior shoulder tissue flexibility. A direct measure (ultrasound) of posterior glenohumeral joint capsule thickness may be a better way to assess tissue characteristics associated with tissue flexibility as opposed to a measure of glenohumeral internal rotation as this measure can be influenced by humeral retroversion. Collectively these studies provide evidence to support the idea that impaired posterior shoulder soft tissue flexibility influences scapular position and scapulothoracic motion, as well as a basis for future research.

Clinical Implications for Rehabilitation Guidelines

Normal shoulder girdle motion is dependent upon coordinated activity of the scapulothoracic and glenohumeral musculature to produce and control scapulothoracic and glenohumeral movements. From a clinical perspective, it is imperative to recognize this when examining an individual with shoulder pain and dysfunction. Understanding how the primary shoulder girdle muscles work together to produce and control scapulothoracic and glenohumeral movements, and what happens when impairments to these muscles occurs, gives clinicians a strong basis for evaluating and treating individuals with shoulder pain and dysfunction as well as designing rehabilitation interventions.

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Scapular Examination

4

Phil McClure, Aaron D. Sciascia, and Tim L. Uhl

Scapular Motion and Pathology

Recognition of the biomechanical role of the scapula in normal shoulder function has led to several clinical studies attempting to associate abnormal scapular motion, so-called scapular dyskinesis, with shoulder pathology such as shoulder impingement [1–6] or instability [6]. These studies have included several methods of capturing scapular motion including Moire' topography, electromechanical digitization, radiographic methods, magnetic resonance imaging, and electromagnetic tracking devices. Results of studies assessing three-dimensional (3-D) scapular motion in those with pathology have been inconsistent. Subjects with shoulder impingement have been found to demonstrate increased posterior tilting [7, 8], decreased posterior tilting [1, 3, 4], decreased upward rotation [1, 3], increased upward rotation [2, 8], increased superior translation [4, 8], and increased internal rotation [3, 6]. The variability of findings in these studies is further confusing as the magnitude of differences between those with healthy and pathological shoulders are typically small, with differences in the 3–5° range. Therefore, it is unclear whether these differences are really of clinical significance even though statistical significance was observed in several of the studies. Furthermore, recent prospective investigations have found conflicting results regarding the relationship between scapular dyskinesis and the presence of symptoms in overhead athletes. Two studies found no relationship between the presence of scapular dyskinesis and shoulder symptoms in high school baseball players [9] and collegiate water polo players [10]. Clarsen et al. [11] did find a positive relationship between scapular dyskinesis and development of shoulder symptoms over a season in Norwegian elite handball players. Therefore, despite some authors claiming a strong relationship between abnormal scapular motion and shoulder pathology [12–14], the actual research evidence supporting this assertion is limited. Other clinical tests predicated on altering symptoms with manual scapula repositioning may hold promise in clarifying which patients truly have scapular dysfunction driving symptoms [15–17].

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Examination

The scapular examination should be carried out in association with a complete shoulder examination. The goal is to identify abnormal scapular position and motions (dyskinesis) that may be associated with underlying shoulder dysfunction [18–20]. Because scapular dyskinesis is commonly present in asymptomatic subjects, relating it's presence to the patient's symptoms is critical. The assessment of scapular dyskinesis is challenging due to motion of the scapula underlying skin and other soft tissues. The three-dimensional motion of the scapula that is dependent on plane of elevation and motions performed further challenges the examiner. Methods of identifying scapular dyskinesis have been described with adequate reliability, though the validity of these tests may be questioned because they lack direct correlations with symptoms [18]. The premise of this chapter is to outline a systematic clinical examination of the scapula incorporating three major components:

1. Visual observation to determine the presence or absence of scapular dyskinesis in a symptomatic patient
2. The effect of manual corrections of scapular position and motion on symptoms
3. Evaluation of surrounding tissue that may contribute to scapular and shoulder dysfunctions [18]

The integration of these components is detailed in this chapter and is summarized in an assessment algorithm in Fig. 4.1.

Visual Observation

The scapula must be directly visualized during the examination. One common mistake is failure to visually inspect the scapula position at rest from the posterior view (Fig. 4.2). This must be done in order to comprehensively evaluate a patient with shoulder pain. Visual observations of the scapula at rest as part of a typical postural evaluation must take into consideration the cranial and spinal alignment issues. Initial visual observation for spinal scoliosis and kyphosis should be routinely performed as these can be underlying biomechanical sources altering scapular mechanics and producing apparent scapular dyskinesis [21, 22].

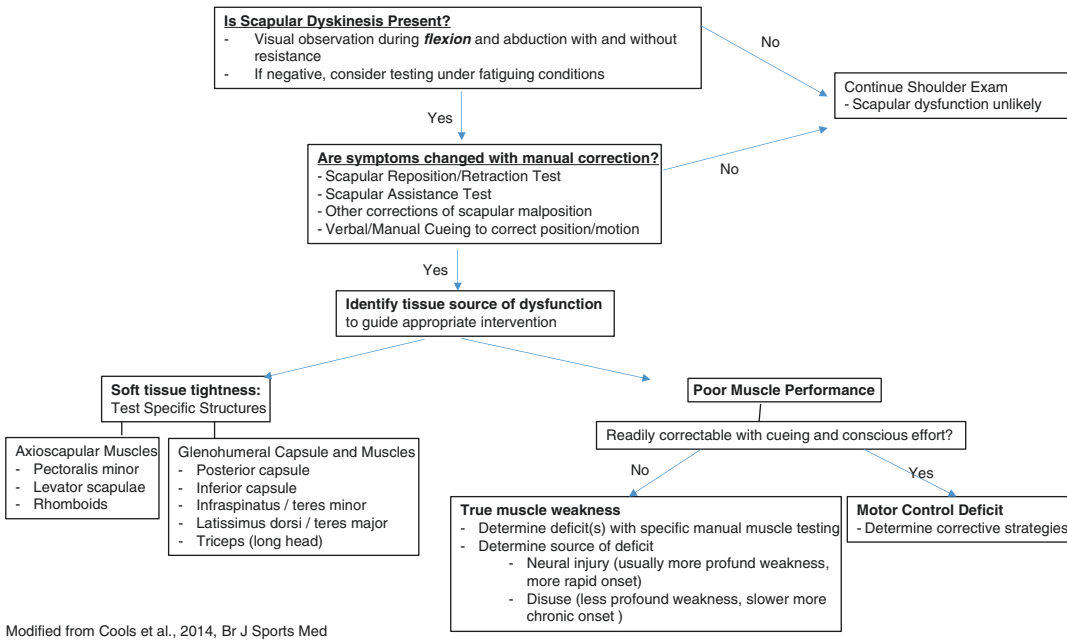

Fig. 4.1 Clinical scapular examination algorithm (Modified from Cools et al. 2014, Br J Sports Med)

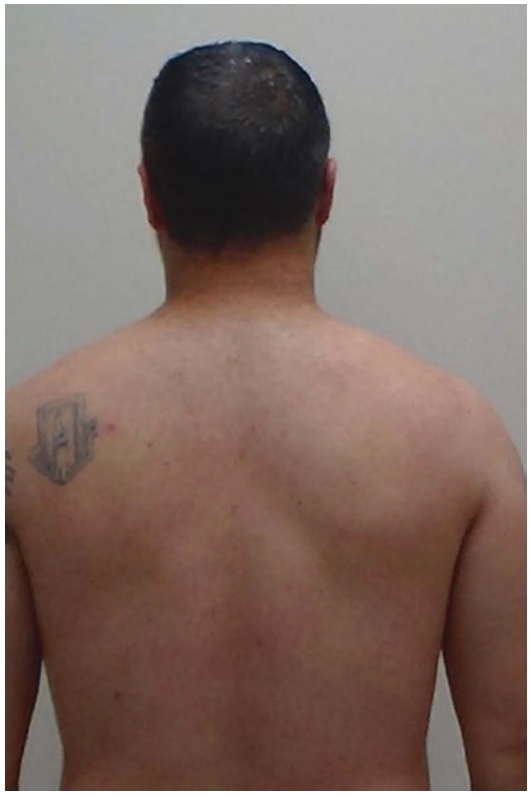

Fig. 4.2 Visual observation of the scapula from a posterior view

Many authors have suggested that forward head posture and increased thoracic kyphosis may contribute to scapular protraction and lead to adaptive shortening of postural muscles or muscular strength imbalances [23–26]. A protracted scapular position may be associated with a narrowed subacromial space [27, 28] and a flexed thoracic spine, and forward shoulder position alters scapular motion and results in diminished force output with elevation (Fig. 4.3) [23, 29].

Static assessment of scapular malposition has been demonstrated to be present in patient with and without shoulder pathology [6]. Warner used an enhanced visual observation using Moire' topography in patients with rotator cuff impingement, shoulder instability, and healthy cohort. Between 30 and 50% of 29 patients with shoulder pathology were found to have static scapular malposition when holding a 4.5 kg weight in hand with elbows flexed to 90° [6]. It is important to note that 3 of 22 or 14% of non-injured healthy cohort had asymmetric scapular position. This is an important point to remember in evaluating the scapula that asymmetric position does not necessarily indicate pathology.

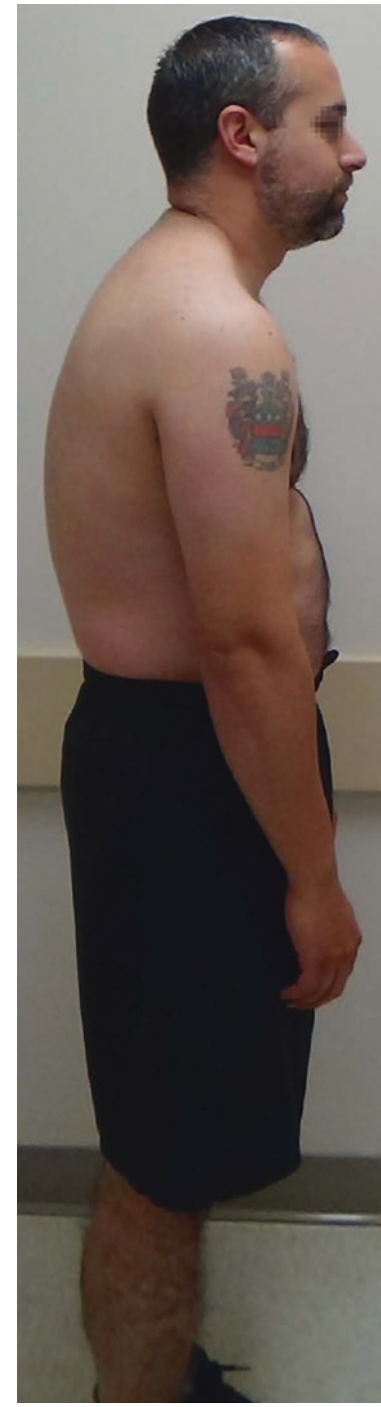

Fig. 4.3 Lateral view of upper quarter and trunk with noted forward head and rounded shoulder positioning

The lateral scapular slide test is a static measurement of the side-to-side difference of the distance from the inferior angle of the scapula to the adjacent spinous process [30]. The measures are performed with the arms in three different positions, arms at the side, hands on hips, and arms abducted to 90° in maximal internal rotation. A side-to-side difference of >1.5 cm is considered pathological. This test has demonstrated fair to moderate levels of reliability and is easily applied in a clinical setting [30, 31]. However, the validity of this test has been questioned because of the findings that both symptomatic and asymptomatic individuals will demonstrate asymmetry when measured in this manner [32, 33]. Additionally, it is possible to have symmetrical pathologic dyskinesis; therefore, validity is questionable when comparison is made only to the contralateral side. Furthermore, the static and two-dimensional nature of this test fails to fully assess the dynamic three-dimensional motion found to occur with scapular movement [10, 32, 34]. This inadequacy of measurement along with questionable validity of results requires the use of other methods of scapular assessment during clinical examination.

Visual assessment schemes for classifying scapular dyskinesis have been developed in an attempt to resolve the issues with linear or static measures [15, 34, 35]. These methods involve classifying scapular movement during shoulder motion into normal or abnormal categories. These measures are considered more functional in application and more inclusive with the ability to judge scapular movement in three-dimensional patterns. Kibler et al. [15] were the first to describe a visually based system for rating scapular dysfunction that defined three different types of motion abnormality and one normal type. Reliability values for this system were too low to support clinical use, and the test was subsequently refined in two studies using a simplified method of classification [35].

The scapular dyskinesis test is a visually based test for scapular dyskinesis that involves a subject performing weighted shoulder flexion and abduction movements, while visual observation of the scapula is performed [34]. This test consists of characterizing scapular dyskinesis as absent or present, and each side is rated separately. Dyskinesis is defined as the presence of either winging (prominence of any portion of the medial scapular border or inferior angle away from the thorax) or dysrhythmia (premature, or excessive, or stuttering motion during elevation and lowering) (Fig. 4.4). Interrater reliability of this test, after brief standardized online training https://www.arcadia.edu/college-health-sciences/departments-faculty/physical-therapy/shoulder-research-center, has been shown to be better than other previously described visual classification systems. Concurrent validity was assessed in a large group of overhead athletes, and it was shown that those judged as demonstrating abnormal motion using this system also demonstrated decreased scapular upward rotation, less clavicular elevation, and less clavicular retraction when measured with three-dimensional motion tracking [10]. Abnormalities were far more prevalent during shoulder flexion compared with frontal plane abduction. These results support the assertion that shoulders visually judged as having dyskinesis using this system demonstrate distinct alterations in three-dimensional scapular motion, particularly during flexion. However, while visually observed dyskinesis resulted in an altered three-dimensional motion, subjects with dyskinesis were no more likely to report symptoms during sports [10].

Uhl et al. [35] used essentially the same criteria (winging or dysrhythmia) to classify any subject that demonstrated an abnormality in scapular motion into the "yes" classification, and normal movement was classified as "no." They studied both symptomatic patients with various soft tissue pathologies as well as an asymptomatic group. The "yes/no" test was found to have superior interrater reliability and demonstrated better specificity and sensitivity values when using asymmetry found with three-dimensional testing as a gold standard [35]. An important finding that was consistent with previous research [10] also demonstrated a higher frequency of dyskinesis during shoulder flexion in patients (54%) compared with asymptomatic subjects (14%), whereas no differences between groups were detected during scapular plane elevation.

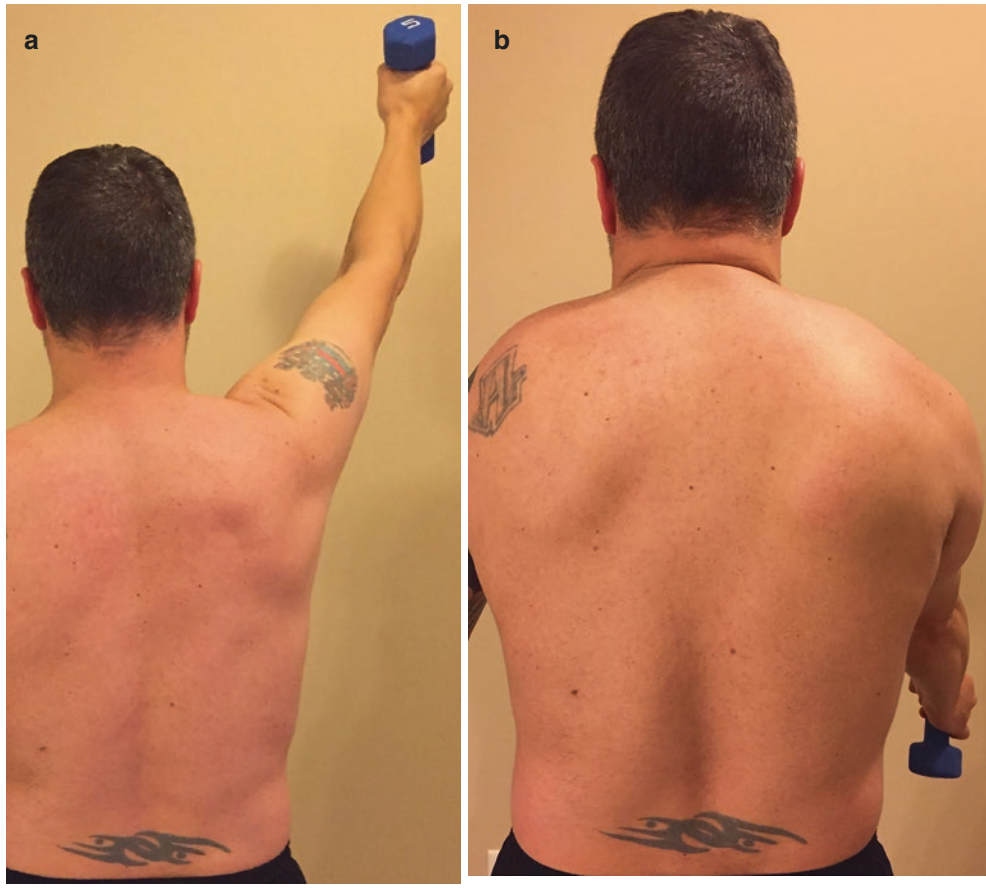

Fig. 4.4 Posterior view of scapular dyskinesis test during elevation and lowering with prominence of scapular medial border winging on the right side

Manual Correction

Because scapular dyskinesis is a common finding in asymptomatic individuals, a basic problem in evaluation is deciding if the presence of scapular dyskinesis is an important abnormality perpetuating symptoms. The possibility exists that alterations of scapular motion could be compensatory strategies to avoid stress on pain-sensitive tissue. Symptom alteration tests have been developed as a way to infer scapular malposition in driving symptoms by manually redirecting scapular movement during provocation testing. If altering scapular position causes an immediate decrease in symptoms, this provides direct evidence that scapular dyskinesis is a contributing factor to shoulder symptoms in that specific patient. The two main symptom alteration tests are the scapular assistance test [16, 30] and the scapular reposition or retraction test [17, 36].

The scapular assistance test involves manually assisting scapular upward rotation during shoulder elevation and determining this effect on pain (Fig. 4.5) [37]. This test was later modified by Rabin incorporating scapular posterior tilting as well (Fig. 4.6) [16]. A positive test is when pain with elevation is either decreased or abolished during the assisted maneuver. This test has demonstrated acceptable levels of reliability [16], increased subacromial space [38], increased upward rotation, and posterior tilt of the scapula [38].

The scapular retraction test involves manually positioning and stabilizing the entire medial bor-

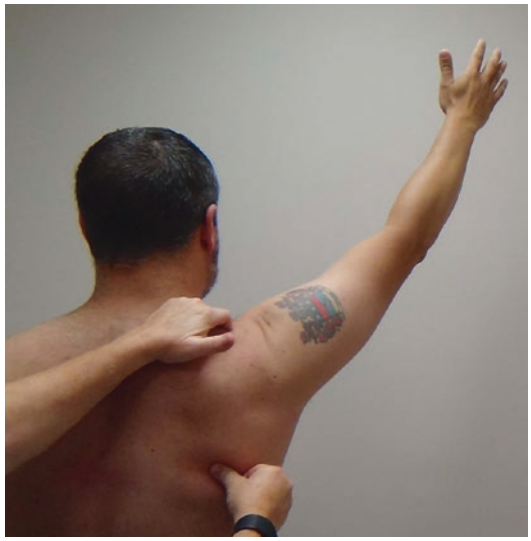

Fig. 4.5 Scapular assistance test applying anterior and laterally directed force on the inferior scapular angle with the examiner's thumb

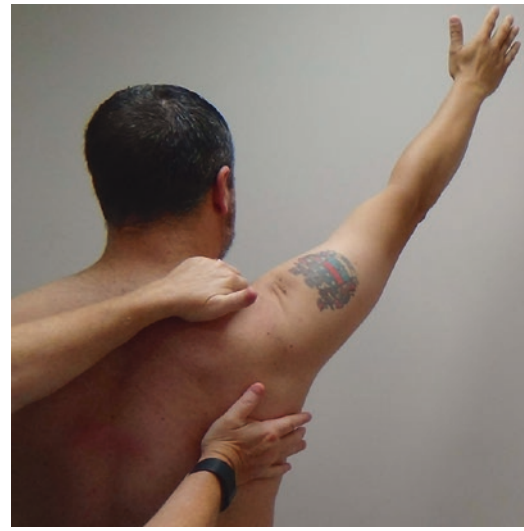

Fig. 4.6 The modification of the Scapular assistance test in which the entire hand is used to apply the anterior and laterally directed force to the inferior scapular angle

der of the scapula in a retracted position on the thorax [37]. This test was developed to help identify patients in which strength loss in shoulder elevation may be due to a loss of proximal stability of the scapula. The test is performed by asking the patient to retract both shoulder blades, and the examiner stabilizes the medial border of the scapula with their forearm (Fig. 4.7). The test is considered positive when the patient demonstrates a reduction of pain or an increase in shoulder elevation strength when the scapula is stabilized during isometric arm elevation in the scapular plane at 90° [19, 37]. Kibler et al. [36] studied this test in symptomatic and asymptomatic subjects. Their findings demonstrated that there was no change in pain levels, and all subjects demonstrated improved strength output, regardless of symptoms.

The scapular reposition test is a modification of the scapular retraction test that involves emphasizing scapular posterior tilting and external rotation but avoiding full scapular retraction (Fig. 4.8) [17]. This modification was based upon previous investigations that have found a decrease in shoulder elevation strength with maximal active scapular retraction [29]. This test has demonstrated acceptable reliability and when performed on a large group of overhead athletes; roughly half of those with pain (46/98) during impingement testing had reduced pain, and 26% had a substantial increase in isometric elevation strength. Therefore, this test may be helpful at identifying a subset of patients with shoulder pathology that may benefit from interventions designed to improve scapular muscle function.

Surrounding Tissue Evaluation

Once an examiner determines that scapular dyskinesis is present and determines it is a contributing factor to the overall shoulder pathology, examination of the surrounding tissue should be performed to identify those factors that may be responsible for causing the altered scapular motion. Many structures have been implicated as possible contributors to the development of scapular dyskinesis. These include deficits in strength or motor control of scapular stabilizing muscles [17, 18, 30, 39], postural abnormalities [23, 26, 40], and impaired flexibility [13, 41]. Therefore, a comprehensive examination of all of these components is necessary (Fig. 4.1).

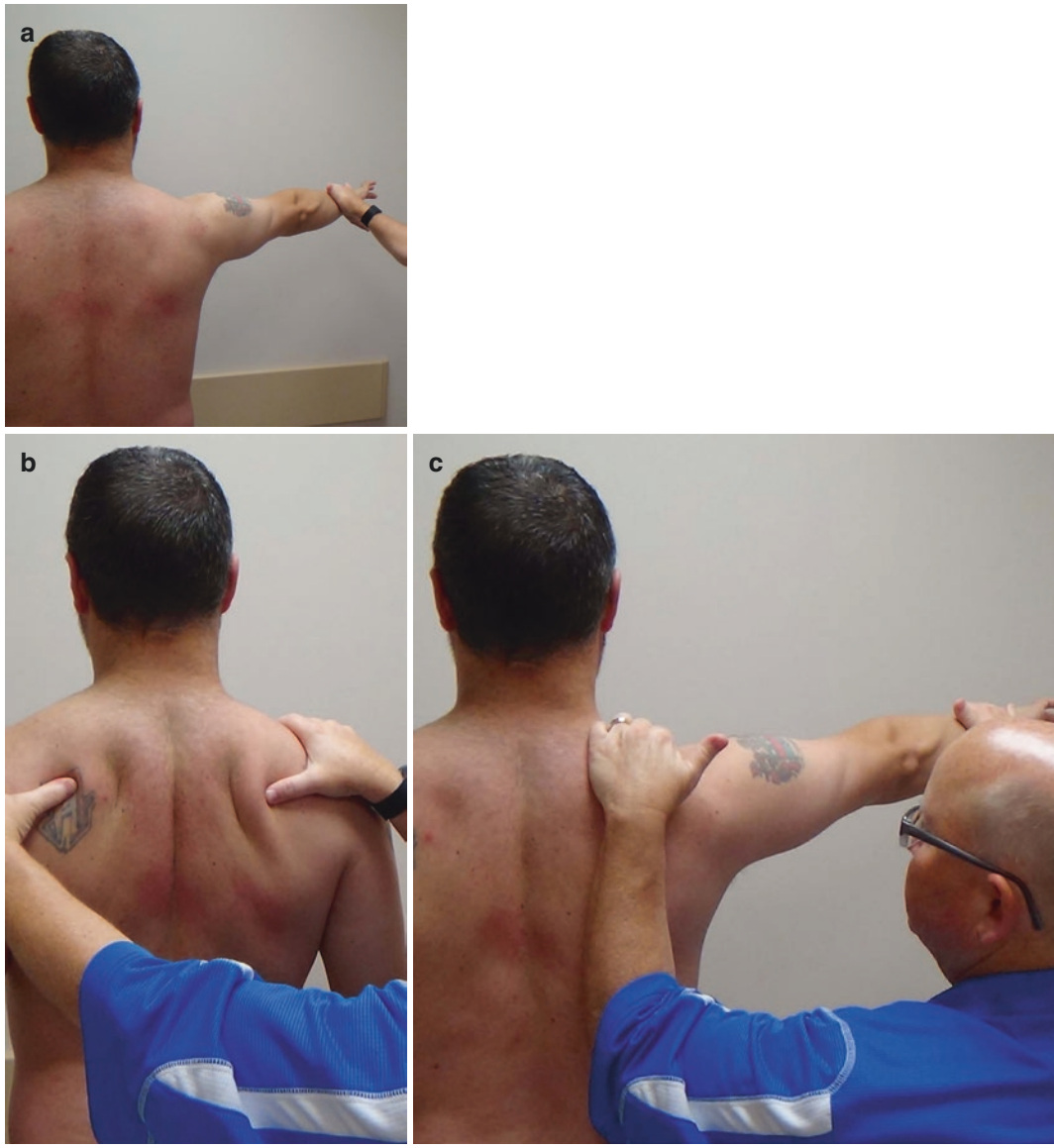

Fig. 4.7 The scapular retraction test is divided into three components: (**a**) the clinician tests arm strength without the scapula stabilized or retracted, (**b**) the patient is asked to actively retract the scapula, and (**c**) the clinician stabilizes the medial border of the scapula with one forearm, while the other arm applies a downward force on the abducted arm

Muscle strength of key scapular stabilizers can be assessed using standard positions and procedures described by Kendall et al. [42]. The key muscles to test are the axioscapular muscles [43]. Underlying neurological injury to the long thoracic, spinal accessory, or dorsal scapular nerves should be investigated as potential causes of scapular dyskinesis.

The serratus anterior innervated by the long thoracic nerve has a significant contribution to scapular upward rotation, internal rotation, and clavicular protraction. Assessing the ability to protract the scapula around thorax or hold against a retraction load is necessary to confirm the serratus anterior is functioning correctly. The ability to elevate the arm overhead, specifically in the

sagittal plane, and protract the scapula around the thorax while resisting a retraction force will indicate that serratus anterior is functioning correctly (Fig. 4.9). The presence of "winging," inability to keep the inferior medial border of the scapula stabilized to the thorax, during sagittal plane elevation would suggest the serratus anterior is not functioning well. This could be attributable to poor motor control or also to true muscle weakness associated with disuse or nerve injury. This is an important distinction that may influence intervention strategies. If the winging is due to poor motor control, the patient should be able to quickly correct the problem with appropriate cueing and may also perform normally on a manual muscle test. However, weakness and winging that are not easily corrected and persist during isolated manual muscle testing may indicate underlying neurological deficit of the long thoracic nerve pathology [44].

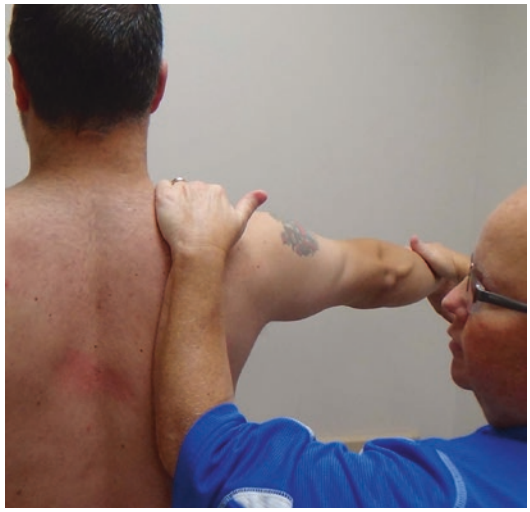

Fig. 4.8 The scapular reposition test is similar to the scapular retraction test except the patient is not asked to actively retract the scapula, so there are only two components: (**a**) the clinician stabilizes the medial border of the scapula with one forearm, (**b**) while the other arm applies a downward force on the abducted arm

Along with the serratus anterior, the upper and lower trapezius functions in a force couple to upwardly rotate the scapula. In particular the trapezius musculature is key stabilizer of the scapula when the arm is in frontal plane abduction [19, 39, 45–47]. A key concept in testing these muscles is that even though resistance is applied through the arm, weakness is identified by early "breaking" of the scapula rather than the arm. In patients with rotator cuff or deltoid

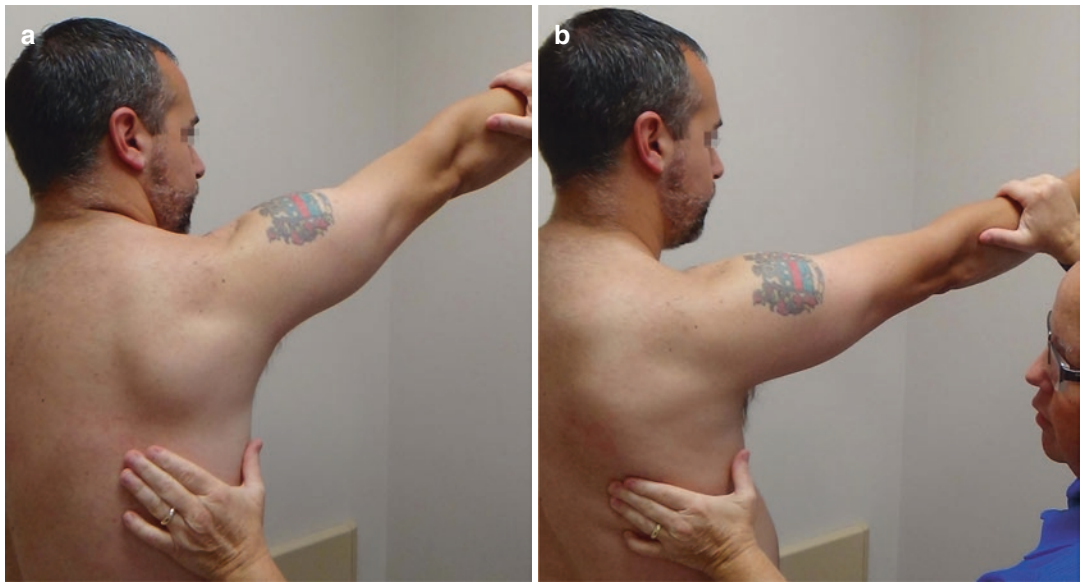

Fig. 4.9 Serratus anterior manual muscle test evaluating the ability of the scapula to stabilize along the thoracic wall against a downward and posteriorly directed force (**a**). Presence of winging or posterior scapular displacement away from the thorax indicates serratus anterior weakness (**b**)

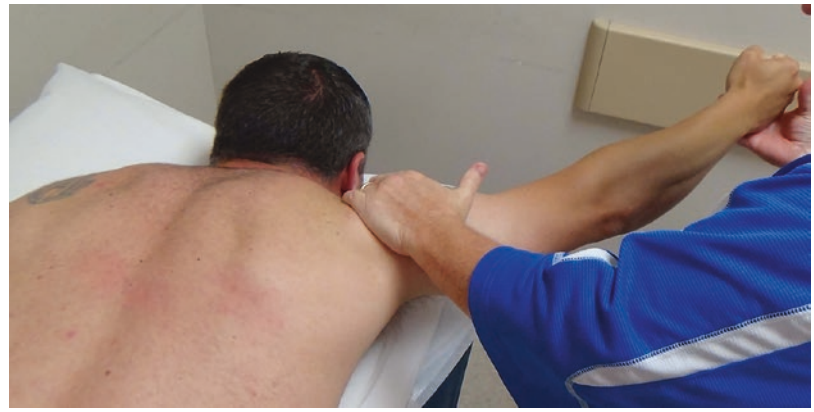

Fig. 4.10 Lower trapezius manual muscle test performed in prone with force applied at the posterior lateral aspect of the acromion, directing force anteriorly and toward scapular musculature over rotator cuff and deltoid muscles

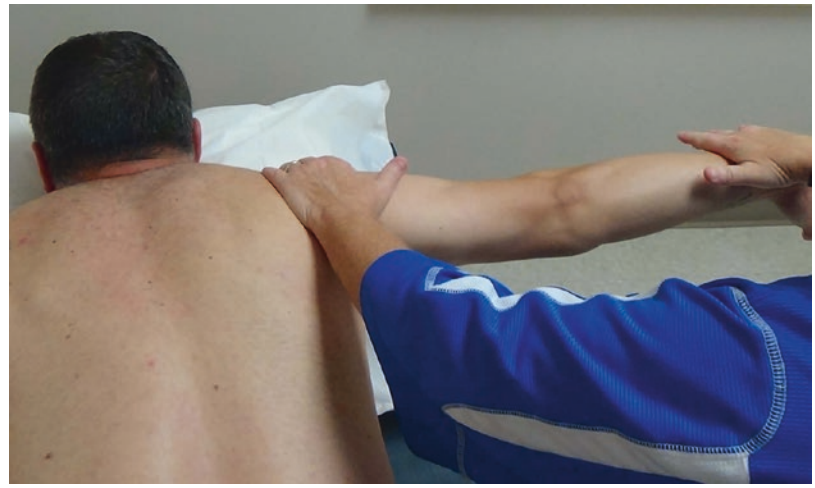

Fig. 4.11 Middle trapezius manual muscle test performed in prone with force applied at the posterior lateral aspect of the acromion, directing the force anteriorly in order to bias the scapular musculature over the rotator cuff and deltoid muscles. If a long lever arm is used, pay close attention to which gives way first, the scapular retraction or the horizontal abduction of the arm

weakness, the arm may need to be supported and resistance applied directly to the scapula to accurately determine scapular muscle weakness. Resistance applied in specific directions onto the scapula should provide a more specific test to evaluate scapular muscle strength. Scapular elevation or shrugging biases the upper trapezius muscles but rarely demonstrates weakness in a muscle test. Assessing lower trapezius muscle strength should be performed in prone with the patient elevating their arm away from the floor with arm abducted 135° (Fig. 4.10). Applying a resistive force in line with the lower trapezius muscle to the posterior lateral acromion to force the scapula toward the ground biases the activation of the lower trapezius muscles [48]. Assessment of both the rhomboid, a dorsal scapular-innervated muscle, and the middle trapezius can be performed by having the patient lie prone and retract the scapula while applying a downward force on the posterior lateral acromion toward the floor (Fig. 4.11). The ability to discriminate between these two muscles using multiple test positions has not been identified to date [48, 49]. It is critical to get adequate scapular retraction when placing patient into retraction in order to engage the scapular retractors when testing.

Assessment of shoulder muscle flexibility and all shoulder joint mobility is critical to completely evaluate potential causes of scapular dyskinesis. Adaptive shortening of the pectoralis minor muscle has been identified as a contributor to abnormal scapular kinematics and implicated as a factor that may contribute to shoulder impingement syndrome [41, 50]. Sahrmann [50]

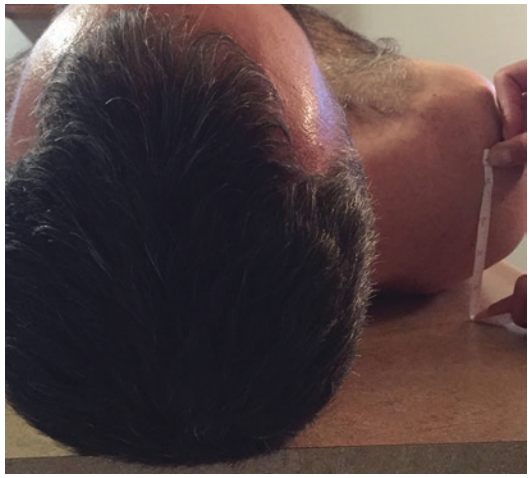

Fig. 4.12 Measurement of pectoralis minor length as suggested by Sahrmann from supine position to anterior aspect of the acromion

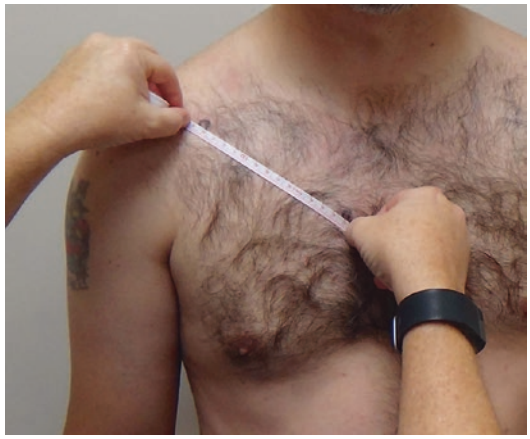

Fig. 4.13 Pectoralis minor length measured from the fourth rib to the coracoid process

has described an assessment method for pectoralis minor length that involves taking a linear measurement with the patient supine from the treatment table to the posterior aspect of the acromion, with any measurement >2.54 cm suggesting tightness (Fig. 4.12).

Although highly reliable, some have questioned the validity of this method as it failed to discriminate those with shoulder pain [51]. Another assessment method that has been described involves using a tape measure or caliper to record the linear distance between the anatomic origin and insertion of the pectoralis minor muscle (Fig. 4.13) [52]. This measurement was found to have satisfactory intrarater reliability (intraclass correlation coefficient = 0.82–0.87) and good concurrent validity. This linear measure requires careful palpation and has not demonstrated differences in patient with and without symptoms of shoulder impingement [53].

Another assessment of forward shoulder posture has been described with the patient standing upright measuring the distance from the anterior acromion to the wall (Fig. 4.14) [54]. This technique has been used to identify individuals with forward shoulder posture due to anterior structure tightness. This measurement technique has not identified a specific muscular tightness but has been responsive to therapeutic interventions addressing forward posture [26, 55].

Traditional range of motion measures of external rotation and internal rotation at 90° abduction with the scapula stabilized to determine the mobility of the glenohumeral joint is an important assessment in evaluating the causes of scapular dyskinesis. Limitations of the glenohumeral joint can be a source of abnormal scapular motion and must be addressed in both the assessment and treatment of shoulder pathologies.

Posterior shoulder tightness (capsular or rotator cuff) has been associated with excessive protraction of the scapula [56] and may contribute to scapular dyskinesis [13]. Two common methods of assessing posterior shoulder tightness are internal rotation at a 90° abduction (Fig. 4.15) [57, 58] and spinal level reached with reaching behind the back [59, 60]. These two methods have demonstrated acceptable levels of reliability for clinical use. Gerber et al. [61] showed that different parts of the posterior capsule restrict internal rotation with the arm by the side versus 90°. Therefore, authors have recommended that clinicians use both assessment methods to allow for a more comprehensive assessment of posterior shoulder tightness [62]. Measurements of shoulder internal rotation are affected by humeral and glenoid version and therefore make it difficult to distinguish between soft tissue tightness and

bony alterations causing diminished internal rotation. To overcome this problem, Laudner et al. [63] have described measuring horizontal adduction with the arm at 90° elevation and the scapular blocked with satisfactory reliability (Fig. 4.16).

Ultimately, the goal of any examination is to determine a diagnosis and develop appropriate intervention to address impairments identified or dysfunctions identified during the examination. The role of the scapular examination is to identify what if any scapular impairments are potentially contributing to a patient's shoulder pain. The three components of the shoulder examination will assist to identify the presence of tissue inflexibility or poor motor function impairments. In order to institute an intervention, detailed in Chaps. 15 and 16, the level of tissue irritability when a patient presents needs to be included in the examination process in order to apply the correct intervention level. A three-level categorization of tissue irritability has been described to consider when applying appropriate intervention [64]. In the presence of a highly state of irritability (Stage 1), the patient with poor motor function presents in a high level of pain, disability, and significant guarding of active shoulder motion. The intervention approach would need to be a minimal tissue loading and pain reduction inter-

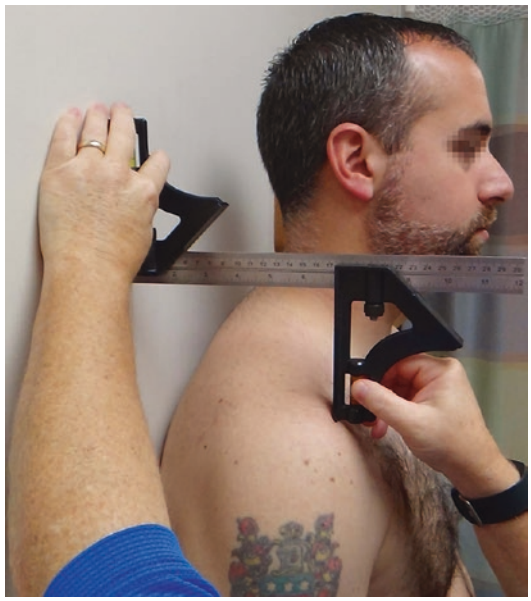

Fig. 4.14 Forward shoulder posture measured in standing using a double square method

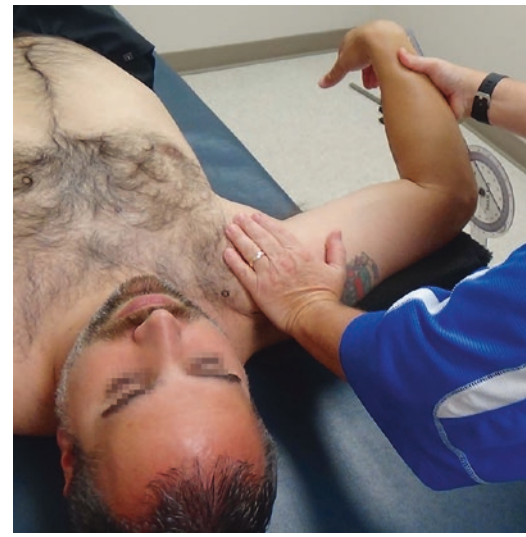

Fig. 4.15 Measuring internal rotation of the glenohumeral joint in the plane of the scapula with scapula stabilized

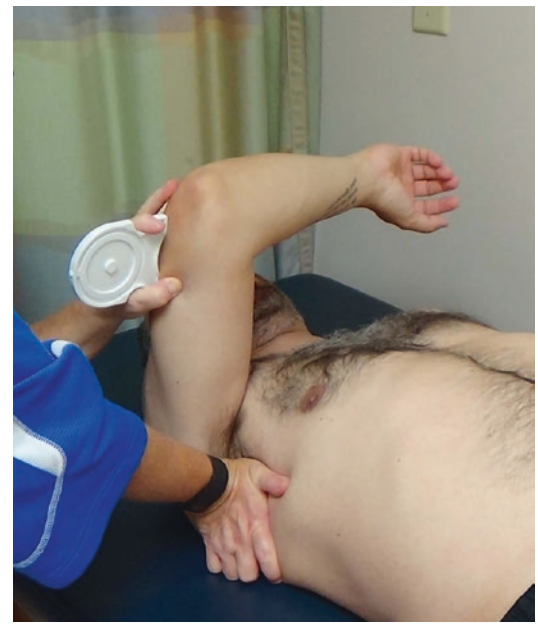

Fig. 4.16 Posterior shoulder tightness measured with horizontal adduction while stabilizing the scapula. Zero position of the humerus would be perpendicular to the plinth; motion into more horizontal adduction would be represented as positive value, and less would be recorded as a negative value

vention due to the high level of tissue irritability. However, in a patient with similar impairments of decreased motor control of the scapular musculature but is in a low irritability state of low pain and disability, a more focused and direct intervention addressing the impaired scapular muscle function can be applied as the tissue irritability level is low (Stage 3). The skilled clinician can integrate both the clinical scapular examination with the appropriate staged tissue irritability by performing a comprehensive examination allowing them to determine appropriate scapular impairments and develop a stage-appropriate and effective intervention.

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Part II

The Scapula and Shoulder Pathology

The Scapula and Impingement/ Rotator Cuff Disease and Treatment

5

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Background

Scapular dyskinesis is present in most shoulder injuries (68–100%) [1]. Major progress has occurred in our understanding of scapular dyskinesis. Scapular dyskinesis is now identified around the world and the implications are broad. Our understanding of the scapula now extends far beyond our rudimentary basis of "scapular winging" and neurologic conditions. We can now begin to explore the impact of the scapula on other conditions and explore a more cause and effect relationship. The scapula establishes a platform for effective rotator cuff function and normal shoulder motion. Alterations in scapular motion can be associated with multiple pathologic conditions including rotator cuff weakness and rotator cuff pathology [2]. Clinical data and experimental model systems, including cadaveric and animal models, have also been developed to assess the role of the scapula in impingement and rotator cuff disease. Currently, there is limited evidence to guide our treatment or prevention of rotator cuff injuries that may be secondary to scapular dyskinesis. A better understanding of the role of the scapula in rotator cuff pathology would help optimize clinical management.

This chapter will highlight how the role of the scapula in impingement and rotator cuff disease is currently evaluated through clinical studies and through experimental model systems. Using these studies as a framework, the role of the scapula in the development of these conditions will be highlighted. Lastly, the importance of the scapula in the treatment of rotator cuff pathology will be discussed.

Evaluating the Role of the Scapula in Impingement/Rotator Cuff Disease

Early recognition of scapular dyskinesis was based on clinical observations. Scapular dyskinesis is now reliably characterized with a validated observational test [3]. The scapular dyskinesis test is a dynamic observational test use to reliably diagnose scapular dyskinesis. Clinical observations and reliable diagnostic criteria for scapular dyskinesis are complemented with corrective physical examination maneuvers that lead to symptom alteration and rotator cuff strength

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improvement. The scapular assistance test (SAT) and scapular retraction test (SRT) are corrective physical examination maneuvers that can alter the injury symptoms [1, 4, 5]. These corrective physical examination maneuvers attest the role of scapular dyskinesis and rotator cuff involvement because correction of abnormal scapular positioning can lead to symptom improvement and rotator cuff strength improvement.

Clinical observations and corrective physical examination maneuvers have helped to better identify scapular dyskinesis and its association with rotator cuff pathology, and now epidemiological studies also highlight this relationship. Specific body types and postures have now been linked to scapular dyskinesis and rotator cuff tears. Faulty posture, especially thoracic kyphosis, has been shown to be associated with scapular dyskinesis [6]. An ultrasound study of 379 participants from a single mountain village showed that postural abnormalities were independent predictors of rotator cuff tears with the highest prevalence of rotator cuff tears among individuals with thoracic kyphosis and lumbar lordosis [7]. Postural abnormalities were shown to alter scapular motion and are thought to cause mechanical abrasion and wear on the rotator cuff.

Scapular dyskinesis with protraction, posterior tilt, and upward rotation may be the cause of rotator cuff pathology by causing mechanical abrasion and wear with decreased subacromial space and decreased rotator cuff clearance under the coracoacromial arch [6]. Conversely, scapular dyskinesis could be a negative decompensation for rotator cuff injury and may lead to further shoulder dysfunction.

Unfortunately, clinical studies are unable to address the underlying causes of impingement and rotator cuff pathology. Experimental model systems such as cadaveric and animal models have played a critical role in the characterization and understanding of rotator cuff disease and treatment along with examining the contribution of static and dynamic variants of the scapula. These model systems have advantages and limitations that should be considered when evaluating and interpreting findings.

Cadaveric Studies

Human cadaveric studies have several advantages including anatomic assessment and controlled biomechanical evaluation of the scapula. Static anatomic variants such as acromial type and glenoid orientation and their association with rotator cuff pathologies have been studied [8–11]. Biomechanical testing through control of scapular orientation and alterations in applied forces and/or simulated tears have also been investigated [12, 13]. Unfortunately, cadaveric studies are limited in that they cannot address the underlying causes of injury and are unable to evaluate the injury process over time. In addition, most shoulder biomechanics research focuses on actuation of the glenohumeral joint and doesn't include scapulothoracic motion or attempt isolated alterations in scapular orientation without consideration of the dynamic and complex 3D motions observed in vivo [14, 15].

Animal Models

The rat shoulder model has been previously identified as anatomically and functionally similar to the human shoulder [16]. Specifically, both the rat and human have similar bony architecture and soft tissue anatomy as the human, including the presence of a coracoacromial arch, under which the supraspinatus tendon passes during forward locomotion (Fig. 5.1). Reuther et al. have recently extended the rat shoulder model to include induction of scapular dyskinesis through acute nerve injury of the spinal accessory and long thoracic nerve [17, 18]. The use of animal models for shoulder research has several advantages over clinical studies including the ability to repeat and control study parameters and the ability to examine longitudinal changes in behavior, function, and tendon properties and elucidate the mechanism by which these changes occur. Unfortunately, there are several limitations that should be recognized in the rat model including the fact that the use of a quadruped animal does not exactly replicate the human condition and that induction of scapular dyskinesis through acute nerve injury represents only a small

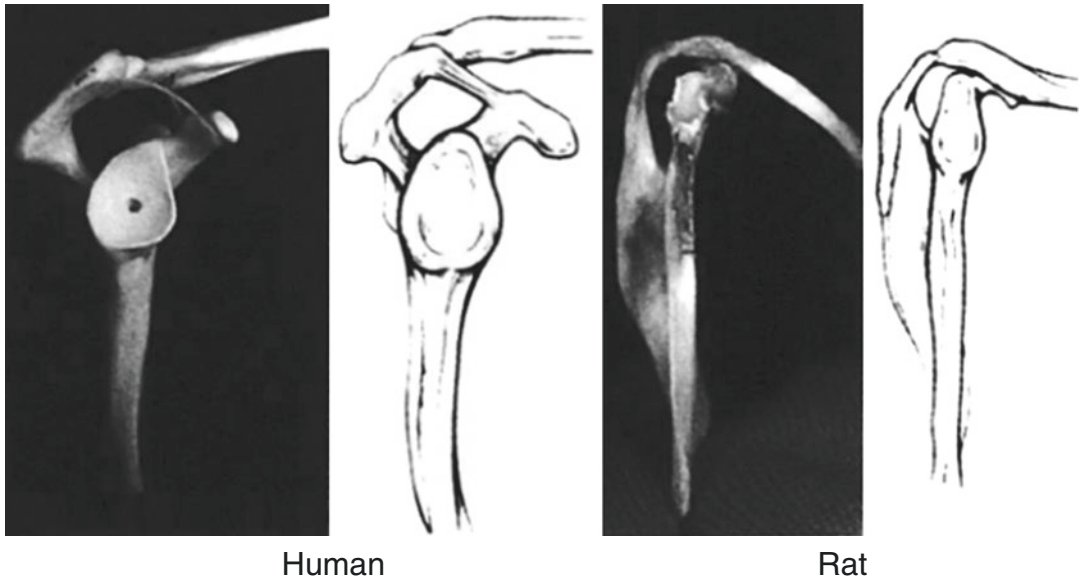

Fig. 5.1 Comparison of human shoulder anatomy to rat shoulder anatomy. This view demonstrates the presence of a coracoacromial arch in both the human and rat, under which the supraspinatus passes [16]

percentage of patients clinically. However, this model allows researchers to assess the cause and effect relationship between abnormal scapular motion and rotator cuff pathology in an anatomically similar model as the human and in a controlled and repeatable manner.

Development of Rotator Cuff Disease/Impingement

Rotator cuff tears are very common with up to 20% of the general population reported to have a rotator cuff tear and greater than 50% prevalence in the sixth and seventh decades [19]. Scapular dyskinesis has been reported in a high proportion of patients with rotator cuff tears [20]. Scapular dyskinesis was present in 28% of patients with symptomatic, atraumatic rotator cuff tears in this prospective multicenter cohort study. Scapular dyskinesis was associated with worse pretreatment shoulder function scores.

In addition to the associations observed clinically, experimental model systems have identified both static and dynamic variants of the scapula that may contribute to the development of rotator cuff disease and impingement.

Static Anatomic Variants

Static anatomic variants of the scapula, including anatomy of the acromion and glenoid, and their association with rotator cuff pathology have long been studied in cadaveric experimental models. Bigliani et al. developed a classification system of acromial shape that included "flat" (Type 1), "curved" (Type 2), and "hooked" (Type 3) (Fig. 5.2) [21]. A correlation was observed between Type 3 acromion and the presence of rotator cuff tears in cadaver shoulders. In support of this work, Flatow et al. performed another cadaveric study which assessed excursion of the rotator cuff under the acromion through biomechanical testing, stereophotogrammetry, and radiographs [22]. The authors found an increase in subacromial contact with Type 3 acromions. Other features of the acromion associated with rotator cuff disease include anterior tilt of the acromion [23], lateral extension of the acromion [24], lateral tilt of the acromion [25], and the presence of an os acromiale [26]. Related to the acromion morphology is the finding that a narrow supraspinatus outlet is associated with rotator cuff tears [27].

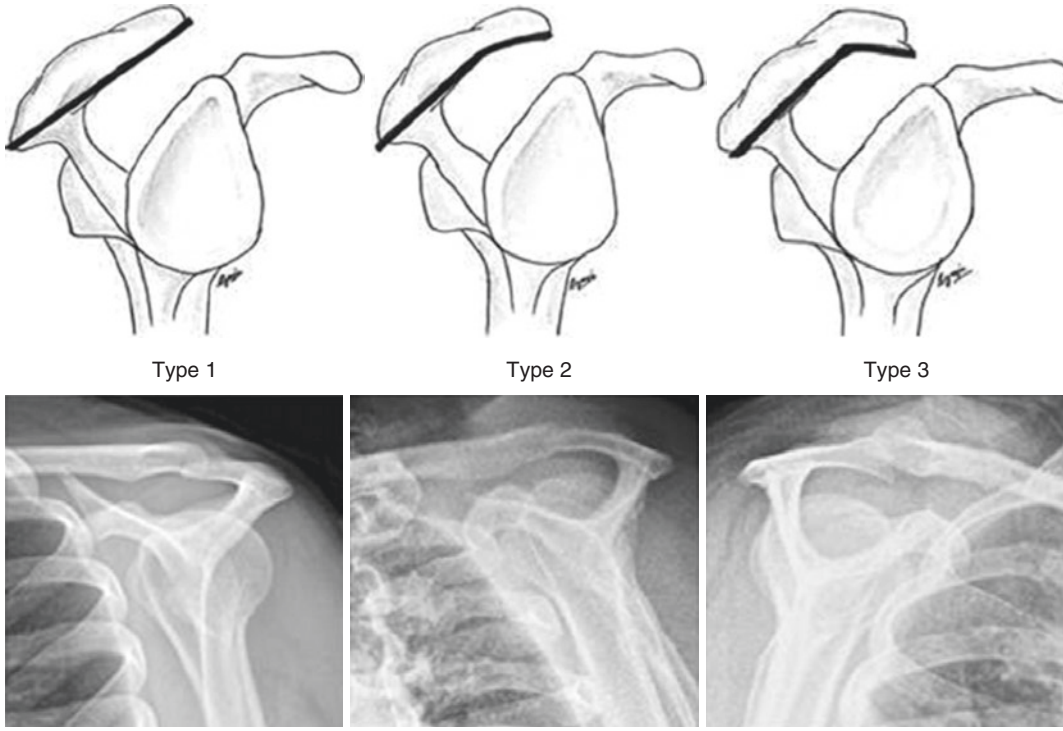

Fig. 5.2 Bigliani's acromion classification of the undersurface of the acromion with corresponding radiographs [29]

Cadaveric studies have also demonstrated that glenoid orientation also plays an important role in rotator cuff pathology. Glenoid inclination, a measure of the angle of the glenoid in plane of the scapula, was examined in cadaveric shoulders with and without rotator cuff tears. A greater glenoid inclination angle, or a more upward-facing glenoid, was observed in shoulders with cuff tears [28]. Another cadaveric study observed an increased risk for superior humeral head migration with greater glenoid inclination, indicating that a more upward-facing glenoid could contribute to cuff pathology and impingement [11, 28]. In contrast, Kandemir et al. found no difference in glenoid version or inclination in cadavers with intact versus torn rotator cuffs [9]. Finally, there is a relationship between the lateral extension of the acromion and the glenoid inclination angle that produces the "critical shoulder angle" such that larger angles are associated with rotator cuff tears, and smaller angles are associated with glenohumeral osteoarthritis [10].

Scapular Kinematics and the Dynamic Influence

Biomechanical evaluation of cadaver shoulders has provided insight into the role of the scapula in glenohumeral joint mechanics and pathology. Impingement (including internal and external or subacromial) has been carefully investigated in cadaveric experimental models. Karduna et al. altered scapular orientation (including posterior tilt, upward rotation, and external rotation) and evaluated clearance in the subacromial space [12]. Results demonstrated a decrease in subacromial clearance with increased upward rotation of the scapula. This is contrary to what was expected given clinical data that has observed a decrease in upward rotation with impingement and may suggest a compensatory modification in joint kinematics [30].

The role of scapular orientation in internal impingement, classified as contact between the

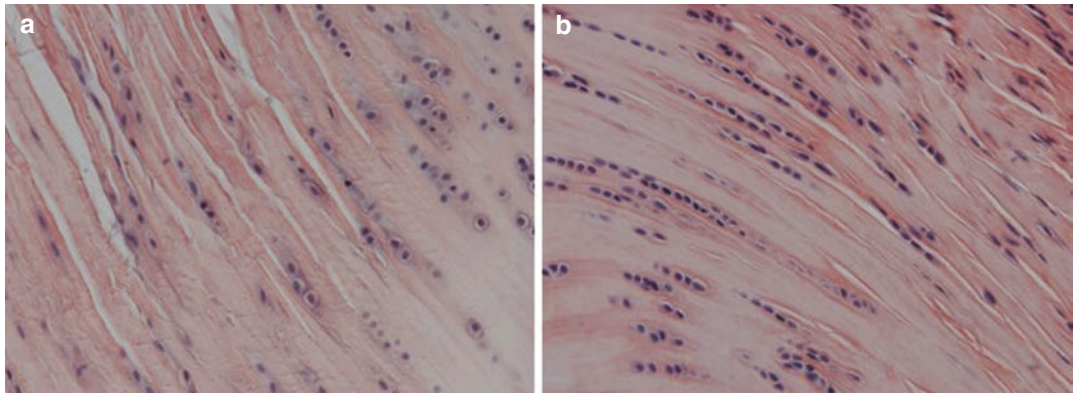

Fig. 5.3 Representative histologic image of the rat supraspinatus tendon demonstrated increased cell density in the presence of scapular dyskinesis (**b**) compared to control (**a**) [17, 18]

undersurfaces of the posterior cuff with the humerus, superior labrum, and glenoid rim, has also been studied through biomechanical evaluation. Mihata et al. modified scapular orientation and evaluated glenohumeral joint positioning and contact pressures and found that decreased upward rotation and increased internal rotation increased the glenohumeral contact pressure and impingement area in cadaver shoulders [13]. This is contrary to the subacromial impingement studies from Karduna et al. and suggests the role of scapular orientation in both forms of impingement is still controversial.

Despite our improved understanding of the role of the scapula in rotator cuff disease and impingement through clinical observations and cadaveric evaluation, evidence is still mixed regarding the cause and effect relationship between scapular dyskinesis and rotator cuff pathology. Recently, a new scapular dyskinesis rat model was developed to better understand this relationship from a basic science perspective [17, 18]. The rat model allows for controlled and repeatable induction of scapular dyskinesis and the opportunity for qualitative and quantitative evaluation of subsequent joint function (including spatial, temporal, and kinetic parameters and passive joint mechanics) and supraspinatus tendon properties (including mechanics, structure, and organization) in response to the prescribed alteration in scapular motion.

The objective of the rat model study was to evaluate the effect of scapular dyskinesis on glenohumeral joint function and tendon properties. In the scapular dyskinesis group, surgical transection of the accessory and long thoracic nerve was performed, and entire medial border prominence of the scapula was observed during ambulation, indicative of abnormal positioning of the scapula and acromion in these animals. The scapular dyskinesis group also demonstrated altered joint function in the form of increased propulsion force, decreased vertical force, and increased internal rotation range of motion. Propulsion force is required for forward locomotion in the rat, and an increase in this parameter may indicate greater stress being placed on the glenohumeral joint. A decrease in vertical force suggested a functional deficit and possible pain. The increased internal rotation range of motion suggested a loosening in the posterior structures of the shoulder do to the unstable scapula. The scapular dyskinesis group also had altered tendon properties (including mechanical, histological, and structural) (Fig. 5.3). There are two possible mechanical mechanisms for these alterations: (1) altered acromial position and reduced subacromial clearance led to tendon mechanical abrasion and wear and (2) increased demand was placed on the rotator cuff in the scapular dyskinesis group in an attempt to restore dynamic stability to the joint. This study was the first to directly

identify scapular dyskinesis as a causative mechanism of altered glenohumeral function and rotator cuff tendon properties.

A follow-up study by Reuther et al. examined the impact of scapular dyskinesis in an overuse population [17, 18]. As expected, the combination of overuse and scapular dyskinesis had a greater effect on tendon properties than scapular dyskinesis alone. This study suggested that the risk for shoulder injury in patients with scapular dyskinesis might be higher in individuals who frequently perform overhead activities.

While there is an abundance of biomechanical and animal model research evaluating the relationship between scapular dyskinesis and rotator cuff disease, clinical evidence is also robust. Warner et al., using Moire topography, demonstrated abnormal scapular positions at rest in patients diagnosed with impingement syndrome [31]. Symptomatic patients with rotator cuff disease have abnormal scapular kinematics, and muscle activity ratios comparted to asymptomatic patients with disease and those with normal anatomy [32–36]. Shoulder pain related to impingement in swimmers is associated with abnormal recruitment patterns of the serratus and lower trapezius [37], and the pain associated with rotator cuff tears is linearly related to scapular dyskinesis [38].

Scapular rehabilitation may be helpful in treating rotator cuff-related shoulder pain and fatigue [39]. Two recent systematic reviews of the literature concluded the scapula-focused approach was significantly better at improving patient disability early [40], and scapular strength [41], but the effect did not seem as impressive for pain or range of motion. The limited number of trials in the literature (seven) made definitive conclusions difficult.

In summary, clinical evidence and cadaveric and experimental model systems have demonstrated both static and dynamic variants of the scapula can contribute to rotator cuff pathology. Abnormal position and motion of the scapula can increase stresses on the rotator cuff and lead to impingement and damage. Careful considerations should be given to these findings when managing these conditions and patient expectations clinically.

Importance of the Scapula in Treatment of Rotator Cuff Disease

Scapular dyskinesis has been identified as a non-surgically modifiable factor to treat patients with rotator cuff tears [20]. Physical therapy has been studied as a viable treatment option in patients with symptomatic, atraumatic full-thickness rotator cuff tears in a prospective multicenter study [42]. Physical therapy with scapular exercises was effective in treating 75% of the patient population for up to 2 years.

Clinical evidence and ex vivo and in vivo experimental model systems have demonstrated a clear association between scapular static and dynamic variants and rotator cuff disease and impingement. As a result, these variants should be taken into consideration when treating patients with these pathologies. Early investigations in this area have begun to examine the role of the scapula in rotator cuff healing following surgical repair. Reuther et al. evaluated how scapular dyskinesis affected tendon healing following repair in a rat model [43]. Results demonstrated deficits in tendon mechanical properties in repaired tendons in the scapular dyskinesis group compared to repaired tendons in rats with normal scapular motion. This was the first study to demonstrate that scapular dyskinesis may diminish rotator cuff healing following repair. Clinically, it is possible that successful preoperative scapular rehabilitation may be necessary in order to achieve successful outcomes postoperatively; however, there is limited long-term clinical data evaluating the impact of scapular dyskinesis on rotator cuff repair healing, and this topic warrants further investigation.

Experimental model systems have also been utilized to assess the role of the acromion in rotator cuff pathology and treatment. Acromioplasty (resection of undersurface of the acromion) or subacromial decompression (removal of tissue in subacromial space) is often performed in conjunction with rotator cuff repair in order to provide greater clearance for the rotator cuff under the acromion and alleviate pain. Cadaveric biomechanical evaluation has demonstrated support for this technique with data that showed subacromial

decompression decreases peak pressures on the rotator cuff [44]. Similarly, in vivo experimental data in the rat model demonstrated that reduction of acromial space and subsequent external subacromial impingement in combination with overuse activity induces greater damage on the rotator cuff tendons than overuse alone [45].

Summary

In summary, clinical evidence and ex vivo and in vivo experimental model systems have shown that abnormal scapular motion and position can lead to impingement, increased stress on the rotator cuff, and tendon injury if left untreated. Evaluation and rehabilitation of abnormal scapular kinematics may help prevent tendon injury and could improve tendon healing following repair.

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The Scapula and the Throwing/ Overhead Athlete

6

Stephen J. Thomas and John D. Kelly IV

Introduction

Throwing is a very unique and complex act that often begins as early as 2 years of age. At this time children are developing the necessary neuromuscular control required to throw in a coordinated and efficient manner. Similar to the development of all other learned tasks such as walking or running, the neuromuscular system requires large repetitions to become efficient. At first the motion is very uncoordinated, especially between the lower and upper extremity, but with more repetitions these segments become much more efficient and transform into a fluid motion [1]. With continued development of the neuromuscular system and skeletal growth, the acceleration of segments and ultimately the ball will increase. At this time increased stress and force are placed on the body especially at the upper extremity [2, 3]. As stress on the muscles of the shoulder and scapula increase, so will tissue adaptations. At first these adaptations are important for creating increased strength and stability. However, for adaptations to continue in a positive direction, two factors must be properly managed. First is the frequency of applied stress. There must be a balance between stress and frequency of its application. As stress increases the frequency at which it is realized must equally decrease. Since there is no way of continually monitoring stress, this is often difficult to manage clinically. Therefore, many organizations like Little League Baseball have incorporated pitch counts as an attempt to limit the frequency [4]. However, this is also difficult to monitor as players participate in multiple leagues and showcases. In addition, these guidelines are very general and don't consider the individual stress for each player. The second factor that must be considered is recovery. Although managing stress and frequency are important, allowing proper recovery can minimize harmful adaptations such as chronic fatigue and muscle atrophy following high-frequency bouts. One area that experiences high stress due to eccentric muscular contractions is the posterior scapular stabilizers. During the deceleration phase of throwing, the scapular stabilizing muscles must both provide stability for the rotator cuff muscles to function properly and also protract around the thoracic cage to dissipate force [5–8]. Due to both the high stress and frequency, these muscles will fatigue both acutely and chronically.

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The acute and chronic fatigue that occurs in the scapular stabilizers will often affect the position and motion of the scapula [5, 6]. The static position is typically caused by tightness of soft tissue structures, and the altered motion of the scapula is typically caused by an altered neuromuscular control pattern coupled with soft tissue tightness. These adaptations will greatly affect the normal functioning of the scapula. When the scapula is not functioning properly, it often places more stress on the distal segments, like the shoulder and elbow. This additional stress on the distal segments can cause a degeneration of stabilizing tissues resulting in injuries like labral and ulnar collateral ligament tears. This chapter will go into depth about the normal and abnormal functions of the scapula in overhead athletes and how they present clinically. In addition, it will discuss the clinical implications to abnormal function.

Normal Scapular Function

Static Position

To understand how to evaluate and treat the scapula in overhead athletes, there must first be an understanding of what is normal. In a clinical setting, the first thing that is performed during a scapular assessment is observing the athlete in a static resting position. Due to the stress and frequency placed on the throwing arm, there are asymmetries that are often present even in healthy players. A common asymmetry observed is an increased amount of scapular upward rotation on the dominant arm [9–12]. This is often thought to be a positive adaptation since increased upward rotation would theoretically increase the subacromial space and minimize the risk of subacromial impingement. However, when examining subacromial space in overhead athletes, the results are inconsistent. Thomas et al. [13] found that the subacromial space was not different bilaterally at both 0° and 90° of glenohumeral abduction. The 90° position was examined due to it being the functional position of throwing and the most common position that replicate subacromial impingement symptoms. However, in contrast, Maenhout et al. [14] found that the subacromial space was increased on the dominant arm at 0°, 45°, and 60° of glenohumeral abduction. These positions were examined due to recent research that has demonstrated the insertion of the supraspinatus is medial to the acromion and not able to be impinged once the glenohumeral joint is abducted beyond 60° [15]. Combining the results of both studies may suggest that the development of subacromial impingement occurs at lower positions of abduction. However, once the degeneration of the supraspinatus tendon has progressed, the patient may be symptomatic at 90° of glenohumeral abduction due to the larger amount of internal stress or tension on the supraspinatus tendon.

The position, level of competition, and age may also have an effect on the amount of scapular upward rotation that is present in overhead athletes. First, Laudner et al. [16] found that pitchers had less upward rotation when compared to position players. Second, Thomas et al. [17] found that college baseball players had less scapular upward rotation compared to high school players. Lastly, Cools et al. [18] found that older (>16 years old) tennis players had less scapular upward rotation compared to younger (<14) players. Pairing the results of these three studies together may conclude that the amount of exposure has a detrimental effect on scapular upward rotation. In fact, additional research has found that over the course of both a high school and college season, players lost scapular upward rotation [19, 20]. This may suggest a chronic fatigue of the muscles involved with scapular upward rotation (upper trapezius, lower trapezius, and serratus anterior) even in healthy asymptomatic players. Maintaining proper scapular upward rotation is important to allow optimal functioning of the glenohumeral joint and minimize the risk of developing overuse injuries such as subacromial impingement syndrome [5, 8].

Kinematics

The overhead throwing motion is one of the fastest motions the human body can produce with velocities over 7000° per second at the glenohumeral joint [21]. Due to these extreme velocities at the glenohumeral joint, the scapula also has to

function at high speeds to maintain proper glenohumeral strength and stability. The scapula has previously been described as a sea lion balancing a ball on its nose [22, 23]. The sea lion (scapula) must move to maintain the balance and stability of the ball (humeral head). Due to the difficulties of assessing high-speed kinematics of the scapula in vivo, only two studies have examined the motion of the scapula during throwing [24, 25]. To simplify the kinematic study, the scapula was evaluated at specific portions of the pitching motion (stride foot contact, maximal external rotation, and maximal internal rotation). At stride foot contact, the scapula was found to be in a retracted, slightly upwardly rotated, and anteriorly tilted position. Moving from that position to maximal external rotation, the scapula further retracted and upwardly rotated. It also moved into external rotation and posterior tilt. It has been suggested that at maximal external rotation of the pitching motion, the scapula acts as a funnel to transfer energy from the lower extremity and trunk to the arm [7, 8]. Full scapular retraction will maximize the amount of energy transferred to the shoulder, while scapular upward rotation, external rotation, and posterior tilting will allow maximal clearance of the supraspinatus tendon. Maximum internal rotation occurs after ball release and is required to dissipate the large amount of energy created during the acceleration phase. At this position, it was found that the scapula was protracted, internally rotated, and anteriorly tilted. These scapular positions are at the other extreme of the available range of motion compared to maximal external rotation. To efficiently dissipate energy, joints will move through large ranges of motion. Dissipating energy over a greater range will lower the peak stress placed on the surrounding soft tissue structures (capsule, ligaments, tendons, and muscles). In theory, this will protect the structures from overuse, degeneration, and injury. Although it is important to know the normal high-speed motion of the scapula during throwing, it is impossible to assess clinically. Therefore, clinicians often have patients perform slow and controlled shoulder elevation and observe the motion of the scapula. During elevation the scapula on the dominant arm has been shown to have more upward rotation, posterior tilting, and internal rotation compared to the nondominant arm [12]. Also throwers often have more upward rotation, internal rotation and retraction compared to non-throwing athletes [9]. Therefore, it is unlikely that an overhead athlete will have perfect scapular symmetry. There are often subtle differences present. These differences are important to note when assessing overhead athletes clinically.

Kinetics

Kinetics or forces are required to produce the velocity and acceleration that occurs during throwing, thereby being linked with kinematics. Without kinetics, normal motion would not occur. Although there are occurrences when extreme forces are produced and result in a traumatic injury, during normal throwing, kinetics are submaximal and don't result in acute tissue disruption [26]. Instead the mechanical stimuli will cause tissue to adapt [27]. Typically this adaptation will result in a much stronger tissue that is able to withstand larger forces. However, if the load is too large or with too high of a frequency, it can cause tissue degeneration [28, 29]. Since throwing is a high-velocity activity, it has been shown to produce large forces and torques throughout the upper extremity [26, 30–32]. The two main phases of throwing that produce the most force are the acceleration and deceleration phases. During the acceleration phase, there are large anterior (300 N) and superior (400 N) forces occurring at the shoulder [26]. These forces are thought to be counterbalanced by contraction of the rotator cuff and biceps tendon to maintain glenohumeral stability. During the deceleration phase of throwing, a compression force of over 1000 N occurs and can be equated to ~1.5× body weight [26]. This is more than double the forces that are experienced during the acceleration phase. This force is created by the eccentric contraction of both the posterior rotator cuff and scapular stabilizers to help dissipate energy [5, 6]. As we discussed prior, from maximal external rotation to maximal internal rotation, the scapula moves through a large range of motion. This large motion reduces peak forces on the sur-

rounding joint structures and minimizes the microscope damage to the muscles during eccentric contraction, potentially speeding recovery.

Strength

Muscular strength is very important in the overhead athlete. Due to the large kinetics and repetitions that occur during throwing, the muscles of the scapula must adapt and become stronger. In fact several studies have investigated strength asymmetries on the dominant arm of overhead athletes. All three divisions of the trapezius muscle (upper, middle, and lower) have had increased strength when examined clinically [11, 33, 34]. In addition, the serratus anterior muscle has also been found to be stronger on the dominant arm [11]. These specific muscles are crucial to the proper functioning of the scapula especially during upward rotation. As players develop, there are often increases in muscular strength, velocity, and acceleration of the upper extremity. This will also increase the amount of eccentric force produced by the scapular stabilizers to decelerate the arm and minimize the stress that is often propagated to the glenohumeral joint and elbow. Therefore, maintenance of scapular strength throughout a game, season, and career is critical to minimize shoulder and elbow injuries.

Muscle Activity

Muscle activation is often isolated to the neuromuscular system unlike strength, which is often a combination of both the neuromuscular and mechanical (actin and myosin) elements. Due to this, examining muscle activity of the scapular stabilizers in overhead athletes will provide a more complete understanding of scapular muscle function during such tasks. During examination of scapular muscle activity, the upper trapezius and serratus anterior were found to have the strongest activity between maximal external rotation and maximal internal rotation of the overhead throw [35]. Increased serratus anterior activity was also found on the dominant arm during a simulated deceleration phase of throwing [36]. These results coincide with the kinematic results discussed previously. During this phase of throwing, the scapula is maintaining upward rotation and moving into protraction to absorb energy. The upper trapezius will help maintain upward rotation, and the serratus anterior will move the scapula into protraction. Interestingly, it has also been found that the activation of the scapular muscles correlates with the activation of the gluteus medius muscle on the contralateral leg [35]. This demonstrates a neuromuscular link between the dominant scapula and the contralateral hip in overhead throwers. Another important aspect of muscle activity to examine is timing. Latency or the proper timing of muscles should occur to function normally and produce normal kinematics. One study found the upper and middle trapezius had an increased latency on the dominant arm [37]. The upper trapezius also had an increased latency when compared to the middle trapezius and serratus anterior. For proper scapular function, the serratus anterior and lower trapezius must activate first. Activation of the upper trapezius prior to the lower trapezius and serratus anterior would cause a scapular hitch instead of upward rotation potentially leading to impingement of the rotator cuff. In fact, this abnormal motion is often seen in patients with rotator cuff tears [38].

In conclusion, due to the repetitive stress placed on the upper extremity during overhead throwing, the scapula often presents with normal asymmetries. Without knowledge of these normal asymmetries, clinicians may identify them as abnormal, and patients will be managed incorrectly. Therefore, understanding the normal function of the scapula and surrounding muscles is vital to assess the overhead athlete. Proper knowledge will allow for an adequate assessment to not only treat injured athletes but also to develop prevention programs.

Adaptations to Stress

Scapular Dyskinesis

Scapular dyskinesis has been defined as an observable alteration in the position or motion of the scapula [8, 39]. Although it is often thought to be,

it is not a diagnosis or an injury. Instead it is an impairment of optimal scapular motion and can be a risk factor for shoulder and elbow injuries. The prevalence of scapular dyskinesis in healthy asymptomatic overhead athletes has been reported as 61% compared to 33% in non-overhead athletes [40]. Therefore, dyskinesis may be present prior to the injury and contribute to the development of dysfunction. Interestingly, a rat model study examined the effect of scapular dyskinesis and demonstrated that it leads to the degeneration of the supraspinatus and biceps tendons [41]. This suggests that if dyskinesis is present in an overhead athlete, a corrective treatment approach should be instituted to minimize the risk of injury. It has also been reported that 94% of overhead athletes with shoulder or elbow injuries also have scapular dyskinesis [5]. This is more evidence linking scapular dyskinesis with shoulder and elbow injuries. From a clinical perspective, patients can often improve shoulder and elbow symptoms solely with a targeted scapular exercise program. By reestablishing normal scapular function, the stress on the surrounding glenohumeral and elbow joint structures can be minimized, thereby reducing or eliminating symptoms. The specific adaptations associated with altered scapular function in overhead throwing athletes will be discussed in detail.

Muscular Fatigue

One of the first adaptations that overhead athletes commonly experience is muscular fatigue. In general fatigue can also be thought of having two components that occur simultaneously: neural and mechanical [42]. Neural fatigue will cause nonoptimal firing patterns and reduced amplitudes of neural impulses. Instead of having very complex firing patterns that lead to optimal activation of muscles, the activation becomes less complex with large groups of motor units within muscles firing simultaneously [43, 44]. This is an attempt to make up for the reduced neural amplitude. This compensation pattern will result in uncoordinated scapular kinematics that leads to more force being placed on the glenohumeral posterior capsule and rotator cuff to decelerate the arm during follow-through.

Mechanical fatigue is typically caused by the microdamage of myosin and actin bonds during eccentric muscular contractions [45]. As the amount of damaged myosin and actin increases within the scapular stabilizers, the ability to mechanically generate force and absorb energy is reduced. This will also cause a negative feedback loop into the neural component, thereby creating nonoptimal neural firing [44]. Therefore, determining the optimal recovery required for throwing athletes will minimize the detrimental effects that occur due to acute and chronic fatigue and allow the scapular stabilizers to improve the ability to absorb eccentric energy during throwing.

In overhead athletes we can also examine fatigue both acutely and chronically. Acute fatigue commonly occurs over the course of one game or training session, while chronic fatigue typically occurs over the course of a season. One study visually identified the presence of scapular dyskinesis in a group of swimmers prior to and following a swimming practice [46]. Prior to the start of practice, none of the swimmers were identified as having scapular dyskinesis. However, immediately following practice, the presence of scapular dyskinesis increased to 82%. Another study examined scapular kinematics and subacromial space prior to and following a shoulder fatigue protocol in recreational overhead athletes [47]. The researchers found that the scapula was in more upward rotation, external rotation, and posterior tilt at both 45° and 60° of abduction following fatigue. In agreement, the subacromial space was also increased. Another study also found increased upward rotation following fatigue; however, lower trapezius muscle activity was decreased [48]. These results are surprising since the change is thought to be in a beneficial direction (more upward rotation, external rotation, and posterior tilt). Due to the subjects being recreational athletes, they potentially are still able to compensate during fatigue to minimize the risk of injury. In contrast, a recent study examined scapular upward rotation prior to and following a tennis-serving protocol in a group of college tennis players [49] found that upward rotation decreased immediately following the fatigue but returned to baseline at 24 h. This result is in agreement with prior hypothesis that scapular fatigue would

decrease upward rotation, thereby decreasing the subacromial space and compressing the supraspinatus tendon.

It is also important to examine overhead athletes over the course of a competitive season to assess chronic fatigue. Previous research has found that both high school and college baseball players have profound decreases in scapular upward rotation at various positions of glenohumeral abduction (60°, 90°, and 120°) upon completion of the competitive season [10, 19, 20]. In addition, another study found that pitchers had decreases in upward rotation, while position players had increases over the course of a season [50]. This suggests that the large force and repetitions that pitchers accumulate compared to position players are leading to a chronic fatigue of the muscle responsible for scapular upward rotation (upper and lower trapezius and serratus anterior). As stated previously the large repetitive eccentric force produced by the scapular stabilizers to decelerate the arm and maintain stability at the glenohumeral can cause significant acute fatigue during a game and without proper recovery will lead to chronic fatigue. With an improperly functioning scapula, the glenohumeral muscles (rotator cuff) will have to compensate. This will then accelerate the rate of microdamage and fatigue of the rotator cuff, thereby progressing the compensation more distally to the elbow [51].

Soft Tissue Tightness

In addition to muscle fatigue, the overhead athlete often develops tightness of several soft tissue structures, which include muscle, tendon, and capsule. Since the scapula serves as an attachment site for up to 18 muscles and tendons, tightness of these structures can affect the position and motion leading to long-term altered function. In this section we will discuss the common structures that develop tightness and the consequences to scapular function.

Pectoralis Minor

The pectoralis minor has its origin on the coracoid process of the scapula and inserting on ribs 3–5. The normal function of the pectoralis minor is to both protract and depress the scapula. As stated previously protraction of the scapula is very important to properly decelerate the arm during throwing. However, due to the chronic nature of overhead activities and the repetitive use of this muscle in normal scapular function, it often develops excessive tightness. It has been found that the length of the pectoralis minor directly correlates with a clinically identified forward shoulder posture [52]. More specifically, athletes with a tight pectoralis minor had increased anterior tilting and internal rotation of the scapula [53]. These positions have been shown to decrease the subacromial space [54] and are associated with an unstable scapula [5]. Interestingly, an intervention of pectoralis minor stretching did increase the length of the muscle/tendon unit but did not reestablish normal scapular kinematics [55]. This may suggest that long-term tightness alters the neuromuscular control and strength of the scapular stabilizers. Therefore, an isolated-stretching protocol only addresses one of the detrimental adaptations, suggesting that the optimal treatment may include muscle reeducation and strengthening of the scapular stabilizers.

Posterior Shoulder Tightness

It is well known that overhead athletes develop posterior shoulder tightness. This is presented clinically as a loss of internal rotation on the dominant arm compared to the nondominant. Therefore, it has been termed glenohumeral internal rotation deficits (GIRD). GIRD has been demonstrated to affect the position and motion of the scapula. One study found baseball players with 15° or more of GIRD had less scapular upward rotation and more protraction compared to players with 14° or less [56]. Similarly, another study identified that posterior shoulder tightness correlated with a forward shoulder posture [57]. Yet another study found that those with an average of 24° of GIRD had less subacromial space, and 6 weeks of stretching the posterior shoulder not only reduced GIRD but also increased the subacromial space [58]. This suggests that excessive GIRD can place unwanted stress on the scapular stabilizers leading to deficits in the

neuromuscular control and placing the rotator cuff at risk of injury. It also demonstrates that isolated stretching of the posterior shoulder can increase the subacromial space.

It is important to consider that GIRD is comprised of three tissue adaptations. First, humeral retroversion is a bony adaptation that occurs prior to skeletal maturity [59–61]. At birth the throwing humerus is in excessive retroversion (more glenohumeral external rotation and less internal rotation). In normal development the humerus transitions into a position of anteversion [61]. However, when the humerus is exposed to the stress of throwing at a young age, the humerus will remain in retroversion. Therefore, this adaptation has been shown to produce less internal rotation on the dominant arm compared to the nondominant arm [62]. Retroversion is often thought of being a positive adaptation due to acquiring additional external rotation without stretching or injuring soft tissue structures. The next two are soft tissue adaptations and therefore can contribute to scapular alterations if present. Posterior rotator cuff tightness has been suggested to also contribute to GIRD. When examining GIRD acutely over the course of a game, it was found that GIRD increased immediately following the game and remained increased for up to 3 days [63]. Due to the sudden loss of internal rotation, this increased GIRD has been attributed to the eccentric damage of the posterior rotator cuff muscles. However, directly measuring isolated posterior rotator cuff tightness is not possible, and therefore currently there is no direct link to contributing to altered scapular function. Posterior capsule tightness/thickness has also been shown to occur in overhead athletes and contribute to the clinical presentation of GIRD [60, 64, 65]. It has been found that college baseball players have a thicker [64] and stiffer [65] posterior capsule on the dominant arm, and it is negatively correlated to glenohumeral internal rotation [64]. This suggests that the thicker the posterior capsule, the less internal rotation is present. Interestingly, a positive correlation was also found between posterior capsule thickness and scapular upward rotation [64]. This suggests that the thicker the capsule, the more upward rotation is present. When tightness/thickness of the posterior capsule is present, it causes increased tissue stiffness [65] that decreases the available motion and therefore pulls the scapula into increased amounts of upward rotation. This increased upward rotation would not be beneficial since cadaver studies have demonstrated tightness of the posterior capsule causes a posterior/superior shift in the position of the humeral head on the glenoid face [66, 67]. This position would decrease the subacromial space and lead to subacromial or internal impingement.

Teres Major

Recent research has identified that overhead athletes can experience a loss of external rotation (<5° greater on throwing arm) on the throwing arm [68]. Clinically measuring the true loss of external rotation is often difficult due to the presence of humeral retroversion. However, using the amount of humeral retroversion to correct glenohumeral range of motion allows clinicians to identify if a loss of external rotation is present. Similar to other limitations in shoulder range of motion, a loss of external rotation in professional players was found to increase the risk of being on the disabled list for a shoulder injury twofold and undergoing shoulder surgery fourfold [68]. Although research has yet to identify the source of the loss of external rotation, it has been hypothesized to be caused by tightness of the teres major. The teres major is a unique muscle/tendon unit that originates from the inferior portion of the lateral border of the scapula and inserts on the medial ridge of the bicipital groove. Its function as an internal rotator places it under chronic overuse similar to many of the other shoulder muscles. Tightness of the teres major will limit external rotation with the scapula stabilized during a clinical exam. However, during overhead activities, the tightness can pull the scapula into greater amounts of upward rotation and posterior tilting. In fact, increased amounts of upward rotation and posterior tilting have been observed on the dominant arm of the overhead athlete [9, 12]. This forced motion may place increased stress on the scapular stabilizers and accelerate muscular fatigue. However, future research is required to confirm this.

Long Head of the Triceps

The long head of the triceps is a two-joint muscle that originates from the infraglenoid tubercle and inserts on the olecranon process. During overhead motion, the triceps is responsible for decelerating both the shoulder and the elbow. Producing large eccentric force to control both joints during follow-through can result in significant microdamage to the muscle [69]. This repetitive microdamage can lead to chronic tightness within the muscle/tendon unit. In fact, overhead athletes often present with a loss of scapula-stabilized glenohumeral forward flexion on the dominant arm [68]. It has also been demonstrated that a loss of dominant arm forward flexion of ≥5° resulted in a 2.8 times greater risk of elbow injury [68]. Another structure that can also contribute to a loss of forward flexion is the latissimus dorsi. However, the latissimus dorsi does not have an attachment to the scapula and therefore would only limit forward flexion with the scapula not stabilized. Due to the attachment of the long head of the triceps on the scapula, tightness would cause forced scapular upward rotation during forward flexion or even abduction. In addition, due to the triceps being a two-joint muscle, the body may compensate for tightness by altering elbow motion during overhead activities. For example, if excessive triceps tightness is present during the acceleration phase of throwing, the player may move into more elbow extension to alleviate tension. Increased elbow extension during the acceleration phase will place excessive valgus stress on the ulnar collateral ligament (UCL) [26]. Overtime this can have detrimental effects on elbow stability.

Effect of Pain

There are many things that can change the normal kinematics, kinetics, and muscle function at a joint. As discussed, fatigue and soft tissue tightness are very common among overhead athletes. However, something that has not been discussed yet and is extremely common in overhead athletes is pain. The prevalence of pain in overhead athletes is very high, even among youth athletes. About 16% youth baseball players experience shoulder pain, while 29% experience elbow pain [70]. Similarly, elite swimmers have been reported to have an astounding 91% prevalence of shoulder pain during activity [71]. Since large amounts of overhead athletes often participate with pain, it is important to understand the effect pain will have on the function of the scapula. Pain is a neurological signal delivered from structures within the body that are experiencing abnormal stress and interpreted by the brain. In an effort to protect itself, the body will compensate in such a way to avoid the pain. This compensation typically results in temporarily placing increased stress somewhere else. Overhead athletes often experience pain in the late cocking position (90° abduction and elbow flexion with maximum external rotation) or the acceleration phase due to the large joint forces and torques at the shoulder and elbow [26]. It is within these positions that the body will attempt to alter motion to alleviate pain. It has been found that overhead athletes with pain have less scapular upward rotation at both 45° and 90° of glenohumeral abduction [72, 73]. This potentially can further exacerbate compression to the supraspinatus through an impingement mechanism. In addition, athletes with pain have been clinically reported to have increased SICK scapula scores and core instability [74]. Although it is often thought that the pain will create alterations in kinematics, it remains to be determined if the altered kinematics were present prior to the pain. Previous research has identified several scapular adaptations in healthy asymptomatic overhead athletes. However, future prospective research is required to confirm the cascade of events leading to pain.

In conclusion, overhead athletes are very unique due to the demands of the activities required during competition. Therefore, understanding the normal scapular function and the common adaptations that occur in this population will improve a clinician's ability to assess, prevent, and treat injuries. The next section will guide the clinician through a thorough clinical exam and treatment approach in this population.

Clinical Implications of Scapular Dyskinesis

Scapular dyskinesis has protean effects on shoulder function as stated previously. Clinically, overhead athletes with scapular dyskinesis often present with injury to other soft tissue structures within the shoulder. This section will focus on discussing the clinical implications of these injuries in the presence of scapular dyskinesis and the evidence-based approach to a comprehensive exam.

Rotator Cuff

All rotator cuff muscles originate on the scapula. Thus, a stable scapula is essential to provide a secure "platform" for optimal cuff function. The scapula retraction test illustrates this principle: measurable increase in resisted forward flexion strength may be noted when a dyskinetic scapula is "reduced" or manually stabilized [75] (Fig. 6.1). Secondly, proper scapula position is necessary to optimize the length-tension relationship of each of the four rotator cuff muscles. For example, a protracted scapula does not afford proper tension for supraspinatus action since the origin (scapula) and insertion (greater tuberosity) of the muscle/tendon unit are appreciably shortened. Consequences of cuff weakness include loss of concavity-compression function with resultant loss of inherent shoulder stability [76]. Since the rotator cuff is a significant dynamic stabilizer to the glenohumeral joint, increase strain to static stabilizers (labrum and capsule) will ensue. In addition, a weakened muscle/tendon unit may be less likely to withstand the eccentric loads realized during shoulder motion. For example, during the follow-through phase of throwing, large tensile forces are generated at the posterior cuff during deceleration [77]. An infraspinatus that is working at a mechanical disadvantage will not only fatigue sooner and transfer increased load to the posterior capsule but also be expected to fail at lower eccentric loads. In other words, a muscle/tendon unit operating at a suboptimal length-tension relationship will fail at lower stresses. It is therefore no surprise that approximately one third of patients with labral tears have a concomitant rotator cuff tear [78]. A compromised infraspinatus will furthermore be less effective in restraining anterior shear during deceleration with a concomitant increased load to the leading edge of the supraspinatus tendon. In addition, the infraspinatus, by virtue of its posterior force vector, protects the glenohumeral joint from excessive anterior translational stress, especially during the abduction external rotation position (ABER) [79–81]. Thus, it stress shields the anterior capsule and labrum from undergoing attritional damage.

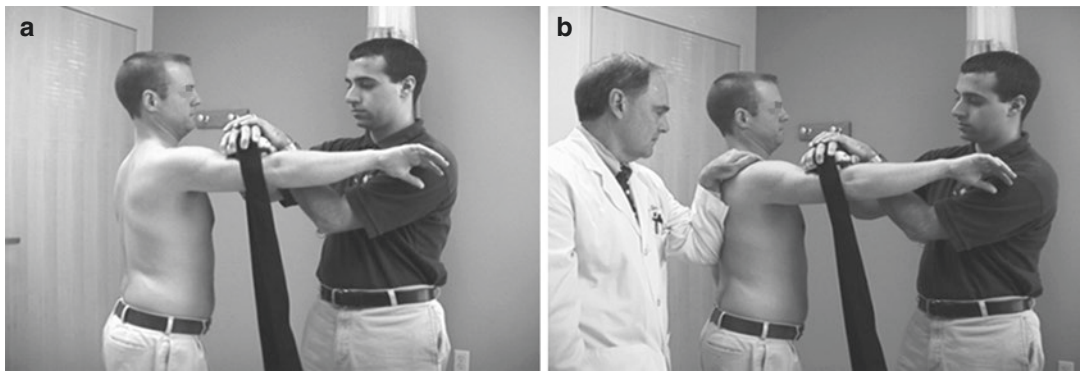

Fig. 6.1 (**a**) The empty-can position for testing. (**b**) The scapular retraction position for testing. The arm is in the empty-can position. The scapula is lightly held in retraction by forearm pressure on the medial scapular border, while the patient exerts maximum resistance against the handheld dynamometer (Reprint from Kibler WB, Sciascia A, Dome D. Evaluation of apparent and absolute supraspinatus strength in patients with shoulder injury using the scapular retraction test. Am J Sports Med. 2006;34(10):1643–7, with permission from SAGE)

Increased strain to the posterior capsule has been implicated in the genesis of GIRD, and undersurface leading-edge supraspinatus injury has been posited to occur due to increased eccentric loading during follow-through. A protracted scapula lessens the translational zone of movement by which the scapula can absorb deceleration stress; thus, increase strain will be realized at the posterior cuff and capsule. The "full tank of energy" position of late cocking described by Kibler et al. [8] is dependent on adequate scapular retraction. If retraction is insufficient, full external rotation of the humerus is precluded, and velocity of the throw is diminished. The rotator and elbow will also attempt to compensate for the reduced amount of energy resulting in increased stress.

Hypertwist

Throwers achieve extraordinary degrees of external rotation due to increased retroversion and anterior capsular laxity and "pseudolaxity" of the anterior capsule due to GIRD [5, 6]. This supraphysiologic humeral rotation creates inordinate shear stress in the rotator cuff, which may manifest as interlaminar rotator cuff tearing. The superior and inferior laminae of the supraspinatus and infraspinatus may separate and form the partial articular tears with intratendinous extension (PAINT lesion) as described by Conway [82]. This shear stress can be exacerbated by a malfunctioning scapula. For example, if the scapula does not posterior tilt during maximum external rotation, the rotator cuff will experience additional twisting, thereby increasing the likelihood of a PAINT lesion.

Internal Impingement

Excessive scapular protraction and anterior tilting lessens the distance between the glenoid and greater tuberosity during the late cocking phase of throwing. Loss of scapular retraction also causes the thrower to increase horizontal abduction, thus throwing out of the scapular plane with resultant-increased contact between the posterior-superior labrum and greater tuberosity. "Pinching" of the supraspinatus may result with subsequent damage to cuff tendon fibers. This internal impingement [83] manifests with undersurface tearing of the supraspinatus/infraspinatus junction (Fig. 6.2). In time, this labral "pinching" may lead to posterior-superior labral fraying (Fig. 6.3). Scapular protraction and GIRD are inextricably linked. Loss of internal rotation will lead to scapula windup, as described by Kibler et al. [84], whereupon the scapula migrates into protraction in order to provide internal rotation. If a thrower is restrained from following through due to a tight posterior capsule, the scapula will migrate "up and around" the thorax in order to allow the arm to be directed toward home plate. Weakness of both static and

Fig. 6.2 Arthroscopic demonstration of undersurface rotator cuff tearing at the supraspinatus/infraspinatus junction

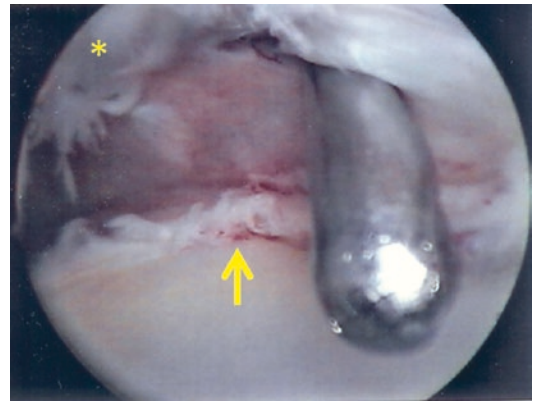

Fig. 6.3 Arthroscopic demonstration of posterior-superior labral fraying (*arrow*) with concurrent posterior-superior undersurface rotator cuff tearing (*asterisk*)

dynamic restraints will eventuate in a scapula that rests in an internally rotated (protracted) position. In addition, posterior capsular contracture causes a relative posterior-superior shift of the humeral head in late cocking, further increasing "peel-back" stresses to the posterior-superior labrum and potentiating the creation of a type two labral injury [85]. The "relocation test" will be positive in this scenario as posterior pressure applied to the upper humerus will lessen tuberosity/glenoid contact with the scapula manually placed in an optimal position.

External Impingement

A protracted scapula diminishes space between the humeral head and acromion. As the scapula protracts and follows the contour of the ribs, it tilts forward and increases acromial—great tuberosity contact in forward flexion [54]. A painful abduction arc that is relieved with scapula assistance (scapula assistance test) may confirm the presence of a symptomatic functional impingement. In fact, Muraki et al. [86] showed that the presence of a tight posterior-inferior capsule increases humeral head-coracoacromial contact pressure during the follow-through phase of throwing.

Scapula and the Elbow

The incidence of elbow injuries in pitchers has skyrocketed [87]. Surely increased pitch counts share the blame for this epidemic. However, the scapula plays a major role in the etiology of elbow injuries, especially the failure of the ulnar collateral ligament (UCL). There have been established relationships of GIRD [88] and total range of motion deficits [88–90] and UCL injuries. A loss of internal rotation (GIRD) essentially diminishes long-axis rotation of the upper arm. Proximal segment impairment will predictably transfer increased load distally (elbow) in order to achieve the internal rotation necessary to propel a baseball to home plate. In fact, Suzuki et al. [91] have shown that scapula fatigue leads to compensatory motions at the elbow. The loss of humeral external rotation seen in throwers suffering from UCL injury may serve as a protective mechanism [89] by which the thrower avoids the inordinate valgus elbow torque realized with extreme humeral external rotation. A protracted scapula potentiates throwing "out of the scapular plane", i.e., in relative humeral horizontal abduction. This increases the duration of valgus loads application to the elbow during throwing. The longer the upper arm is behind the thorax, the more the elbow will realize a valgus moment. Secondly, a "dropped elbow" seen during some deliveries increases the distance from the center of the body's rotational axis to the end of the moment arm (the hand). This increase in moment arm length merely increases the amount of centripetal force applied to the elbow. Reasons for lowering the elbow during pitching are many and include core weakness, posterior capsular tightness, scapular protraction, and cuff weakness.

Examination

Rotator Cuff

As stated, the throwing shoulder experiences cuff injury in three chief locations: leading edge of supraspinatus, due to eccentric load failure, interlaminar tears of the supraspinatus and infraspinatus due to hypertwist, and the junction of the supraspinatus and infraspinatus due to internal impingement. An effective examination should delineate a fairly precise zone of injury.

Supraspinatus

The "Whipple test" (Fig. 6.4) as described by Savoie et al. [92] detects weakness of the leading edge of the supraspinatus. The test is performed by asking the patient to forward flex and place the arm in extreme adduction. Pain and/or weakness during resistance of forward flexion constitute a positive result. While both the "full can" and "empty can" appear to equally load the supraspinatus proper, the "full can" may serve as a superior test to measure entire supraspinatus integrity since it is generally

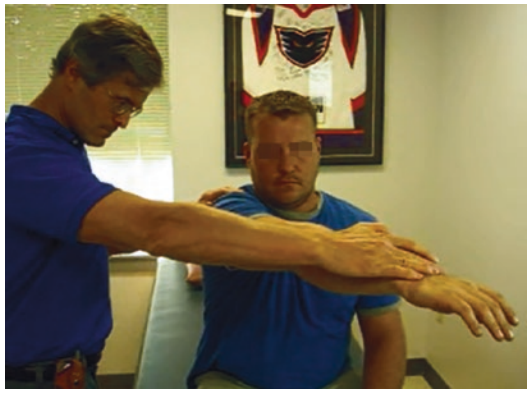

Fig. 6.4 The Whipple test is performed by positioning the patient's shoulder in 90° of shoulder forward flexion with maximal horizontal adduction. The examiner then applies an inferior-directed force at the distal forearm, while the patient maintains the position. A positive test is weakness or pain

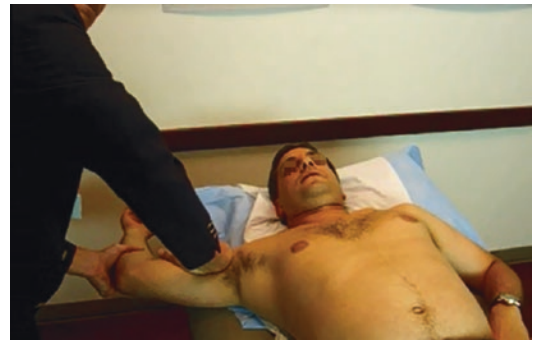

Fig. 6.5 The relocation test is performed by positioning the patient supine on the examine table with the shoulder in 90° of abduction and 90° of external rotation. In this position the examiner applies a posterior force to the anterior proximal humerus, thereby alleviating potential internal impingement symptoms

associated with less pain provocation [93]. Thus, weakness demonstrated with a positive Whipple test in the face of a normal "full can" test suggests a partial-thickness anterior supraspinatus lesion. Weakness during the Whipple test is truly positive when the scapula is held in retraction (scapular retraction test) since a protracted scapula (unstable base) will compromise supraspinatus function [75].

Internal Impingement

Compression of the supraspinatus between the posterior-superior glenoid and greater tuberosity occurs in the late cocking or ABER position. The relocation test (Fig. 6.5) is superb for detection of this phenomenon. In the ABER position, posterior pain that is relieved with a posterior force applied to the proximal humerus is considered a positive sign for internal impingement, i.e., a positive relocation maneuver. As stated previously, posterior force applied to the humerus lessens the impingement by increasing the tuberosity glenoid distance.

Labral Tear

Although numerous examination tests to detect labral injury have demonstrated inconsistent results [94], the dynamic labral shear (DLS) test has been shown by Kibler et al. [95] to demonstrate excellent sensitivity, specificity, and accuracy. During this maneuver, the abducted arm is brought into extreme external rotation and horizontal abduction. The arm is then forcibly lowered and thereby "shearing" the posterior cuff against the posterior-superior labrum (Fig. 6.6). Kibler et al. [95] have also shown that although the O'Brien's test, whereby the forward flexed, adducted, and internally rotated arm resists downward pressure, was less sensitive than the DLS in labral tear detection, the combination of both tests demonstrated the most consistent prediction of arthroscopic findings of labral injury.

Imaging Findings in Thrower's Shoulder

Advanced imaging modalities, especially MRI scans, can greatly aid in diagnosis of scapula-related cuff and labral injury. MRI arthrograms, whereupon dye is injected into the shoulder capsule, have enhanced the yield of detecting cuff and labral injury [96] (Fig. 6.7). However, it must be noted that the highly sensitive new-generation MRI scanners may detect many "inconsequential" labral tears [97]. In fact, some labral stretching may be adaptive and allow the thrower to

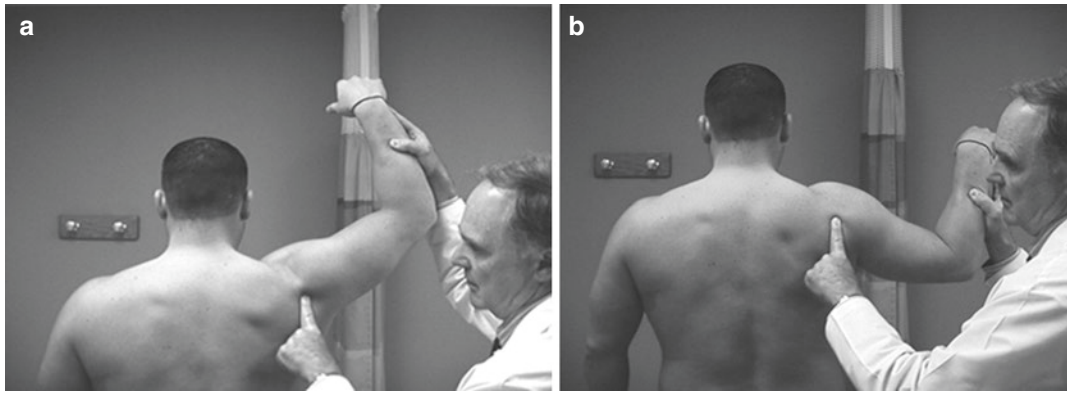

Fig. 6.6 (**a**) Dynamic labral shear (DLS) test. With the patient in a standing position, the involved arm is flexed 90° at the elbow, abducted in the scapular plane to above 120°, and externally rotated to tightness. It is then guided into maximal horizontal abduction. (**b**) The examiner applies a shear load to the joint by maintaining external rotation and horizontal abduction and lowering the arm from 120° to 60° of abduction. A positive test is indicated by reproduction of the pain and/or a painful click or catch in the joint line along the posterior joint line between 120° and 90° of abduction (Reprint from Ben Kibler W, Sciascia AD, Hester P, Dome D, Jacobs C. Clinical utility of traditional and new tests in the diagnosis of biceps tendon injuries and superior labrum anterior and posterior lesions in the shoulder. Am J Sports Med. 2009;37(9):1840–7, with permission from SAGE)

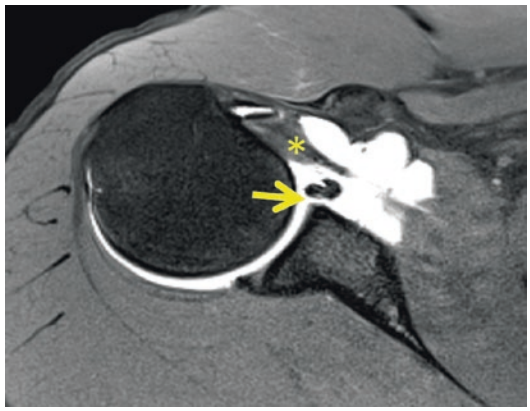

Fig. 6.7 MRI arthrogram indicating enhanced visibility of a cord like MGHL (*asterisk*) and Buford complex—anatomic variant mimicking labral tear (*arrow*)

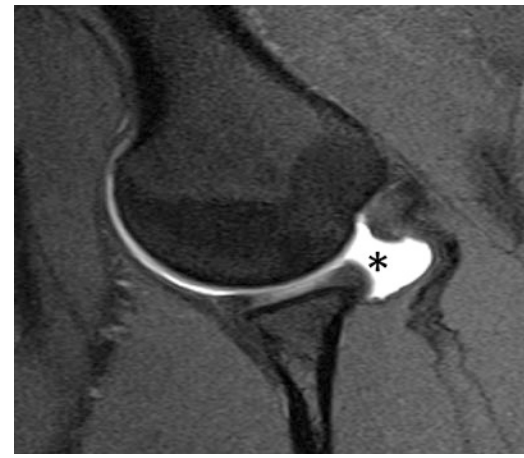

Fig. 6.8 MRI arthrogram in the ABER position. The posterior-superior glenoid, rotator cuff, and labrum are easily identified (*asterisk*)

obtain the "slot." Thus, all imaging findings must support the exam findings. For rotator cuff undersurface tearing, the MRI ABER view, whereupon axial images are obtained in ABER, has been shown to increase sensitivity in detection of partial articular-side damage [98]. Furthermore, the ABER view has been found to increase detection of both internal impingement and glenoid labrum tears [99, 100] (Fig. 6.8). Subtle undersurface tearing of the infraspinatus, as seen in internal impingement, may present with a small cystic change on the posterior humeral head in the vicinity of the supraspinatus/infraspinatus junction (Fig. 6.9).

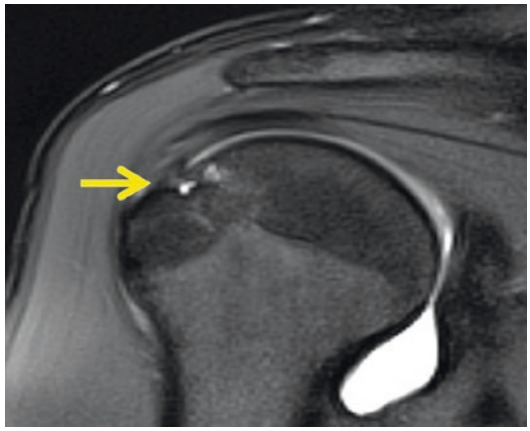

Fig. 6.9 MRI indicating cystic changes on the insertion site of the supraspinatus (*arrow*) due to chronic internal impingement

Treatment

Key Principles of Rehabilitation

Scapula-related throwing injuries are chiefly treated conservatively with restoration of "shoulder homeostasis" being the goal. A principle goal should be restoration of symptomatic scapular malposition. Tate et al. [101] have discovered that not all scapular asymmetry is accompanied by symptoms. In fact, relief of cuff or labral-related pain with scapula reposition (retraction or scapular assistance test) indicates a consequential scapular position issue. Repositioning of the scapula is realized through strengthening the scapular retractor muscles and stretching tightened structures which potentiate protraction. Many athletes who engage in weight training pay nearly undivided attention to protraction-lending exercises (bench press) while neglecting important scapular retraction exercises such as scapular pinches, close-grip rows, and prone horizontal abduction with external rotation [102], which are necessary for shoulder homeostasis. The "low row" is ideal for selectively activating serratus anterior and rhomboids. Blackburn exercises [103] are superb at training retractors as well. "Lawn mower" pulls, as popularized by Kibler et al. [104], introduce more core activation, while concomitantly training scapular retraction. Comprehensive kinetic chain evaluation is paramount in returning the thrower to full activity. Subtle findings in the overhead athlete may include a weakness in stance leg abduction, lead leg loss of hip internal rotation, lead leg quad tightness, and loss of stance leg ankle dorsiflexion. Posterior capsule and cuff tightness must be addressed with sleeper stretches and cross-body adduction stretches in order to prevent the recurrence of scapular "windup" [84].

Indications for Surgery

Failure of at least 3 months of quality and "enlightened" physical therapy with positive examination findings of a consequential labral tear warrants arthroscopic evaluation. In addition, the presence of overt mechanical symptoms such as locking, catching, and persistent "dead-arm" sensation is also an indication for arthroscopic intervention. As stated, one must be mindful that not all labral separations are pathologic and that some labral stretching may, in fact, be adaptive.

Surgical Pearls

The lateral decubitus position affords excellent exposure to all aspects of the shoulder, especially the posterior and inferior recesses, and is strongly preferred by the senior author. A standard posterior viewing portal approximately 2 cm medial and 2 cm distal to the corner of the acromion is established first. Two anterior portals, one at the anterolateral corner of the acromion (AL portal) and another 2 cm lateral to the tip of the coracoid (standard anterior portal—AP), are established next. While viewing from the AL portal, a liberating type instrument is used from the AP to free the labrum from the glenoid. Attritional labral stretching does not warrant repair. Complete labral separation with concomitant fissuring of glenoid and/or labral surface usually indicates a consequential labral injury. A dynamic "peel-back" test whereupon the arm is removed from traction and placed into ABER will reveal not only frank labral separation but also increased posterior-superior humeral translation and contact with supraspinatus fibers (internal impingement).

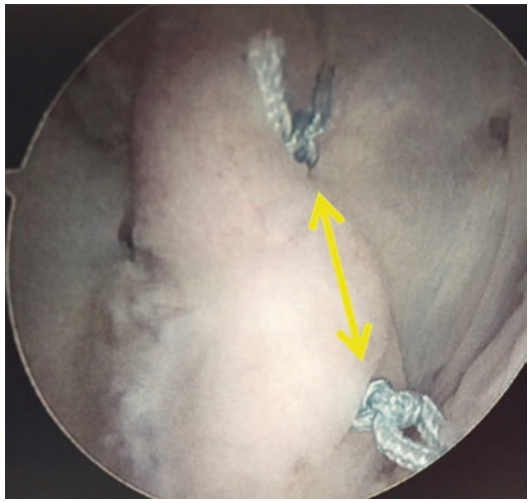

Fig. 6.10 Arthroscopic view of a horizontal mattress suture knot used to secure the labrum (*double arrow*)

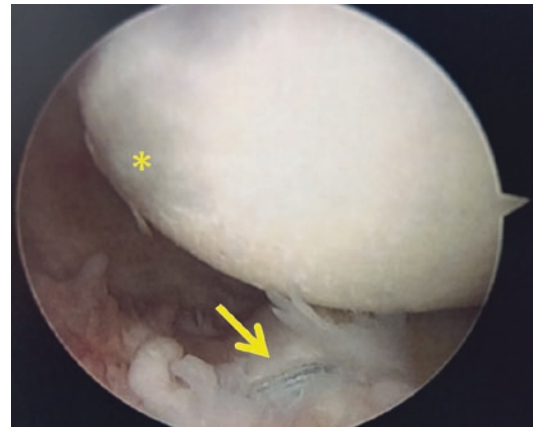

Fig. 6.11 Arthroscopic view demonstrating chondromalacia to humeral head (*asterisk*) due to suture abrasion from prominent suture material (*arrow*)

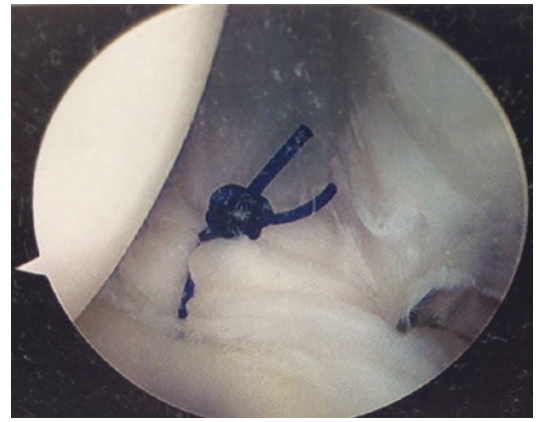

Fig. 6.12 Arthroscopic view demonstrating the use of a simple suture configuration using PDS absorbable suture

Signs of cuff failure, whether fraying of the supra/infra junction as seen in internal impingement or undersurface supraspinatus fiber disruption due to eccentric load-induced cuff failure, further confirm that the labral injury may have engendered adverse consequences. With the AL viewing, portal percutaneous anchor insertion is accomplished via the "Port of Wilmington" [78]. Sutures are shuttled via a Neviaser portal, and great care is taken to capture labral tissue only. Over constraint, especially of any posterior capsular tissue, can be disastrous to a thrower. The senior author favors less compliant suture material as the superior labrum has inherent flexibility. Some of the newer suture materials available today are extremely stiff and do not afford the labral excursion necessary to negotiate overhead throwing. Secondly, the senior author favors horizontal mattress knotted configuration for two reasons (Fig. 6.10). First, a horizontal suture pattern restores labral height to its native configuration [105]. As Yoo et al. [106] have shown, shoulder function post-instability surgery correlates with labral height. Knotless suture configurations "push" the labrum onto the glenoid and do not roll the labrum onto the articular surface such as seen with knots. Secondly horizontal mattress configurations displace suture material well away from the articular surface. The senior author has witnessed considerable suture abrasion from prominent suture material in several cases (Fig. 6.11). If one chooses a simple suture configuration, then an absorbable suture material, such as PDS, is recommended (Fig. 6.12).

Posterior Capsular Release

The senior author only rarely performs posterior capsular release. Indications include true stretch nonresponders, which are encountered only rarely when the help of a shoulder therapist is enlisted [6]. Furthermore, the capsule must be

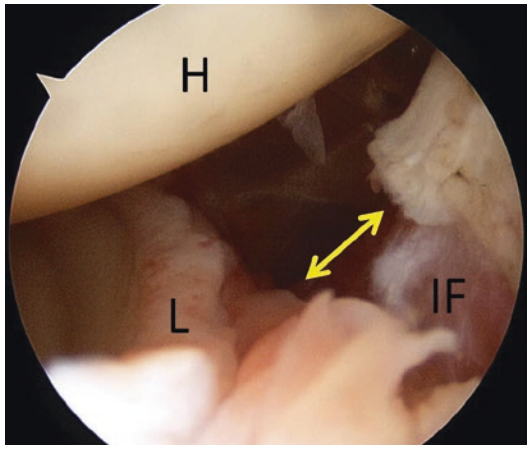

Fig. 6.13 Arthroscopic view of the posterior-inferior shoulder with the (*H*) humeral head superior and the (*L*) labrum inferior. Posterior capsule release (*double arrow*) is confirmed by visibility of the infraspinatus muscle (*IF*)

demonstrated to be thicker and more robust. If a thin posterior capsule is encountered, then release is contraindicated. Stretch nonresponders tend to be more mature throwers (late collegiate or professional) who maintain a symptomatic internal rotation deficit of at least 25° or a total arc of motion loss of at least 5°. Angled capsular punches are used, lifting "upward" while cutting in order to avoid axillary nerve injury (Fig. 6.13).

Prevention

The large preponderance of throwing-related injuries to both the shoulder and elbow is indeed preventable. A comprehensive kinetic chain evaluation in the adolescent overhead athlete is paramount in sidestepping cuff and labral injury. A chief component of the kinetic chain is the scapula. Scapular asymmetry may initially be a subtle adaptive change to the thrower, but in time the adverse mechanical consequences of scapular malposition will exact its toll resulting in muscle inhibition and sometimes tissue breakdown of the rotator cuff and labrum. Symptomatic scapular asymmetry, if recognized and corrected early, can avert labral and rotator cuff injury in the throwing athlete.

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Scapular Dyskinesis and Glenohumeral Instability

7

W. Ben Kibler and Aaron D. Sciascia

Anatomy and Biomechanics

From a biomechanical perspective, the glenohumeral (GH) joint is a closed chain mechanism comprised of the bones, ligaments, and muscles that balances stability against excessive translations with mobility necessary to achieve positions and motions of the arm and hand to accomplish specific tasks [1]. For almost all normal shoulder/arm functions, GH kinematics that results from this balance resembles a ball and socket arrangement.

The scapula, as the "G" of GH, is a key element in the closed chain mechanism. The scapula plays multiple roles in creating and maintaining the ball and socket kinematics. First, the glenoid must be dynamically positioned in three-dimensional space to maintain the "glenohumeral angle"—the orientation of the glenoid cavity and the long axis of the humerus from the head to the elbow—in a "safe zone" that minimizes glenohumeral shear [2], maximizes concavity/compression [3, 4], and minimizes muscular activation necessary to maintain joint stability [5]. This angle has been estimated clinically by Jobe to be ±30° and has been verified by a biomechanical study that showed that muscle activation was most efficient in maintaining joint stability when the glenohumeral angle measured ±29.3° [6]. If the angle is maintained within these parameters, the resultant force vectors are directed within the glenoid cavity, shear forces are minimized, tension on the ligaments is minimized, and the muscle activation requirements are minimized, creating the most efficient joint conditions for stability. In this position, all of the intrinsic shoulder muscles of the rotator cuff can pull in relatively straight lines to maximize concavity/compression into the joint.

The dynamic stabilization is important due to the lack of adequate static stabilization from the bony anatomy and the ligaments in the midranges of motion. This dynamic positioning is part of an integrated coordination of multiple segments throughout the entire kinetic chain in an anticipated response to the required demands and loads on the shoulder during daily and athletic activities.

Achievement of this scapular position requires that the scapula be positioned in anticipation of arm and shoulder movements. There are several reasons for this anticipatory requirement. The speeds, forces, and motions around the shoulder are frequently too rapid and occur too quickly for sensory feedback to adjust muscle activation to move the scapula [1, 5]. Scapular movement by

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itself creates only up to 40% of the observed forces necessary for forward shoulder and arm acceleration [5]. The majority of the forces developed through the kinetic chain activation sequence to move the arm forward come from the hip/trunk activation (core stabilization), which creates interactive moments to position and move the arm in space [7, 8], similar to the movement of the end of a whip. In normal shoulder movements, these anticipatory motions are part of a biomechanical closed chain that couples scapular and arm motions [1, 5, 8].

The muscular activation sequences that allow this anticipatory bony positioning are learned, preprogrammed patterns, defined as force-dependent activation patterns [5, 9], that integrate multiple muscles to move multiple joints [10–12]. These patterns use feed-forward sensory information to position the bones and joints in the most efficient manner. They are highly developed and are quick to drop out with injury or disuse.

Typical muscle activation patterns involve stabilization of the contralateral hip and trunk extension as a base for scapular activity [13], anterior and posterior core stabilization for force development at the shoulder [14], sequential activation of contralateral, then ipsilateral abdominals before rotator cuff activation [15], and activation of scapular stabilizers before rotator cuff activation [16].

The functional and observable result of the muscle activations producing dynamic positioning is scapulohumeral rhythm (SHR), the coupled synchronous movement of the arm and scapula. SHR has been likened to a "ball on a sea lion's nose" (Carter Rowe Personal Communication), describing the dynamic nature of the nose (the glenoid) actively moving in anticipation and response to movement of the ball (humerus) to keep the ball centered on the nose.

Second, the scapula is the stable base and point of origin for all of the intrinsic and extrinsic muscles that dynamically stabilize the GH joint in almost all ranges of motion. Coordinated, balanced muscle activations are responsible for maximizing GH stability through about 90% of the joint motions in all planes [1]. Maximal activation of all the rotator cuff and deltoid muscles in concentric and eccentric activities to increase the joint concavity/compression occurs off a stabilized scapula [17–20]. Demonstrated muscle strength can be improved by as much as 24% off a stabilized scapula [18, 20].

Third, optimal scapular position and motion are required to limit loads on the ligaments and other passive constraints in the joint. Increased scapular protraction creates excessive tensile loads on the anterior inferior GH ligament [21], increasing the risk of GH instability. Also, increased glenoid antetilting in protraction increases compression and shear loads on the posterior superior glenoid labrum, creating injury and decreasing the effectiveness of the labrum as a washer and a gasket to maximize GH stability [1, 22]. For example, altered trunk and scapular position during the overhead tennis motion increases the internal joint forces and is associated with joint injury [23].

In summary, the scapula's roles in GH stability are directed toward developing the maximal efficiency to maintain the rather fragile ball and socket kinematics in the face of the large loads, forces, and strains imposed by athletic and industrial demands on the shoulder. Alteration of the scapular roles may decrease this efficiency, leading to increased loads, possible injury, and increased dysfunction, and may make treatment more difficult.

Alterations of the Scapula Associated with Glenohumeral Instability

Alterations of static scapular position or dynamic scapular motion, collectively termed scapular dyskinesis (Fig. 7.1), are frequent in patients with demonstrated GH instability occurring in between 67 and 80% of patients [2, 24, 25]. Scapular dyskinesis appears to alter normal shoulder biomechanics and joint stability by altering normal scapular kinematics. Type I (excessive anterior tilt) and type II (excessive

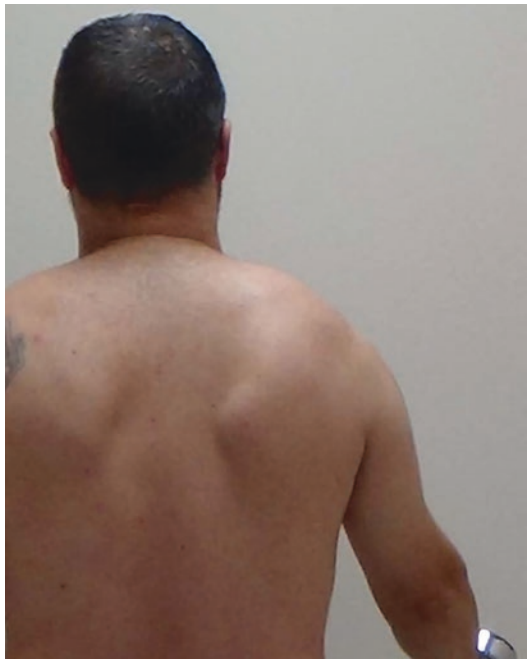


Fig. 7.1 Example of scapular dyskinesis during arm lowering

lateral rotation) dyskinesis positions have the effect of increasing the glenohumeral angle beyond the "safe zone," of increasing anterior shear, and of increasing tensile loads on the anterior band of the inferior glenohumeral ligament [21, 26]. Excessive scapular protraction, which results from type I or II patterns, also decreases maximum rotator cuff activation, decreasing the "compressor cuff" muscle function that establishes dynamic stability. Type III (lack of acromial elevation) position creates impingement upon arm elevation, establishing the "instability/impingement" connection [27]. However, no specific dyskinesis pattern is commonly associated with a specific type of GH instability. In many cases, the dyskinesis is seen as a result of the injury, but in some cases it may be a key causative factor. In any case, it should be considered an impairment of optimal shoulder stability and function. It appears that dyskinesis is primarily due to altered muscle flexibility, strength imbalance, and/or altered muscle activations but can be seen following bony or joint injury.

In patients with posttraumatic anterior or posterior instability with Bankart lesions, the dyskinesis is most frequently secondary to the anatomic injury, and total restoration of scapular kinematics requires restoration of the pathoanatomy. However, in cases where the operative treatment is delayed, to try to finish out a season, or nonoperative treatment is elected, rehabilitation of the muscles to stabilize the scapula is an integral part of the overall treatment [28–31].

In patients with GH instability due to repetitive microtrauma, which is usually a process over time, weakness and inhibition of the lower trapezius and serratus anterior, coupled with inflexibility in the pectoralis minor and latissimus dorsi, are common findings that create the scapular dyskinesis and scapular protraction [24, 32].

Patients with MDI have been shown to exhibit inhibition of the subscapularis, supraspinatus, lower trapezius, and serratus anterior, coupled with increased activation of pectoralis minor and latissimus dorsi [33–35]. These activations protract the scapula and inferiorly tilt the glenoid, removing most of the bony contribution to inferior stability. The latissimus dorsi activation pulls the humeral head inferiorly, creating the characteristic instability in the midranges of GH motion. The scapula will demonstrate the inferior angle prominance dyskinetic pattern when the patient indicates the instability.

In summary, scapular dyskinesis is commonly associated with all types of GH instability. The dyskinetic positions and motions create and exacerbate altered GH kinematics and muscle activations and impair shoulder function by decreasing the "sea lion's" ability to maintain "the ball" on its nose. This increases the dysfunction of the instability and can decrease the effectiveness of nonoperative or postoperative rehabilitation protocols. Evaluation for the presence or absence of scapular dyskinesis should be included as part of a comprehensive examination of the unstable shoulder.

Physical Examination

Scapular Evaluation in Glenohumeral Instability

Clinical evaluation of scapular position and motion is often difficult due to the overlying musculature and the lack of objective reproducible tests to measure the scapula. The examination may be made more reproducible by examining in a specific sequence involving position, motion, strength, and dynamic stabilization tests.

Static scapular position may be evaluated by observing the resting posture of both scapulae. Marking the superior and inferior medial borders with a marker is a good help. Scapular dyskinesis patterns can often be demonstrated by observing the resting position of the scapula. The altered position at rest has been termed the **S**capula malposition/**I**nferior medial border prominence/**C**oracoid pain/scapular dys**K**inesis (SICK) scapula and is characterized by apparent inferior drooping, which is actually due to anterior scapular tilting.

Palpation of tender areas in the upper and lower trapezius and palpation of tender areas in the pectoralis minor and latissimus dorsi can identify areas of pain that cause muscle inflexibility and inhibition or hyperactivity of muscle activation that may need to be treated as part of the clinical problem.

Dynamic examination of scapular motion can be reliably performed by clinical observation of the motion as the arm elevates and descends. This motion requires coordinated, sequenced activation of the muscles to maintain the closed chain mechanism. Failure to maintain this results in increased scapular internal rotation, with consequent medial border prominence [26, 36]. Clinical observation of medial border prominence in symptomatic patients has been correlated with biomechanically determined dyskinesis [37], and this method is clinically reliable enough (sensitivity and positive predictive value between 0.64 and 0.84) to be used as the basis for determination of the presence or absence of dyskinesis [38, 39]. The exam is conducted by having the patients raise the arms in forward flexion to maximum elevation, and then lower them 3–5 times, with a 3–5 pound weight in each hand [40, 41]. Medial border prominence on the symptomatic side is recorded as "yes" (prominence detected) or "no" (prominence not detected).

Scapular stabilizer strength may be clinically estimated by several methods. Scapular pinch estimates scapular retraction ability. The scapulae should be retracted and held in an isometric manner for 10 s. Weak muscles will exhibit spasm within that time span. Wall push-ups estimate serratus anterior strength, especially if done in a "plus" (hyper protraction) position. Type II dyskinesis will be exhibited as the muscles fatigue. A semidynamic evaluation of composite scapular stabilizer strength is the lateral slide measurement [42–45]. This test evaluates scapular position as a marker of dynamic muscle activity to control the scapula against varying loads. The test measures side-to-side differences between a point on the spine and the inferior medial scapular tip in three positions of increasing load on the muscles. Position 1 is with both arms at rest at the side. Position 2 is with the hands on the hips, with neutral extension. Position 3 is with the arms abducted in the scapular plane just below 90°, with maximum internal rotation. Side-to-side differences of greater than 1.5 cm suggest dynamic scapular stabilization is lacking. This test can also be used to monitor rehabilitation progression with side-to-side differences diminishing below 1.5 cm as scapular stabilization improves.

The scapular assistance test (SAT) and scapular retraction or reposition test (SRT) are corrective maneuvers that may alter the injury symptoms and provide information about the role of scapular dyskinesis in the total picture of dysfunction that accompanies shoulder injury and needs to be restored [18, 20, 46, 47]. The SAT helps evaluate scapular contributions to impingement and rotator cuff strength, and the SRT evaluates contributions to rotator cuff strength and labral symptoms. In the SAT, the examiner applies gentle pressure to assist scapular upward rotation and posterior tilt as the patient elevates the arm [46, 47]. A positive result occurs when the painful arc of impingement symptoms is relieved and the arc of motion is increased. In the SRT, the examiner grades the supraspinatus muscle strength following standard manual muscle testing procedures [18, 20]. The clinician then places and stabilizes the scapula in a retracted

position. A positive test occurs when the demonstrated supraspinatus strength is increased, or the symptoms of internal impingement in the labral injury are relieved in the retracted position. In the MDI patient, the SRT will eliminate the position of protraction and inferior tilt and will facilitate normal GH kinematics which diminishes the feeling of instability. Although these tests are not capable of diagnosing a specific form of shoulder pathology, a positive SAT or SRT shows that scapular dyskinesis is directly involved in producing the symptoms and indicates the need for inclusion of early scapular rehabilitation exercises to improve scapular control.

Nonoperative Treatment Options

Rehabilitation of the Scapula in Glenohumeral Instability

Scapular rehabilitation may be used in preoperative, nonoperative, and postoperative contexts [48, 49]. Preoperative scapular rehabilitation is directed toward reestablishing kinetic chain activation patterns to maximize scapular stabilizer activation and controlling scapular retraction capability. This is similar to preoperative preparation prior to anterior cruciate ligament surgery. Postoperative scapular rehabilitation may be started very early in the postoperative period. Kinetic chain exercises for trunk and hip strengthening and scapular retraction exercises may be started, while the arm is still in the sling or other postoperative protection. These exercises establish a stable base for more advanced and shoulder-specific exercises. As healing proceeds and as the arm may be moved into abduction and rotation, closed chain axial load and "clock" exercises strengthen scapular stabilizers while minimizing loads on the repair site. When rotator cuff exercises are indicated, integrated scapular stability/humeral head depression exercises reestablish the compressor cuff activation function off a stabilized scapular.

The results of scapular rehabilitation as part of a nonoperative treatment of GH instability are primarily related to the underlying pathology. Patients with posttraumatic instability frequently have ligament and/or bone injury that will not allow ball and socket kinematics. Patients with instability due to microtrauma may be able to regain function by reestablishing the coupled SHR to maximize concavity/compression and ball and socket kinematics. Since MDI is a very muscle-dependent problem, effective scapular control and resulting muscle activation through rehabilitation are frequently successful in resolving symptoms.

Rehabilitation Guidelines

Specific Scapular Rehabilitation Exercises

Rehabilitation exercises for scapular control can be broken down into three groups—proximal kinetic chain exercises to facilitate scapular muscle strength, flexibility exercises to minimize traction on scapular posture, and exercises specific for peri-scapular activation.

Kinetic chain exercises for trunk and hip start from and end at the "ideal position" of hip extension/trunk extension. They include trunk/hip flexion/extension, rotation, and diagonal motions. Progressions include step-up/down and increased weights.

Specific areas to be addressed for flexibility include the anterior coracoid (pectoralis minor and biceps short head), latissimus dorsi, and shoulder rotation. Tightness in these areas increases scapular protraction. Exercises include the open book (Fig. 7.2) and corner stretch (Fig. 7.3) for coracoid muscles, standing shoulder flexion for latissimus dorsi, and sleeper (Fig. 7.4) and cross body stretch (Fig. 7.5) for shoulder rotation.

Peri-scapular strengthening should emphasize achieving a position of scapular retraction, as this is the most effective position to maximize scapular roles. Scapular retraction exercises may be done in a standing position to simulate normal activation sequences and allow kinetic chain sequencing. Scapular pinch and trunk extension/scapular retraction exercises may be started early in rehabilitation to stat the integrated activation.

Several specific exercises have been shown to be very effective to activate the key scapular stabilizers—the lower trapezius and serratus anterior[36]. They are the low row (Fig. 7.6) and inferior

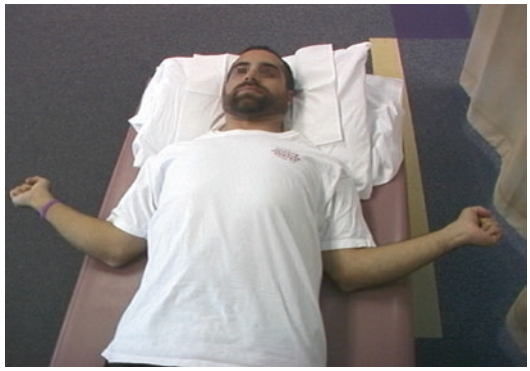


Fig. 7.2 Open book stretch for anterior shoulder tightness

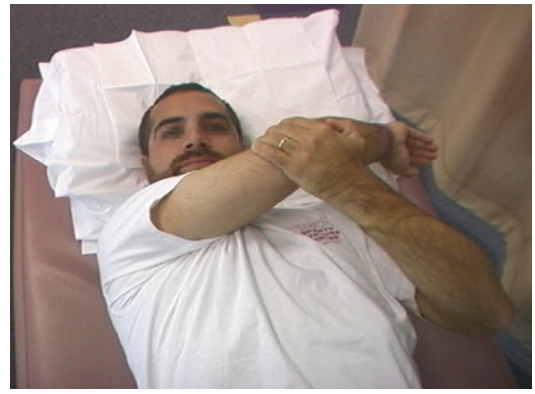


Fig. 7.5 Cross body adduction stretch for posterior shoulder tightness

Fig. 7.3 Corner stretch for anterior shoulder tightness

Fig. 7.6 Low row is an isometric exercise which helps strengthen the lower trapezius and serratus anterior muscles

Fig. 7.4 Sleeper stretch for posterior shoulder tightness

glide (Fig. 7.7), both isometric exercises, and the lawnmower (Fig. 7.8) and robbery (Fig. 7.9). Another effective exercise is fencing (Fig. 7.10).

Closed chain exercises should also be emphasized, to restore the normal activations of the closed chain mechanism. These exercises are characterized by supporting the hand on a stable or movable surface and loading the arm and scapula from distal to proximal. Examples include rhythmic stabilization (Fig. 7.11), scapular clock (Fig. 7.12), and wall washes (Fig. 7.13).

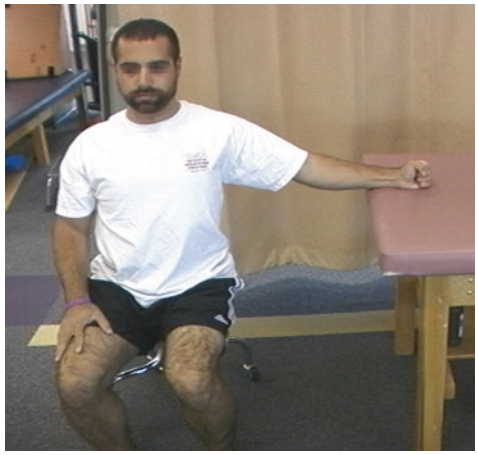


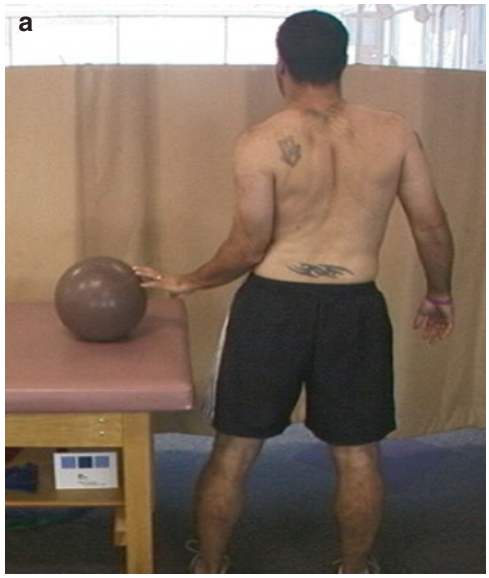
Fig. 7.7 Inferior glide, also used to strengthen the lower trapezius and serratus anterior, is performed by isometrically pushing the arm down into adduction

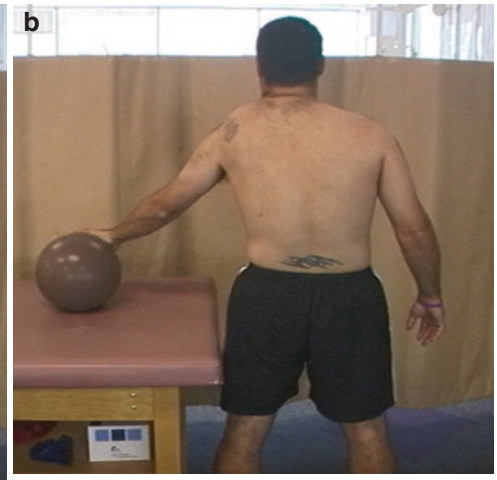
Fig. 7.9 Robbery exercise is performed by "placing the elbows in the back pockets"

Fig. 7.8 (**a**, **b**) Lawnmower exercise utilizes trunk rotation to help facilitate scapular retraction

Fig. 7.10 Fencing is performed by stepping laterally while retracting the scapula against resistance

Fig. 7.11 Rhythmic stabilization





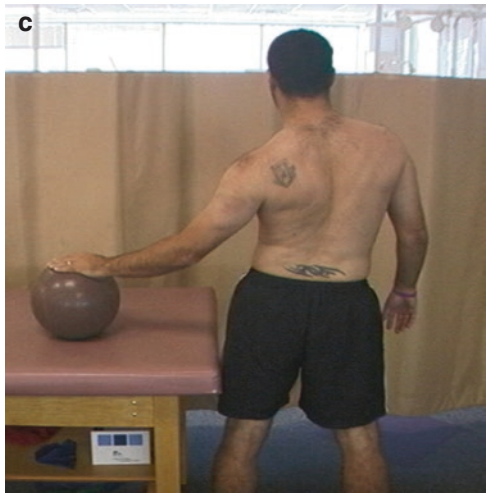


Fig. 7.12 (**a**–**c**) Scapular clock: (**a**) retraction, (**b**) protraction, (**c**) depression

Once scapular control is achieved, integrated scapula/rotator cuff exercises such as punches (Fig. 7.14) and shoulder dumps (Fig. 7.15) that stimulate rotator cuff activation off a stabilized scapula are added. They may be done in various planes of abduction and flexion, with different amounts or types of resistance, and may be modified to be sport specific.

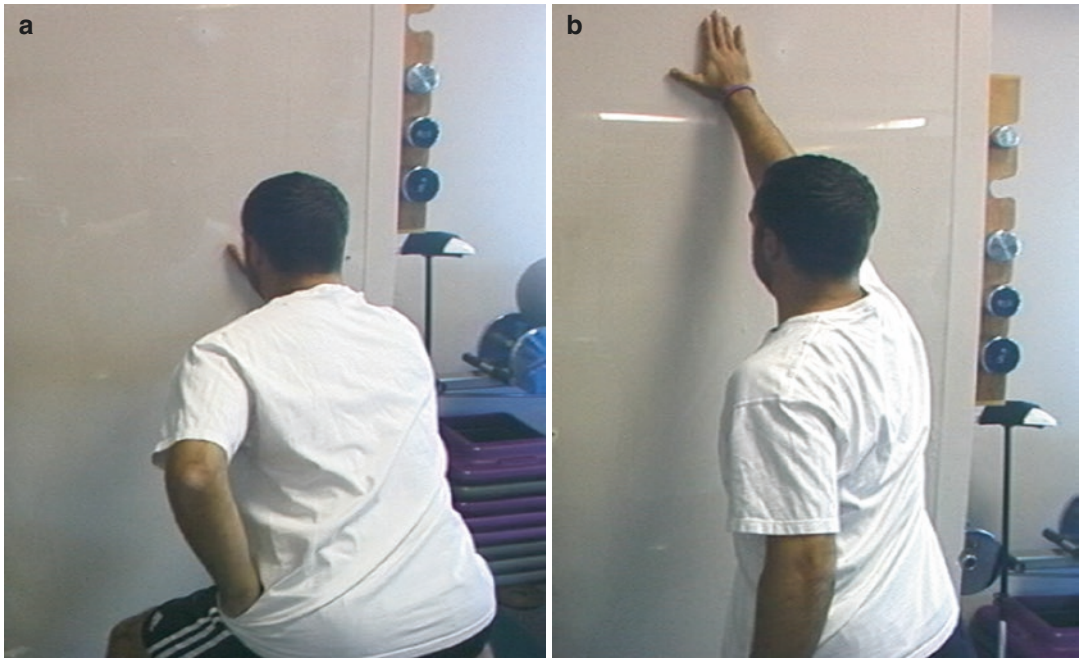


Fig. 7.13 (**a**, **b**) Wall wash is a closed chain exercise which utilizes all kinetic chain segments

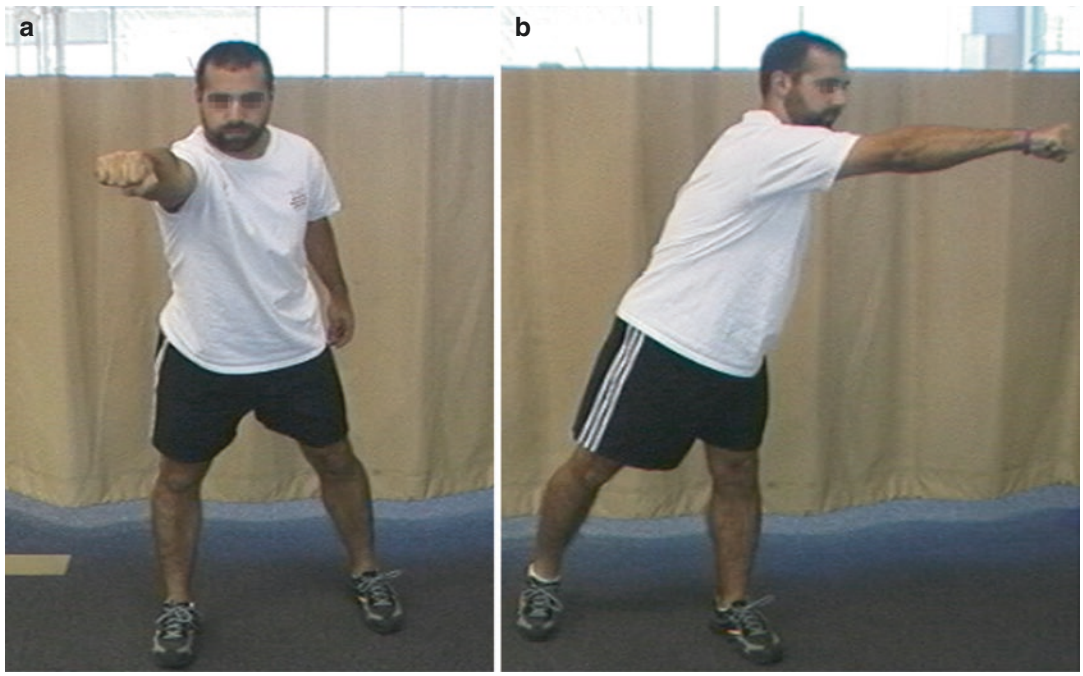


Fig. 7.14 (**a**, **b**) Punches may be performed in multiple planes

Fig. 7.15 Shoulder dump

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The Scapula and Clavicle Fractures

8

Peter W. Hester and W. Ben Kibler

Introduction

Clavicle fractures account for 5% of all adult fractures and 44% of all shoulder fractures [1]. Up to 81% are of the middle third, and half of these are considered "displaced" fractures [2]. Historically, nonoperative treatment with a sling or figure of eight immobilization was considered the preferred standard of care, and this treatment resulted in what were thought to be acceptable functional results [3]. However, more recent studies have suggested that nonoperative treatment may frequently produce less than satisfactory outcomes, with high rates of malunion and nonunion, deficits in muscle strength and endurance, and substantial patient dissatisfaction on patient-reported outcome scores even in patients with established unions that are malpositioned [3–6]. Surgical treatment appears to produce better outcome scores [6], but with its own set of concerns, including the acknowledged risk of surgical complications and the occasional need for a second surgery for hardware removal. These findings have led investigators to emphasize the need for clear identification of: indications for surgery, which clavicle fractures need surgical repair, and what should be the desired outcomes from surgery especially with regard to re-establishing normal scapula position and its functional impact on shoulder motion (Fig. 8.1).

It appears that union of the bone ends is not the sole or even major factor in optimal outcomes of clavicle fractures. Optimal outcomes from clavicle fracture healing depend upon optimum function of the scapula, which requires restoration of the clavicular roles which facilitate normal mechanics in shoulder activity [7–12]. Surgical indications may relate more to addressing the correction of the altered mechanics resulting from the clavicle injury than focusing only on the anatomy. Evaluation of scapular static position and dynamic motion

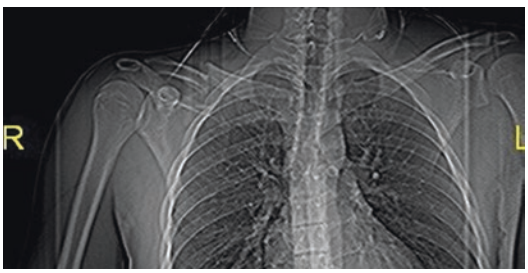

Fig. 8.1 Right clavicle fracture yielding scapula repositioning

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can provide key information relating to the altered mechanics and suggest the need for surgical correction of the anatomy.

Clavicle Anatomy and Mechanics

The clavicle serves as a strut connecting the shoulder girdle to the axial skeleton [9]. Optimal scapulohumeral rhythm and arm function require optimal clavicle anatomy. Its "S"-shaped design allows a wide range of rotation (40–50°) about its long axis, a motion that is key to placing the shoulder and arm in positions for function [13]. In this respect, it is similar to radius function at the wrist. Any loss of the normal curvature of the bone could result in decreased functional ability at the distal joint.

Clavicle length is also an important mechanical factor. Loss of normal proximal (medial) to distal (lateral) length, either by comminution, overriding, or angulation, shortens the strut and, in the presence of an intact acromioclavicular joint, results in scapular internal rotation and anterior tilt, most commonly characterized as scapular protraction [7, 9, 11, 12]. Protraction has been associated with multiple types of pathology such as impingement, rotator cuff tendinopathy, rotator cuff injury, labral injury, and functional muscle weakness [14–21].

Multiple deforming forces can be factors effecting the relative position of the clavicle fracture fragments. Of most concern is the lateral fragment, as this is attached to the scapula. The amount of the initial impacting force can create multiple fracture fragments with capability of displacement, shortening, and angulation. The gravitational force of the weight of the arm will pull the lateral fragment inferiorly and medially around the ellipsoid curvature of the thorax. This position is accentuated by placing the arm in a sling across the front of the body.

Muscle forces will become deforming forces. Medially, the sternocleidomastoid muscle can exert a superior and external rotation force on the proximal fragment. However, the main deforming forces are exerted on the lateral fragment, indirectly through attachments

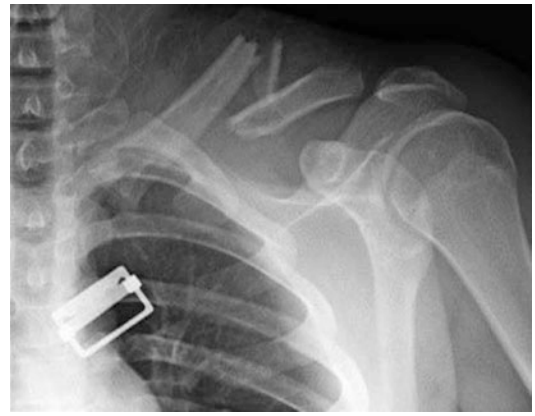

Fig. 8.2 Medial and lateral fragment classic deformation pattern

to the coracoid and humerus. The pectoralis major and minor, the latissimus dorsi, and the anterior deltoid can produce inferior, medial, and internal rotation forces on the lateral fragment. These forces can also produce a position of scapular protraction.

Collectively, these deforming forces frequently produce a position of the lateral fragment that may be overriding the medial fragment but also may be angulated in the anterior/posterior or inferior/superior direction and/or may be anteriorly rotated in relation to the medial fragment (Fig. 8.2). These positions represent a tri-planar or three-dimensional deformity which may not be obvious on two-dimensional radiographs but will be more clearly delineated by a dynamic shoulder examination. Evaluation of the scapula can frequently demonstrate the deformity, since the scapular position will have to conform to the position of the lateral fragment. The presence of scapular protraction, in addition to demonstrating the clavicle deformity, also predicts the functional deficits that may occur if the scapula is maintained in this position by not correcting the clavicle deformity.

Clavicle Fracture and the Scapula

The tri-planar deformation subsequent to clavicle fracture shortening, rotation, and/or angulation yields loss of strut efficiency and may produce

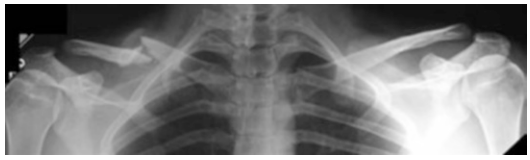

Fig. 8.3 Correlated radiograph with clinical appearance in malunion with limited functional result

dyskinetic patterns for simple activities of daily living as well as more physically demanding pursuits. There is limited, focused anatomic lab work detailing and correlating the deficiencies of clavicle malunion [22]. Malunion is associated with strength loss, rapid fatigue, pain, and limb and shoulder girdle paresthesia (Fig. 8.3). As high as 70% of nonoperatively treated mid-shaft clavicle fractures developed clinically evident scapular dyskinesis [12].

Shields et al. provided the first study to report rates of scapulothoracic dyskinesis following mid-shaft clavicle fractures and showed that SICK scapula scores were worse in these patients with ST dyskinesis [12]. In this retrospective cohort design including 24 patients, the operative group had only 1 of 12 (8%) patients demonstrate ST dyskinesis compared to 8 out of 12 (67%) in the nonoperative group. The nonoperative group reported more pain, decreased strength, and compromised range of motion along with scapula position change.

Ledger et al. reported that shortening of the clavicle changes the shoulder girdle by altering movement constraints with increased upward sternoclavicular angulation by 10° and increased protraction by 6°, which then yields diminished strength of at least 10% in extension, adduction, and internal rotation [22].

Shortening of the clavicle results not just in a reduced moment arm of the pectoralis major inserting on the clavicle mostly decreasing flexion and abduction strength in higher abduction but with secondary challenges to all musculature as scapula orientation to all soft tissue orientations is altered. Veeger and van der Helm described this positional and moment arm alteration changing muscle balance relationships [23]. This concept of maladapted tendons losing mechanical advantage is supported by Jupiter [24].

A simulated clavicle fracture model cadaveric study by Hillen et al. that placed cluster markers on the clavicle, sternum, humerus, and scapula provided rare anatomic insight into scapula positioning in this population [8]. The study performed manual motion trials on intact, resected, and plate-fixed clavicles and demonstrated that in the specimen with the 3.6 cm shortened clavicle, the scapula with the arm at 30° abduction was 20° more protracted, 12° more laterally rotated with 7° decreased posterior tilt, and more retracted in the sternoclavicular joint an average of 1.2° per 1.2 cm of shortening.

In the controlled shortening study, the AC joint was unaffected due to the stabilizing effect of the coracoclavicular ligaments; however, increased movement and rotation occurred at the sternoclavicular joint with implication of arthrosis risk.

Kibler and Sciascia described how diminished tilt and increased lateral rotation alter the acromion position supporting the concept of subsequent impingement and limited rotator cuff function as the anterolateral part of the acromion assumes a more anterior and more lateral position [9].

Andermahr et al. offer that the altered scapula position means that the glenoid orientation is changed as well such that the glenohumeral contact force direction is also impacted with the inference being to subject the labrum and capsule to shear forces not normally anticipated [7]. Veeger and van der Helm supported this concept as rotator cuff stabilizing forces may be altered and potentially yield a higher glenohumeral joint contact force with increased capsulolabral shear [23].

The longer the delay to surgery, the greater the scapular malposition with less than 6 weeks better and more than 40 weeks worse [25, 26].

Acute ORIF with a mean of 0.6 months was preferred to delayed with mean of 63 months [27]. Shoulder flexion endurance decreased in the delayed group, and constant scores were better in the acute group.

Radiograph Interpretation

Plain radiographs are utilized to make the diagnosis of a clavicle fracture and provide information regarding comminution and overriding. However, as two-dimensional tools, they are frequently not able to accurately demonstrate the tri-planar deformity of the fracture or accurately assess scapular position. Scrutiny of radiographs may demonstrate subtle architectural alterations which may suggest scapula malpositioning and resulting dyskinesis and shoulder functional compromise. Be certain to compare like images, at similar trajectory, when making the decision for conservative or surgical care.

Figure 8.4 and 8.5 demonstrate the potential of increased fracture displacement over one week along with altered acromial projection and also suggests the need to compare like radiographs. Also notice the acromion orientation inconsistency which may also be valuable in understanding the degree of rotation and angulation of the lateral fragment (Fig. 8.5). Advanced imaging such as CT scans may be helpful in identifying the severity of the fracture (Fig. 8.6a, b).

With standardized radiographs, one may be able to observe and actually measure a change in scapular position as a result of surgical correction of the clavicle deformity. Pre and post surgical measurement of the apparent medial to lateral scapular width may demonstrate increased apparent width as the scapular protraction is decreased (Fig. 8.7a, b). Also, the amount of inferior scapular displacement may be measured as the distance between the inferior border of the medial fragment and the scapular spine. This "scap gap" will

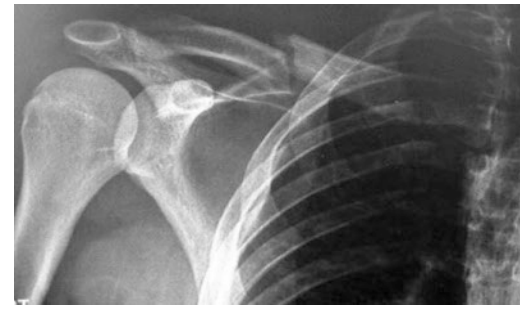

Fig. 8.4 Initial AP radiograph

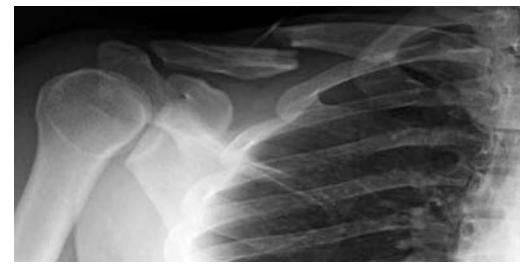

Fig. 8.5 One week follow-up X-ray for fracture seen in Fig. 8.4. A 15 degree angled view demonstrates a marked difference in displacement

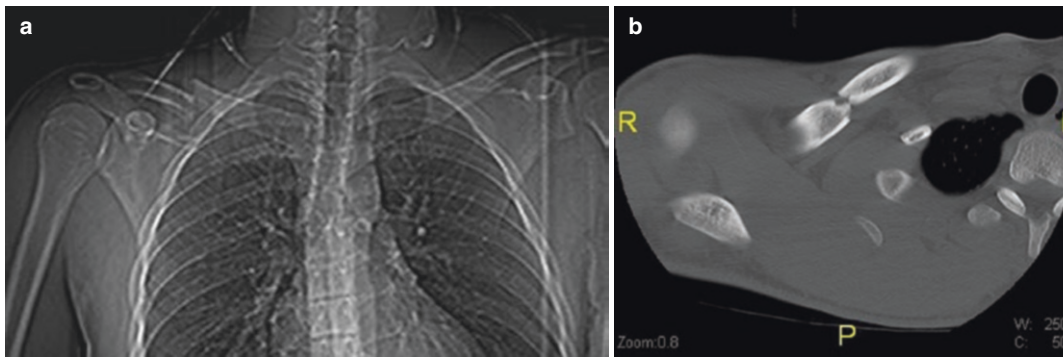

Fig. 8.6 (**a**, **b**) CT demonstrates the fracture malrotation in same patient in Figs. 8.4 and 8.5

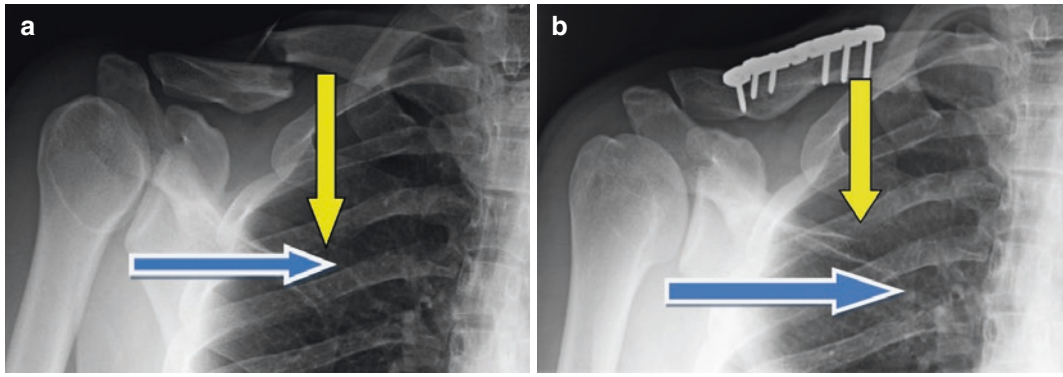

Fig. 8.7 (**a**, **b**) With fracture, a resulting protracted scapula, may yield a relatively narrower scapula width on AP radiographs compared to postreduction *blue arrows* →, as well as a greater medial fragment inferior surface to scapula spine distance →. This area has been termed "scap gap" by the authors

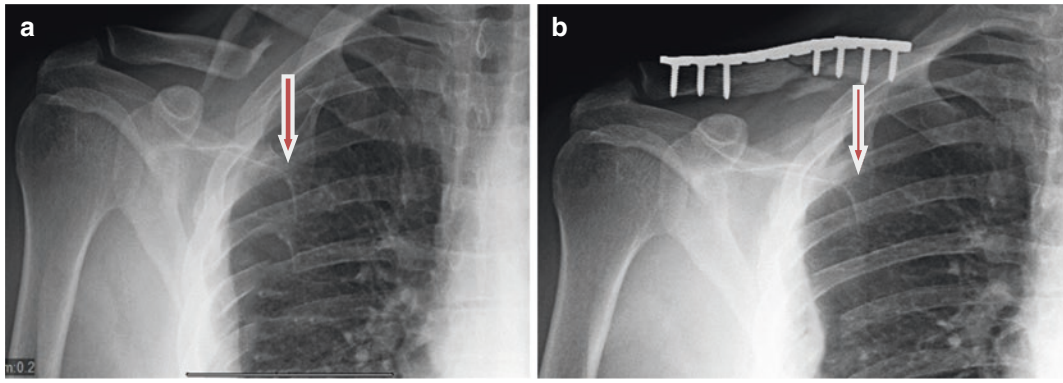

Fig. 8.8 (**a**, **b**) "Scap gap" reduced with fixation

be less after the surgical correction, indicating normalization of the scapular position relative to the clavicle (Fig. 8.8a, b).

Clinical Evaluation

Every clavicle fracture should be evaluated for the possibility of lateral fragment malrotation and scapular dyskinesis. There may be too much pain or swelling to accurately evaluate early after the injury, but by ten to twenty-one days, an accurate evaluation can be performed. Bilateral comparison of shoulder posture can demonstrate drooping of the arm and protraction of the scapula. Observation of the posterior shoulder will demonstrate the position of scapular dyskinesis. By 3 weeks, there is frequently enough callus formation at the fracture site to allow arm movement. Manual stabilization in retraction of the dyskinetic protracted scapula, which reverses the anterior rotation of the lateral fragment, will frequently result in decreased arm pain, increased arm motion, and

increased strength. These findings can be used in counseling patients regarding treatment.

In established nonunions and malunions with continued shoulder symptoms, the principles of the standard scapular evaluation should be carried out as part of the comprehensive shoulder evaluation [28]. Emphasis should be placed on establishment of the presence or absence of dyskinesis and the effect of the scapular assistance and scapular retraction test corrective maneuvers on symptoms. These findings should be used as part of the information in counseling patients regarding treatment.

Treatment Guidelines

All closed clavicle fractures may be initially treated with sling immobilizations. Reevaluation around 10 days to 3 weeks including radiographic and clinical evaluations will allow accurate assessment of clavicle fracture position and scapular position. Patients with no or minimal scapular dyskinesis at this time will usually do well with appropriate nonoperative treatment. Patients with established or increasing clavicle fracture deformity and/or scapular dyskinesis should be counseled about the potential mechanical deficits associated with these radiographic and clinical findings, and the potential benefits demonstrated by the research which has looked at the results and outcome scores of surgical correction.

Goals of Operative Fixation of Clavicle Fracture, Malunion, and Nonunion

Reduction and fixation aims to restore scapula position and parascapular muscle and bone orientation and balance as initially designed to best optimize shoulder motion, strength and endurance. Early reduction and fixation of acute fractures will better assure proper union and reduce the risk of muscle interposition between the fracture ends. This early fixation will also allow for the restoration of optimal range of motion, strength, and endurance (Fig. 8.9a, b). One hundred percent displacement and greater than 15 mm of shortening are strong indications for fixation. Concurrent humeral shaft fracture serves as a strong indication to perform open reduction and internal fixation (ORIF).

ORIF with plate and screw systems offers superior results and control with less complication risk and need for removal than with intramedullary subcutaneous single screw fixation. Appreciating anatomic differences of the clavicle, having two different clavicle plate systems available in the operating room, has been helpful to assure a best contour option.

Postoperatively, the patient is kept in a sling for 3 weeks while maintaining good posture with medial scapula border muscle activation program. Rehabilitation is permitted at the 3-week mark.

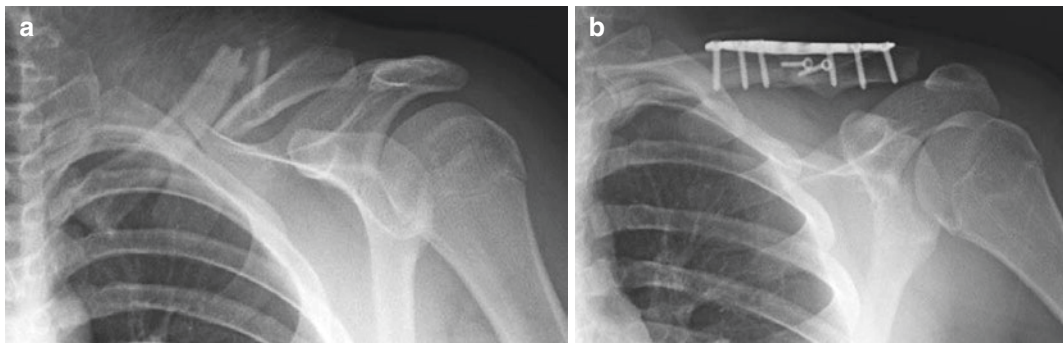

Fig. 8.9 (**a**, **b**) These AP images allow for appreciation of acromial position change with fixation

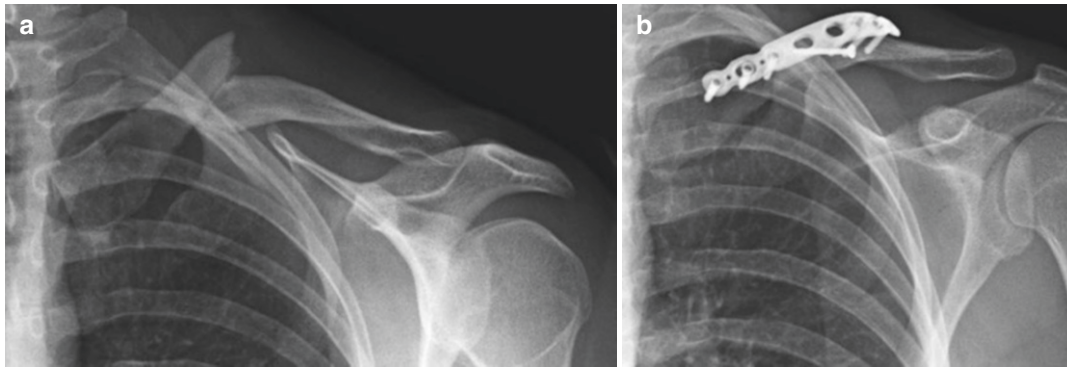

Fig. 8.10 (**a**, **b**) Prior clavicle malunion re-fracture in a professional tree climber along with concurrent humeral shaft fracture treated with ORIF of both fractures allowed for normalized scapula function and return to activity at three months post-operatively

Radiographs should be evaluated at 1, 2, and 3 months postoperatively. Most patients are discharged to regular activity between months 3 and 4.

Malunions often provide the best evidence of the need to consider early ORIF. Simple functional decline and ADL compromise following nonoperative treatment along with increased complexity of surgery must always be considered and addressed with each patient should they consider a conservative plan initially (Fig. 8.10a, b).

Early decision, within the first 2–3 weeks, to perform open reduction and internal fixation is preferred to minimize the challenging issues of callous, adhesions, and muscle shortening derotational forces.

Summary

Clavicle fractures may frequently develop a triplanar three-dimensional deformity in which the lateral fragment may be malrotated, angulated, and shortened relative to the medial fragment. The scapula, attached to the lateral fragment, may move to a dyskinetic position of protraction that can be clinically observed. The dyskinetic position can provide key information regarding the lateral fragment malposition and suggest deleterious functional effects of the protracted scapular position. Surgical indications for ORIF of the clavicle fracture can be better delineated by including knowledge of the presence or absence of scapular malposition in counseling patients regarding treatment.

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The Scapula and Acromioclavicular Joint Separation and Arthritis

9

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Acromioclavicular Joint Anatomy

The acromioclavicular (AC) joint serves many key roles in shoulder function. The AC joint is an important component of the screw axis mechanism that imparts normal shoulder motion [1, 2]. The AC joint stabilizes the clavicular strut and permits anterior and posterior translation, superior and inferior translation, and rotation.

The AC ligaments control most of the anterior and posterior translation of the AC joint with some contributions by the coracoclavicular ligaments. The AC ligaments, especially posterior and superior AC ligaments, confer horizontal stability but also play a role in vertical and rotational stability [3]. The superior AC ligament (56%) and posterior AC ligament (25%) contribute to resistance of posterior translation [4], and the anatomical position of the superior AC ligament also suggests a role as a tension band helping control lateral tilt of the acromion. The conoid with its more medial and posterior insertion on the clavicle contributes to 60% of vertical stability [4], while the trapezoid with its more lateral and anterior insertion on the clavicle contributes to vertical and rotational stability at the AC joint.

Disruption of the Acromioclavicular Joint

Injuries to the AC joint have typically been thought of and categorized in a two-dimensional (2D) fashion. The Rockwood classification [5] has been widely used to classify AC joint injuries and is based purely on radiographic classification. The Rockwood classification assesses the AC joint on anteroposterior (AP) and axillary radiographs assessing superior and posterior translation of the clavicle relative to the acromion. Unfortunately, the Rockwood classification has shown poor reliability for classification and treatment decisions with plain radiographs, and the addition of 3D CT has not been shown to improve the reliability of classification and treatment [6].

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AC joint injuries should be thought of in a three-dimensional (3D) fashion with consideration of the role of the scapula in the injury pattern. Disruption of the AC joint can alter normal scapular motion. The clavicle serves a key role as a mobile strut for scapular and arm motion. The AC joint serves as a unifying link in the screw axis mechanism that governs normal scapulohumeral rhythm [1, 2, 7, 8], and the three-dimensional nature of the normal kinematics of the clavicle and scapula facilitate arm function. These studies demonstrate that as the arm rotates and elevates, the clavicle acts as a mobile strut based on the sternoclavicular joint and elevates, protracts, retracts, and rotates along its axis. The AC joint acts as a stable but slightly mobile connecting link. Finally, the scapula, acting as a mobile but stable base for the humerus and arm, upwardly rotates, posteriorly tilts, externally rotates with arm motion, and translates upward/downward and medially/laterally [2, 7–9]. Loss of the stabilization by ligament disruption allows the scapula to have a "3rd translation," inferior and medial to the clavicle. Many of the clinical deficits creating symptoms and limitations in patients with symptomatic AC separations can be associated with alteration of scapular position and motion. The prominence of the clavicle is mainly due to the 3rd translation. Decreased shoulder range of motion and demonstrated abduction and flexion strength are due to excessive scapular protraction [10–13]. Evaluation of the symptomatic patient should include determination of scapular static position and dynamic motion as part of the diagnosis that guides treatment decisions [14].

Clinical Diagnosis of Acromioclavicular Joint Disruption

Clinical examination can identify the three-dimensional sequelae of AC joint disruption and help guide treatment decisions instead of relying on a purely radiographic and 2D classification for AC joint injuries. Scapular dyskinesis can be identified on clinical examination with accuracy and predictability and can be seen in patients with AC joint injuries [1]. This resulting shoulder dysfunction with scapular dyskinesis can include decreased shoulder motion and strength [1]. We consider patients with AC joint inferior/superior and anterior/posterior laxity on manual testing and the presence of scapular dyskinesis to have "high-grade" injuries. We consider patients with these same clinical findings, except the absence of scapular dyskinesis, to have "low-grade" injuries. All patients with AC joint injuries in our center are systematically treated with scapular rehabilitation prior to operative discussion. Typically the "low-grade" injuries can be treated nonsurgically, while the "high-grade" injuries may require surgery due to the biomechanical disruption and loss of function.

Furthermore, a new modification for type III injuries has recently been proposed to subclassify Rockwood type III AC joint injuries into IIIA and IIIB with recognition of the value of physical examination findings and the role of the scapula. Type IIIA injuries have a stable AC joint without overriding of the clavicle on the cross-body adduction view and without significant scapular dysfunction. Unstable type IIIB injuries have therapy-resistant scapular dysfunction and an overriding clavicle on the cross-body adduction view [14].

Operative Treatment of Unstable Acromioclavicular Joint Injuries: Classification of Operative Types

Both nonanatomic and anatomic reconstruction techniques have been advocated. Nonanatomic techniques include transfers of the coracoacromial (CA) ligament. Cadenat first described transfer of the CA ligament in 1917 utilizing the posterior fascicle on the acromial side and suturing it to the remnants of the conoid ligament and periosteum of the posterior superior clavicle to attempt to recreate the coracoclavicular (CC) ligaments (conoid and trapezoid) [4, 15]. The Weaver-Dunn technique involves a similar nonanatomic transfer of the CA ligament from the acromial side, and many

modifications of the technique have emerged. Unfortunately, transfer of the CA ligament does not recreate the anatomy of the CC ligaments and provides only 25% of the strength of the intact CC complex and does not repair the AC complex [16].

The AC joint is critical for glenohumeral and scapulothoracic function [3]. Proper restoration of the AC and CC ligaments is necessary to completely stabilize the scapula and recreate translation and rotation that optimizes function. Concurrent AC and CC fixation is not a new concept, initially reported by Baum in 1886; however, most of the reported techniques in the literature currently do not involve anatomic AC and CC reconstruction [17]. A recent systematic review indicated that only 13 anatomic techniques (8.0%) were described out of the 162 surgical techniques in 120 different articles [17].

Techniques have emerged that emphasize anatomic reconstruction of the CC ligaments, but the described techniques typically involve distal clavicle resection and are commonly performed without formal repair or reconstruction of the damaged AC ligaments. More recent information suggests that both anatomic AC and CC ligament reconstruction can help restore translational and rotational stability [3] as CA ligament transfer with augmentation does not address anterior-posterior translation of the scapula on the clavicle [18].

Three-dimensional (3D) restoration with anatomic reconstruction of both the CC ligaments and the AC ligaments offers an advantage over CC ligament reconstruction alone due to the ability to restore all three components of the AC joint—horizontal, vertical, and rotational stability as a stable link for scapular motion and scapulohumeral rhythm. Recent publications have taken note of AC joint malreduction and instability following CC ligament reconstruction [19, 20]. The clavicle and acromion appear to be "reduced" on a static anteroposterior (AP) radiograph; however, the bones are malreduced on an axillary or cross-body adduction radiograph, or the AC joint is unstable on examination.

There is often the question of what to do with the distal clavicle during AC joint reconstruction cases with some surgeons opting to resect the distal clavicle versus others opting to preserve the distal clavicle. Treatment of the distal clavicle in the setting of AC joint injuries has varied over time. Historically, operative techniques involved retention of the distal clavicle and rigid AC joint fixation without reconstruction of the CC ligaments. These early techniques were fraught with continued pain and the development of AC joint arthritis [21, 22]. Eventually, isolated distal clavicle resection was proposed to address AC joint injuries, but this technique was fraught with poor results since it did not restore AC joint stability [4]. Clavicle shortening or AC joint instability may contribute to pathologic positioning of the scapula in a protracted and internally rotated position [3]. Our technique involves retention of the distal clavicle as proposed by others [4] to help restore anatomic stability and preserve optimal clavicle strut function and scapular mobility.

The Authors' Surgical Technique for Operative Treatment of Unstable Acromioclavicular Joint Injuries

The AC reconstruction in this study follows the principles established by Carofino and Mazzocca [4, 18, 23]. Modifications of the technique regarding graft passage and graft and ligament attachment were developed to address potential weakness of the described technique. The technique is completely described in a recent publication [24].

The patient is placed in a slightly modified beach chair position. The surgical incision is placed along the anterior superior border of the clavicle from the midportion of the clavicle to the AC joint and across to the lateral edge of the acromion. The dissection is started medially, with reflection of the trapezius fascia by electrocautery. The dissection is carried out longitudinally over the distal clavicle to the acromion, with care taken to stay right on the clavicle in the dissection so that the native anterior and posterior AC ligaments, which are frequently still attached to the acromion, can be identified and mobilized

to be used in the repair (Fig. 9.1). These ligaments are frequently found scarred to the inferior half of the clavicle, and their mobilization facilitates joint reduction.

A method of "docking" the allograft to the acromion has been found to be effective as a sturdy construct. Two 2.4 mm drill holes are made from the lateral acromial edge to the superior acromial edge at the joint (Fig. 9.2a). The acromial edge is lightly debrided to provide an attachment site and to stimulate healing, and a #2 PDS suture is passed as a loop suture passer (Fig. 9.2b) to be employed later.

The CC interval can then be visualized through a deltoid splitting incision. Careful dissection around the coracoid frees up the scar and creates

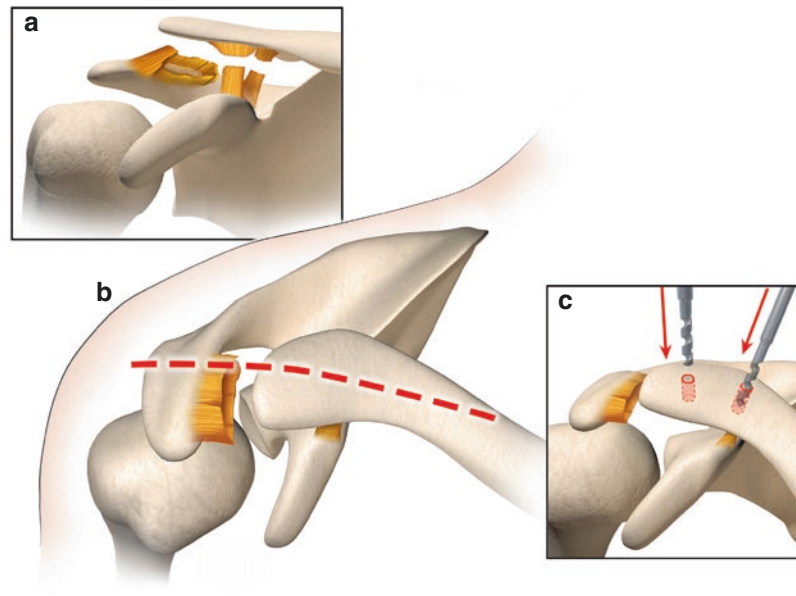

Fig. 9.1 (**a**) High-grade acromioclavicular and coracoclavicular ligament injury with avulsion of the acromioclavicular ligaments from the clavicle and midsubstance injury of the coracoclavicular ligaments. (**b**) An incision (*dotted line*) is made from the lateral acromion to the midshaft of the clavicle. (**c**) Drill holes are made through the clavicle in line with the native attachment of the conoid and trapezoid ligaments

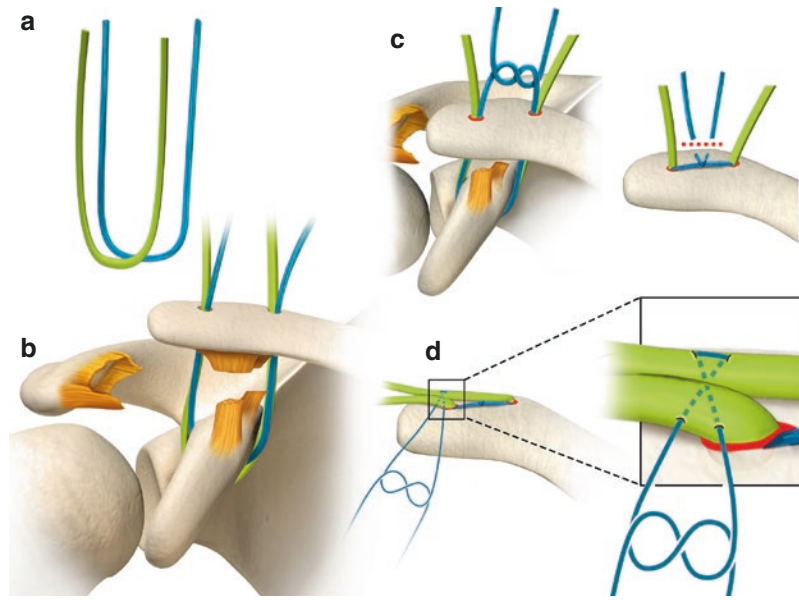

Fig. 9.2 (**a**) The reconstruction includes an allograft semitendinosus tendon (*green*) and 5 strands of No. 2 PDS (*blue*).
(**b**) The sutures and the graft are passed through the anatomically placed clavicle drill holes.
(**c**) The PDS sutures are tied to add stability to the healing graft (excess suture is removed as shown by the *red dotted line*). (**d**) The graft tails are secured together using the stitch configuration shown

a tunnel for graft passage. Passage of the instrument from the medial to lateral side and shuttling the graft from the lateral to medial side minimizes the risks to the underlying neurovascular structures.

The CC reconstruction construct consists of a semitendinosus allograft (6.0 mm × at least 260 mm) and 5 #2 PDS sutures to be used as an internal splint. Each of the graft ends is prepared with a baseball type stitch running about 25 mm on each end. This construct is passed around the undersurface of the coracoid and is passed through anatomically positioned 4.5 mm clavicular drill holes. The conoid drill hole is placed from the posterior superior edge of the clavicle, aimed at the conoid tubercle, a readily palpable landmark on the undersurface which is present directly superior to the medial edge of the coracoid. The trapezoid drill hole is placed about 1 cm anterior and 1.5–2.0 cm lateral to the conoid hole, depending on patient size, and is aimed at the trapezoid ridge on the undersurface at about a 30° angle to the vertical from the lateral coracoid edge (Fig. 9.3). Both limbs of the graft construct are then passed, and the joint is manually reduced by bringing the acromion to the clavicle. The sutures are tied down over the clavicle to provide initial stability for the CC reconstruction. The graft limbs are tensioned and then sutured together over the clavicle with multiple nonabsorbable sutures. Stability of the CC reconstruction can be checked by demonstration of elimination of inferior/superior laxity.

The AC ligament reconstruction includes superior, anterior, and posterior components. The graft tails can be used to reconstruct the superior AC ligaments, and the native tissues can be used to repair the anterior and posterior ligaments. Two biocompatible anchors (PushLock, Arthrex, Naples, FL) double loaded with #1 nonabsorbable suture are placed into the anterosuperior and posterosuperior clavicle, and the sutures are passed through the mobilized native AC ligament tissues, but not tied. The allograft tails are brought to the acromial edge; the correct length to ensure graft tension and attachment to the acromion is determined, and a passing suture of #1 nonabsorbable suture is placed (Fig. 9.4). These sutures are then passed through the previously placed acromial drill holes and tied over the lateral acromion, attaching the graft tails to reconstruct the superior AC ligament. The

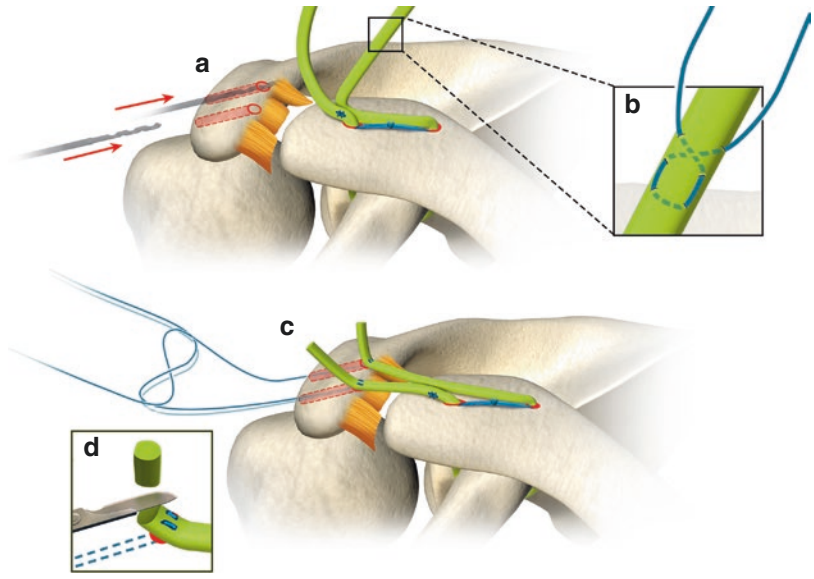

Fig. 9.3 (**a**) Drill holes are placed through the lateral acromion to dock the graft tails into the bone. Sutures are placed into each graft tail (**b**) to assist in properly docking the graft into the previously prepared acromial edge (**c**). (**d**) The excess graft tail tissue is removed

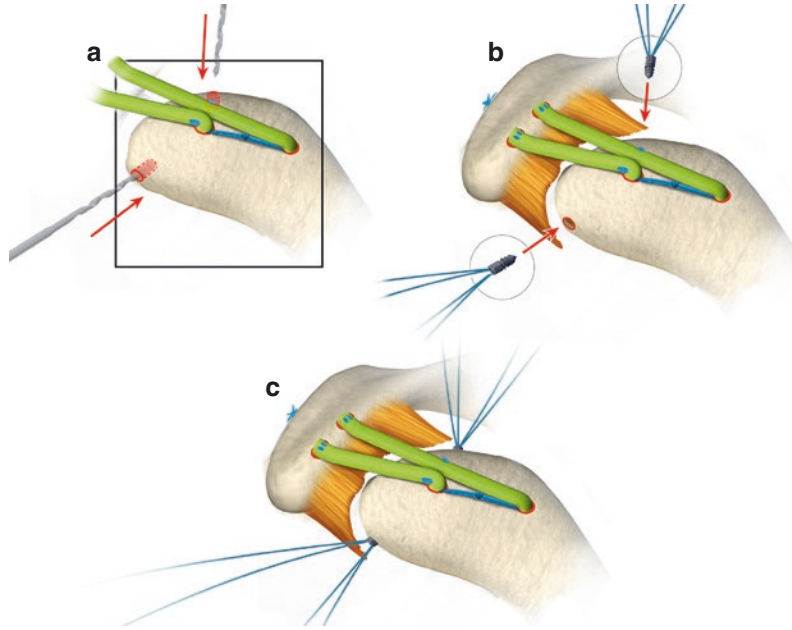

Fig. 9.4 (**a**) Drill holes are placed into the anterosuperior and posterosuperior aspects of the clavicle. (**b**) Suture anchors, which will be used to repair the native acromioclavicular ligaments, are placed into the holes. (**c**) Sutures are passed but not tied

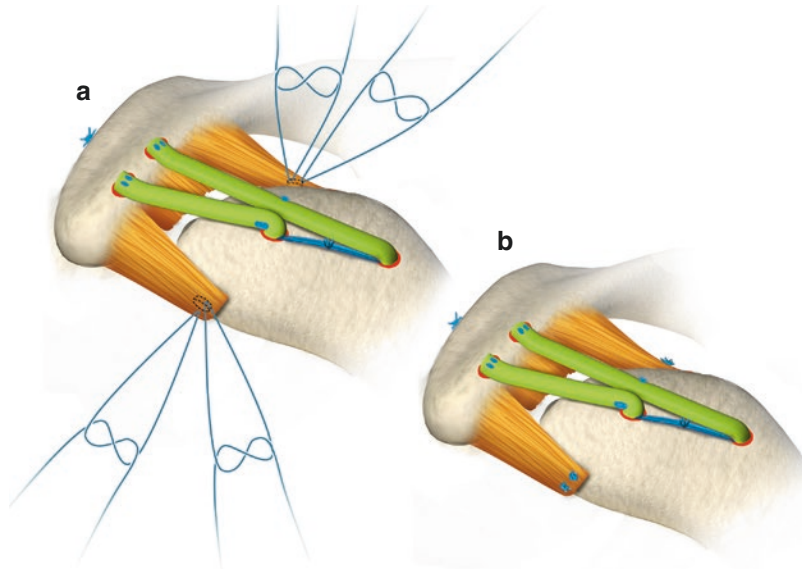

Fig. 9.5 The native anterior and posterior ligaments are repaired (**a**) and tied down to the clavicle (**b**)

anterior and posterior native tissues are then tied down, completing the repair (Fig. 9.5). The stability of the entire construct is then assessed to demonstrate elimination of both inferior/superior and anterior/posterior laxity. The deltoid split is closed, the wound is closed in layers, and a sling and swathe is applied.

Postoperative Rehabilitation After Acromioclavicular Reconstruction

Postoperatively, all patients are placed in a sling and swathe for 4 weeks. Internal rotation and abduction is not allowed for 3 weeks, and active

forward flexion is not allowed for 6 weeks. During the first 3 weeks, the patient is allowed to perform active scapular retraction and depression. All patients are referred to formal physical therapy following the third postoperative week and are provided with a standardized closed kinetic chain protocol designed to minimize shear forces at the glenohumeral joint and to increase proprioceptive feedback through the shoulder and scapula. Scapular mobility and stability are emphasized through the scapular rehabilitation protocols discussed in the rehabilitation chapters of this textbook.

Outcomes After Anatomic Acromioclavicular Reconstruction

There have been many reports on anatomic CC ligament reconstruction but very few reports on anatomic reconstruction of both AC and CC ligaments. Carofino and Mazzocca described anatomic AC and CC reconstruction using semitendinosus graft looped around the coracoid, interference screw fixation into the clavicle, and the remaining limb of the graft is used to reconstruct the posterior and superior AC ligaments [4]. The technique article presented a case series of 17 patients with minimum follow-up of 6 months and average follow-up of 21 months. Significant preoperative to postoperative shoulder function scores were noted for the American Shoulder and Elbow Surgeons Score (ASES), the Simple Shoulder Test (SST), and the Constant score. Three of 17 (17.6%) patients were reported as failures.

Our study population included 23 patients. Fifteen patients (age = 42 ± 18 years; 10 males, 5 females) with 16 injuries requested surgical treatment. One patient had bilateral reconstructions. All had "high-grade" injuries. Five patients were found to have "low-grade" injuries and none requested surgery, while three patients who were evaluated as "high-grade" injuries did not request surgery. This reinforces the idea that scapular dyskinesis, as an indicator of compromised AC function, can be beneficial in helping to determine surgical indications. Average follow-up of the surgical cases was 3 ± 1.5 years (range 1.5–5 years). All 15 patients and 16 shoulders had anterior/posterior laxity on initial clinical exam with 64% of the patients having concurrent superior/inferior laxity. The patients without superior/inferior laxity had acromions that were completely displaced under the clavicles, with fixed dislocation. Four patients had previous AC joint reconstruction surgery including CC ligament reconstruction only (2), hook plate insertion (1), and CC ligament reconstruction with distal clavicle excision (1). Of the remaining 12, 8 were reconstructed within the first 3 months after injury, while 4 were treated from 4 months to 6 years after injury. Postoperatively, there was one loss of anatomic reduction, demonstrated by loss of anterior/posterior stability, which was secondary to distal clavicle osteolysis and loss of AC ligament attachment following a fall. All other patients exhibited dynamically stable anterior-posterior and inferior-superior stability on clinical examination and symmetrical scapular motion at most recent follow-up. X-ray determination of static stability demonstrated CC distances that averaged 1 cm (range 0.59–1.31 cm) at the time of discharge. The 1.31 cm distance was in the patient with loss of AC reduction after a fall. The patients demonstrated significant improvement ($p < .001$) in the preoperative (51, range 11–98) to final (13, range 0–43) DASH scores with an average change in DASH score of 38 ± 27 points. There were no complications relating to the surgery. There were no infections and no reoperations for loss of reduction or removal of sutures or implants. These outcomes were similar to Carofino and Mazzocca's [4].

Conclusions

Anatomic AC joint reconstruction with anatomic AC and CC reconstruction can restore AC joint anatomy and scapular mechanics, achieving excellent outcomes. We have attempted to combine clinical experience with a 3D understanding of AC joint function to better grade and treat AC joint injuries. The presence or absence of scapular dyskinesis is not considered to be an absolute indication or

contraindication for surgery, but from our data it can be used as a marker of impaired scapulohumeral rhythm and shoulder dysfunction and does appear to be a consistent and a valuable piece of information to use in determining indications for surgery.

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The Scapular and Shoulder Arthritis

10

Brent J. Morris, T. Bradley Edwards,
and Thomas W. Wright

Introduction

Altered scapular motion or "scapular dyskinesis" is considered an alteration in the normal resting scapular position or impairment of scapular motion [1]. Major progress has occurred in our understanding of scapular dyskinesis as observational findings are now supported with scientific data. The concept of scapular dyskinesis is now understood and identified around the world, and the implications are broad. Our understanding of the scapula now extends far beyond our rudimentary basis of "scapular winging" and neurologic conditions. We can now begin to explore the impact of the scapula on other conditions, including shoulder arthritis, and attempt to explore a more cause and effect relationship.

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The scapula establishes a platform for effective rotator cuff function and shoulder motion. Scapular dyskinesis is considered to be an impairment of normal scapulohumeral rhythm with the potential to impact shoulder function. Scapular dyskinesis has been reported in up to 67–100% of patients with shoulder injuries [2–4]. Scapular dyskinesis has been associated with multiple shoulder pathologies including impingement, instability and anterior capsular laxity, labral injury, rotator cuff weakness, clavicle fractures, and acromioclavicular joint injuries among others [5–10].

Despite the growing body of evidence related to scapular dyskinesis and multiple shoulder pathologies, there remains limited information regarding the association of scapular dyskinesis and shoulder osteoarthritis. We will review scapular dyskinesis and shoulder osteoarthritis and discuss potential implications.

Scapular Dyskinesis and Primary Glenohumeral Joint Arthritis: Onset, Prevention, and Treatment of Early Posterior Subluxation and Posterior Glenoid Erosion

The prevalence of scapular dyskinesis and primary glenohumeral joint osteoarthritis has not been established. Glenohumeral joint osteoarthritis clearly involves internal joint derangement

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and limitations in glenohumeral joint motion, but it is unclear how scapulohumeral and scapulothoracic motion is affected in this patient population. Furthermore, it is unclear if scapular dyskinesis resolves following anatomic total shoulder arthroplasty.

We have empirically identified scapular dyskinesis in this patient population with primary glenohumeral joint osteoarthritis. Although a prospective study is warranted, we want to apply an understanding of scapular principals and primary glenohumeral joint osteoarthritis to allow us to identify potential treatment opportunities to enhance patient outcomes.

Patients with eccentric posterior glenoid erosion are among the most challenging patients to treat with glenohumeral joint osteoarthritis. Posterior subluxation and eccentric posterior glenoid erosion have long been recognized [11]. Walch et al. proposed a classification system based on computed tomography (CT) scans for primary glenohumeral joint osteoarthritis, which included the B2 glenoid or retroverted glenoid with posterior erosion and biconcave appearance (Fig. 10.1) [12].

Scapular dyskinesis is associated with abnormal scapular protraction [10]. Scapular protraction causes a decrease in scapular posterior tilt [10]. An abnormally protracted scapula in glenohumeral joint osteoarthritis may exacerbate posterior subluxation and contribute to the dreaded B2 glenoid. The challenge is to understand whether the protracted scapular position is the early culprit or whether the protracted scapular position is secondary to shoulder pain from osteoarthritis. Additional work will be needed to better understand the natural history.

Static posterior subluxation with early osteoarthritis in younger patients has been recognized as a challenging entity to surgically treat [13]. Primary shoulder osteoarthritis with static posterior subluxation was proposed as the first stage of primary glenohumeral osteoarthritis that predates posterior glenoid erosion [13]. Posterior humeral head subluxation is thought to be the cause of eccentric, posterior glenoid erosion [14]. Attempts at surgical correction with various approaches included posterior bone grafting with posterior capsule imbrications, posterior capsulorrhaphy, and others were completed. Follow-up demonstrated progression of osteoarthritis and persistent or recurrent posterior subluxation in all patients [13].

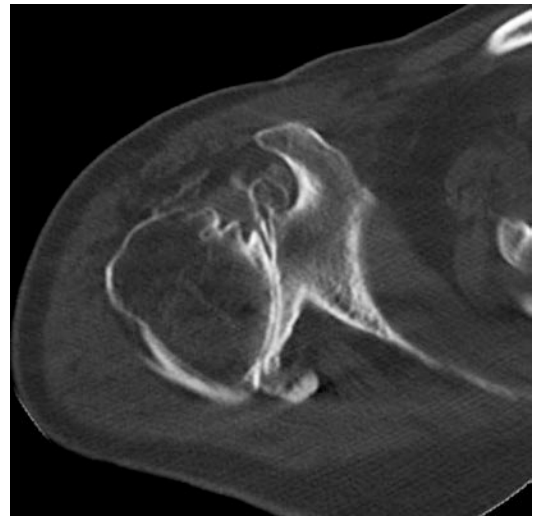

Fig. 10.1 CT arthrogram demonstrating eccentric posterior glenoid erosion with biconcavity consistent with Walch B2

There is a growing body of literature assessing the challenges of the B2 glenoid. However, our current body of literature does not consider the dynamic role of the scapula related to glenoid wear. Static 3D studies are very important for our foundation of understanding glenoid erosion; however, a better understanding of the dynamic role of the scapula will be critical moving forward. The scapula is clearly not a static structure and undergoes tremendous range of motion. McClure et al. measured 3D in vivo scapular kinematics during dynamic movements to help understand normal scapular motion [15]. 3D motions sensors were attached to scapular bone pins placed in healthy volunteers. The average ratio of glenohumeral to scapulothoracic motion in this healthy population was 1.7:1 [15]. Measurements during active scapular plane elevation included an average of 50° upward rotation, 30° posterior tilt, and 24° external rotation [15]. So although small changes in glenoid version are important, the wide variations in scapular motion throughout shoulder range of motion must be considered.

A recent matched cohort study found that B2 osteoarthritic glenoids have significantly greater premorbid glenoid retroversion compared with non-arthritic, normal glenoids using 3D computed tomography reconstruction [16]. The authors concluded that greater premorbid glenoid retroversion is associated with posterior instability and may be a causative factor in eccentric glenoid wear [16]. This study did not consider the dynamic role of the scapula related to glenoid wear.

A separate three-dimensional (3D) comparative study assessed scapulohumeral relationship in osteoarthritic and non-arthritic shoulders [17]. The study sought to better understand eccentric loading in osteoarthritic shoulders. Eccentric, posterior loading has been associated with worse clinical outcomes and correlated with glenoid component failure in anatomic total shoulder arthroplasty [17–21]. Contrary, to the prior cited study, this group concluded that osteoarthritic shoulders "do not have increased native glenoid retroversion predisposing to the development of the pathologic change" [17]. The group did not incorporate dynamic scapular motion, but they recognized that "scapulohumeral evaluation is necessary to understand the biomechanical relationship of the shoulder" [17]. Scapular stabilization exercises prior to surgical intervention may help to improve scapular dyskinesis and the protracted scapular position in this population.

Implications for Surgical Treatment in Early Posterior Subluxation and Posterior Glenoid Erosion and Primary Glenohumeral Joint Osteoarthritis

The patient population with mild posterior subluxation, posterior labral tear, and early glenohumeral joint osteoarthritis remains challenging (Figs. 10.2 and 10.3). In this patient population that is not quite ready for a total shoulder arthroplasty, it is challenging to know the optimal treatment plan. As noted, Walch et al. were unable to determine a successful treatment algorithm in the challenging patient population with static posterior subluxation [13].

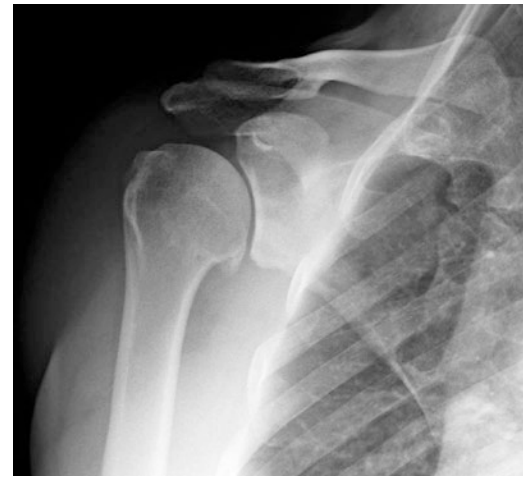

Fig. 10.2 Grashey radiograph demonstrating early osteoarthritis with joint space narrowing and an anterior inferior humeral osteophyte

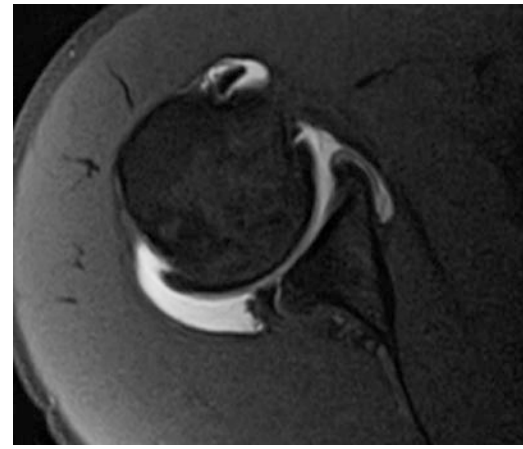

Fig. 10.3 Axial MRI demonstrating mild posterior subluxation with a large posterior labral tear

Our treatment approach for patients with mild posterior subluxation and early glenohumeral osteoarthritis has been modified based on our understanding of the scapula, but follow-up data to substantiate results is currently lacking. We believe that this type of patient may ultimately develop the dreaded B2 glenoid. Preoperatively, we assess the scapula and determine the presence or absence of scapular dyskinesis along with corrective physical examination maneuvers including scapular assistance test and scapular retraction testing. We find that this population

has a positive dynamic labral shear test with posterior joint line pain upon testing. We obtain plain radiographs to assess for osteoarthritis and for posterior subluxation. Magnetic resonance imaging (MRI) is obtained to further assess the extent of glenohumeral joint osteoarthritis, labral pathology, and for posterior subluxation. CT scan is often completed for additional assessment of glenoid morphology. Patients with scapular dyskinesis will undergo 6 weeks of scapular stabilization exercises. We have a comprehensive program that is described in the rehabilitation chapter. Some of our favorite exercises include sternal lift, step out, low row, inferior glide, robbery, and lawn mower exercises. These exercises are described in detail in the rehabilitation chapter.

Surgical intervention typically involves arthroscopic labral repair of the superior and posterior labrum (typically a 4-anchor repair), glenohumeral joint debridement including humeral osteophyte removal when indicated, and biceps tenodesis when indicated. Arthroscopic anterior capsular release is also completed if the patient has limited external rotation. Postoperative scapular rehabilitation is key and range of motion to prevent glenohumeral joint stiffness is critical.

Scapular Dyskinesis and Rotator Cuff Tear Arthropathy: Scapulohumeral Rhythm and the Role of Glenohumeral Joint Versus Scapulothoracic Motion

The prevalence of scapular dyskinesis and rotator cuff arthropathy has not been established. New research has assessed postoperative scapulohumeral and scapulothoracic motion in this patient population following reverse shoulder arthroplasty (RSA), but preoperative information is lacking. Similar to primary glenohumeral joint osteoarthritis, it is unclear if scapular dyskinesis resolves following reverse shoulder arthroplasty. It appears that the constraint in the reverse shoulder arthroplasty construct leads to less glenohumeral motion and places more demands on the scapulothoracic joint. Furthermore, it is unlikely that even an anatomic total shoulder arthroplasty restores the normal glenohumeral joint motion.

The biomechanics following reverse shoulder arthroplasty are clearly different than anatomic shoulder arthroplasty. De Wilde et al. were one of the first groups to assess the scapula following RSA [22]. In four patients following RSA for proximal humerus tumor surgery, scapulothoracic rhythm was assessed and increased lateral rotation or protraction was noted postoperatively [22].

Kwon et al. performed a kinematic analysis of shoulder motion following RSA in 17 patients greater than 6 months post-surgery compared to 12 healthy subjects [23]. The group used 3D electromagnetic motion capture to measure scapulothoracic and glenohumeral joint motion [23]. The majority of motion occurred at the glenohumeral joint, but scapulothoracic motion was significantly increased in the RSA group [23]. The group concluded that shoulder kinematics are significantly altered, and increased scapulothoracic motion is used to achieve shoulder elevation following RSA [23].

Walker et al. evaluated scapulohumeral rhythm in 28 patients greater than 1 year out from RSA using fluoroscopic 3D model imaging [24]. The scapulohumeral rhythm after RSA (1.3:1) was significantly lower than in normal shoulders (3:1) indicative of increased scapulothoracic motion and less glenohumeral joint motion in the RSA group [24]. Prior work in the same cohort revealed significantly increased upper trapezius and deltoid electromyographic activity compared to controls [24, 25]. The group postulated that improved rehabilitation protocols with attention to scapular muscle stabilization might optimize functional outcomes following RSA [24].

Implications for Surgical Treatment with Reverse Shoulder Arthroplasty

Based on the prior cited studies there are some implications for the role of the scapula following RSA. Patients with rotator cuff tear arthropathy clearly have diminished glenohumeral function, and we have empirically noted the presence of

scapular dyskinesis in these patients. Postoperative function is enabled by increased scapulothoracic motion following RSA. Optimization of scapular function can help to prevent increased shear forces across the glenohumeral joint and the RSA components. Catastrophic glenoid failure is rare with improved glenosphere fixation techniques, but long-term studies in the United States are lacking, and scapular function may contribute to preserved glenosphere longevity. We have encountered the rare situation of a patient with a prior scapulothoracic fusion in the setting of rotator cuff tear arthropathy. The scapular principles we have discussed were applied to this patient. A reverse shoulder arthroplasty could be considered for glenohumeral joint pain relief in this scenario; however, we would expect increased shear forces across the glenohumeral joint secondary to loss of scapulothoracic motion and potential for glenosphere failure.

Conclusions

There is limited evidence regarding scapular dyskinesis and shoulder arthritis. We have empirically noted scapular dyskinesis in patients with primary glenohumeral joint arthritis and in patients with rotator cuff tear arthropathy. Application of scapular principles to shoulder arthritis can help provide a foundation for the recognition and treatment of scapular dyskinesis preoperatively and postoperatively in patients with shoulder arthritis. We hypothesize that the challenging B2 glenoid may be accentuated by abnormal scapular protraction. Scapular rehabilitation in patients with early posterior subluxation with a posterior labral tear is suggested, and labral repair and postoperative scapular rehabilitation may prove beneficial in this patient population. Static 3D studies are very important for our foundation of understanding shoulder arthritis and glenoid erosion; however, a better understanding of the dynamic role of the scapula will be critical moving forward. The scapula is clearly not a static structure and undergoes tremendous range of motion. Dynamic scapular modeling may be possible in the future to allow preoperative planning with component placement based on patient-specific factors and scapular motion. These same findings may guide preoperative and postoperative rehabilitation to for specific periscapular therapy to optimize outcomes and maximize implant survivability.

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Scapular Muscle Detachment

11

W. Ben Kibler and Aaron D. Sciascia

Pathoanatomy and Clinical Presentation

Direct injury to the scapular muscles is not well known or well categorized. There is only a single case report that documented a traumatic avulsion of the rhomboids, with symptoms related to scapular winging, which resolved with surgical reattachment [1]. As a result, patients who have sustained a traumatic injury to the arm resulting in symptoms at or around the scapula can experience symptoms for months and/or years without an accurate diagnosis. This can have deleterious effects on the functional consequences. The pathoanatomy appears to be an anatomic or physiologic detachment of the lower trapezius and rhomboids from the spine and medial border of the scapula. As clinical experience with this group of patients has accumulated, it has been seen that patients with this injury will present with a very similar clinical history and physical examination. The salient features of this syndrome include:

1. A traumatic or disruptive event to the scapular stabilizing structures on the medial border with early manifestation of symptoms within the first 2 weeks
2. Pain of a high degree of intensity localized along the appropriate area of the medial border
3. Frequently a palpable defect in the area of pain
4. Weakness and arm dysfunction in positions that require scapular control against arm position (forward flexion, overhead motion, push/pull)
5. Substantial but temporary relief of symptoms on clinical exam by manual scapular stabilization
6. A very consistent group of surgical findings. All of these findings were present in a large portion of patients and form the clinical criteria for diagnosis of the syndrome

A wide variety of etiologic factors have been reported as the initial event. The large majority of cases present after an acute traumatic tensile load, almost half involving seat belt-restrained motor vehicle accidents, but there are multiple other causes such as direct blow trauma, throwing, catching or lifting a heavy object with the arm at full extension, pulling against a heavy object, hanging on the rim after dunking a basketball, and electrical shock such as electrocution or

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cardioversion. The pain along the medial scapular border increases in intensity as the condition evolves and averages 6/10 numeric pain rating at rest and 8/10 upon use. Due to its chronicity and high intensity, the pain may evolve into a centrally mediated chronic pain response [2], with a wide variety of effects on function and response to treatment. There are major limitations of arm use away from the body in forward flexion or overhead positions due to increased strain on the injured tissue. Increased upper trapezius activity and spasm, resulting from lack of lower trapezius activity, can create migraine-like headaches. Neck and shoulder joint symptoms may be present due to dyskinesis and will often become the focus of treatment, including surgery for cervical disk disease, shoulder impingement, or shoulder internal derangement, with infrequent positive results.

The physical exam also exhibits a consistent cluster of findings including the localized tenderness along the medial scapular border, often a noticeable and palpable soft tissue defect, either due to the detachment or the muscle atrophy, altered scapular resting position as well as dynamic dyskinesis including snapping scapula, shoulder impingement and weakness in forward flexion, acromioclavicular and/or sternoclavicular tenderness due to the dyskinesis, and clinical decrease or relief of symptoms with scapular corrective maneuvers.

Clinicians must be diligent at attempting to rule out detached scapular muscles when a traumatic injury mechanism has occurred resulting in pain along the medial scapular border. The clinical findings of scapular pain demonstrated rotator cuff weakness that is improved by scapular stabilization, and limitation of arm use in forward flexion and overhead rotation are all consistent with loss of activation of the lower trapezius and rhomboids [3–5]. These muscles are key muscles in stabilization of the scapula in retraction with arm elevation at or above 90° [5–7], and loss of activation of these muscles is seen in clinical rotator cuff weakness [8, 9] and impingement [3, 10]. Although glenohumeral joint injury may be present, care must be taken to not immediately assume glenohumeral joint; internal derangement is the primary pathology when medial border pain is noted by the patient. In a recent case series report of patients identified as having one or more detached scapular muscles, it was discovered that none of the patients who underwent subacromial decompression for symptoms of impingement received any relief of the shoulder or scapular pain which indicates that the basic problem was at the scapular level [11].

Imaging

At this time, the diagnosis is still a clinical one. Imaging has not been helpful in demonstrating the disruption, loosening, or hypertrophic scar that was found at surgery. In the first report of this condition, only two CT scans and one MRI suggested findings of the injury despite the fact that all 78 patients had multiple imaging studies. Methodological issues that may explain this problem include that the angle of the cuts was such that the area is not well demonstrated, the thickness of the cuts was not precise enough, or that the best MRI visualization method by which these lesions can be evaluated is not known. All of the MRIs were done in the chronic phase, so few signs of acute damage could be seen. The loose attachment of the tissues may obscure the readings for a tear. Two patients in more recent follow-up had MRI scans within 2 weeks of the injury that showed acute disruption and fluid in the rhomboid attachment area. At operation, there was no fluid or inflammation, the typical imaging markers seen in the damaged areas; the detached lower trapezius was loosely draped across the spine rather than retracted off the spine, so no detachment could be appreciated during the sequenced imaging, and the rhomboids are most frequently connected to the border through dense scar tissue. Diagnostic ultrasound may be a better imaging modality due to its capability of detecting thickened tissue. Until a more efficacious method of imaging is discovered, adhering to the specific inclusion criteria and history and physical exam findings is recommended to establish the clinical diagnosis, as these criteria have been successful in identifying patients who will display pathology at the time of surgery and will respond to surgical treatment with predictable outcomes.

In summary, although imaging is not successful in defining the pathoanatomy, the mechanism of injury, the clinical history, and the clinical examination are as consistent as inclusion and exclusion criteria that they form the basis of a clinical diagnosis that identifies the lesion, develops the treatment, and can be associated with predictable outcomes.

Scapular Muscle Reattachment Procedure

Initial treatment for the problem consists of identifying and treating the muscle imbalances and weaknesses that accompany and compensate for the muscle injury and the resulting dyskinesis, plus identifying other possible pathoanatomy in the neck and shoulder. However, the large majority of patients who meet the clinical inclusion criteria for this diagnosis will require surgical treatment to address the muscle detachment.

In order to reattach avulsed scapular stabilizing muscles, the following procedure was developed [11, 12]. Patients are given a general anesthetic and are placed prone with the involved arm down to the side, and chest roll towels are placed to allow the medial scapular border to be identified. The surface landmarks of the medial scapular border and spine are marked. An incision is made from superior to inferior along the medial border of the scapula from the spine to the tip over the area of maximal tenderness or defect. It will often run 6–8 cm inferior from the spine of the scapula but can extend the entire length of the medial border. If the patient complains of pain down to the inferior angle, the incision should be extended inferiorly to this area. The soft tissue is dissected to expose the area of the lower trapezius and rhomboid muscle attachments. The lower trapezius arches across the scapular spine and rhomboids and is used as a guide for localization (Fig. 11.1). The injury can

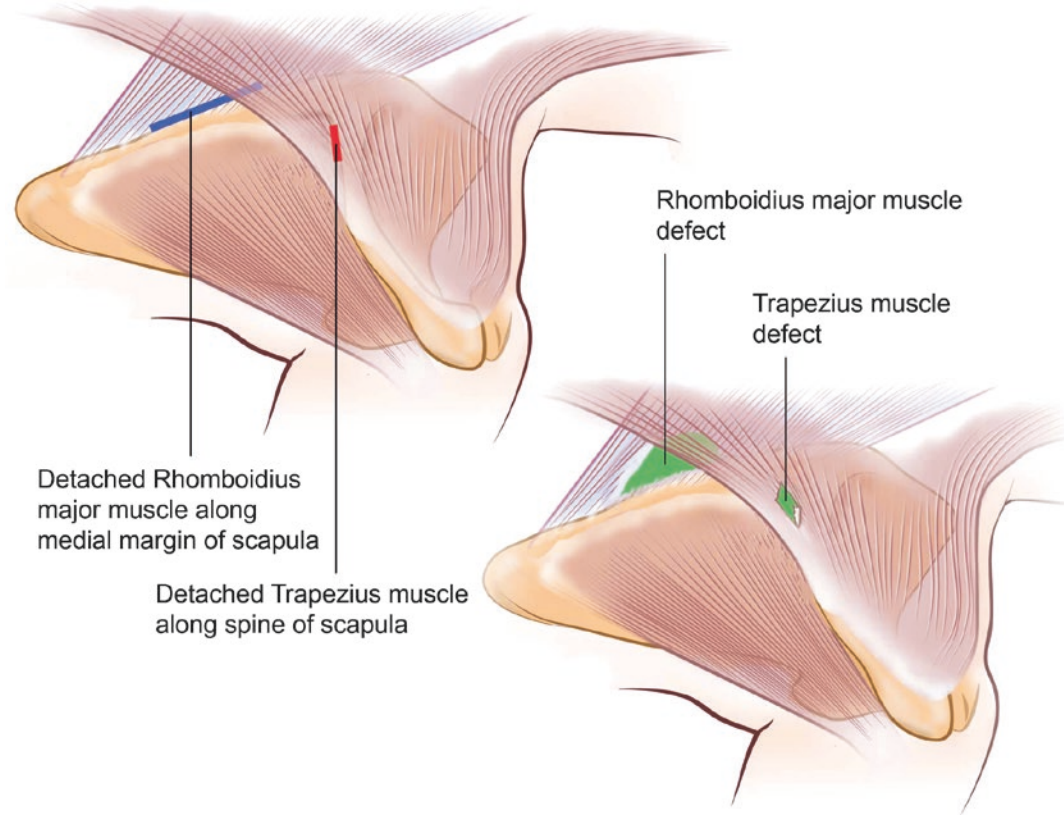

Fig. 11.1 Illustration of the scapular muscle detachment injury to both the rhomboid major and lower trapezius muscles

be appreciated as a detachment of the lower trapezius muscle and/or rhomboid muscles, loose attachment of the muscles via scar tissue, or connected through dense scar tissue. Once the affected muscles have been identified, the scar or connective tissue should be debrided and the muscles mobilized for reattachment (Fig. 11.2a). The infraspinatus muscle attachment is then reflected about 1 cm off the medial border and spine of the scapula in order to place drill holes, in sets of 2,1 cm from the medial border and spine. The holes are placed from a dorsal to ventral direction (Fig. 11.2b). The holes are placed 6–8 mm apart, and the sets are placed 10–15 mm apart along the medial scapular border and on the scapular spine. The total number depends on the length of the repair, although usually only one set of holes is made in the spine. This set is placed from superior to inferior 20–25 mm from the medial scapular border. In cases of extensive lower trapezius injury, two sets should be placed. The lower trapezius and rhomboid muscles are then mobilized to the dorsal aspect of the scapular body and spine. Mattress sutures which run from dorsal to ventral through the muscle and one of the pair of holes and then back from ventral to dorsal through the other hole and muscle are first placed through the rhomboid (Fig. 11.3a). The mattress sutures allow the rhomboids to be reattached on the dorsal surface of the scapula approximately 1 cm from the edge of the medial border. The lower trapezius is then reattached to

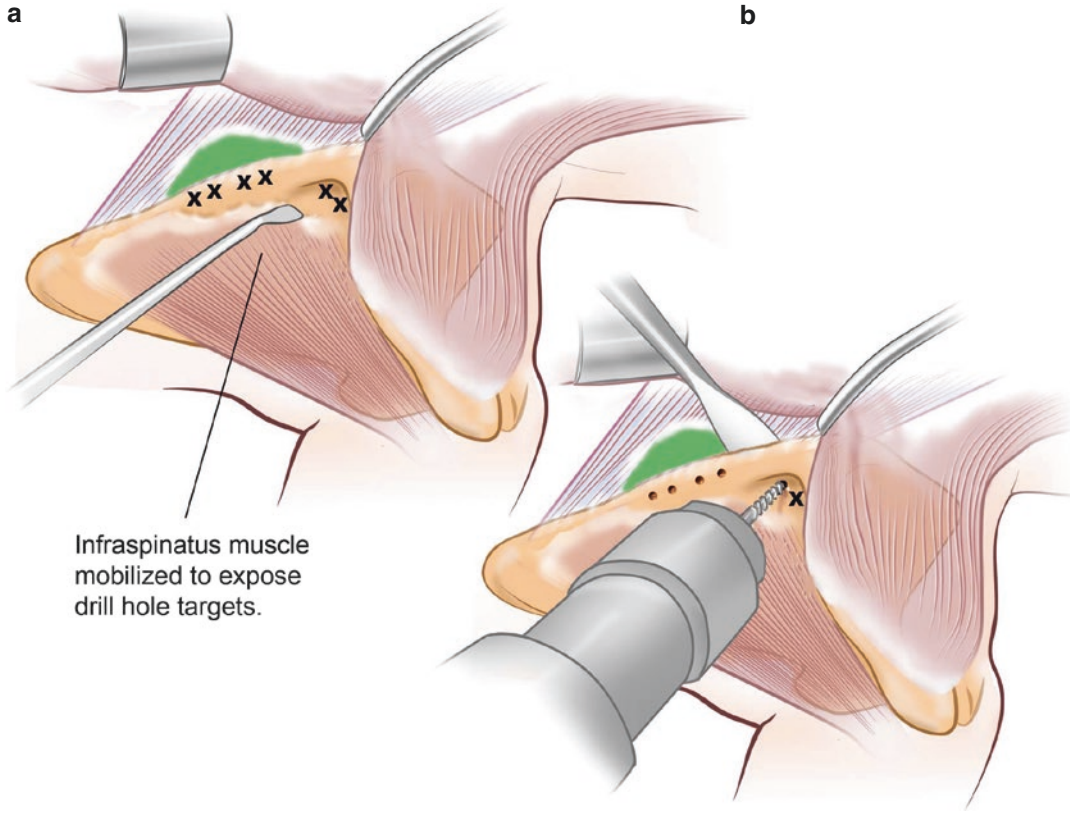

Fig. 11.2 (**a**) Mobilization of the infraspinatus away from the medial border of the scapula prior to drill hole placement. (**b**) Pairs of drill holes being placed in the medial scapular border and spine of the scapula

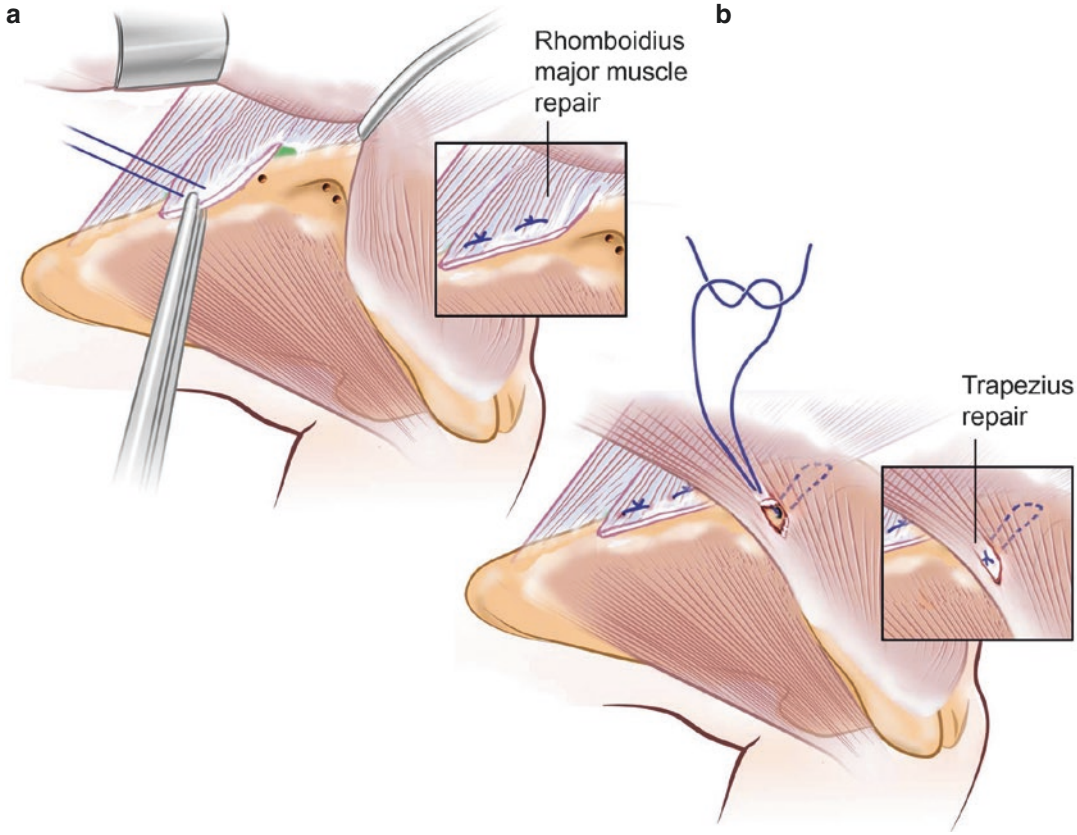

Fig. 11.3 (**a**) Illustration of the reattachment of the rhomboid major. (**b**) Illustration of the reattachment of the lower trapezius

the spine (Fig. 11.3b). The sutures are tied down with the scapula in external rotation. The infraspinatus is then reattached along the medial scapular border using the repair sutures followed by closure of the fascia and subcutaneous tissue. A comparative illustration of the presurgical injury to the postsurgical repair has been provided (Fig. 11.4a, b).

Postoperatively, the arm is protected in neutral rotation for 4 weeks, but gentle scapular retraction is encouraged immediately. During this period of recovery, common tasks such as mobile device use, driving, and other repetitive arm tasks with either the surgical or nonsurgical arm can create pain and muscle spasm due to "crosstalk" between the contralateral scapular muscles. In addition, the typical deconditioning/atrophy seen with postsurgical immobilization allows for easy arm fatigue, increasing the pain and spasm. Therefore, patients are instructed to not perform these tasks until after the sling has been removed about 3–4 weeks following surgery. At 4 weeks, closed chain activation up to 90° abduction with the hand stabilized is started. By 6–8 weeks, as the repair has healed and early strength is gained, motion over 90° is allowed, and the patient is started on the standard scapular strengthening program. Maximum strength is not regained for about 6–9 months, probably reflecting the chronic muscle disuse and atrophy.

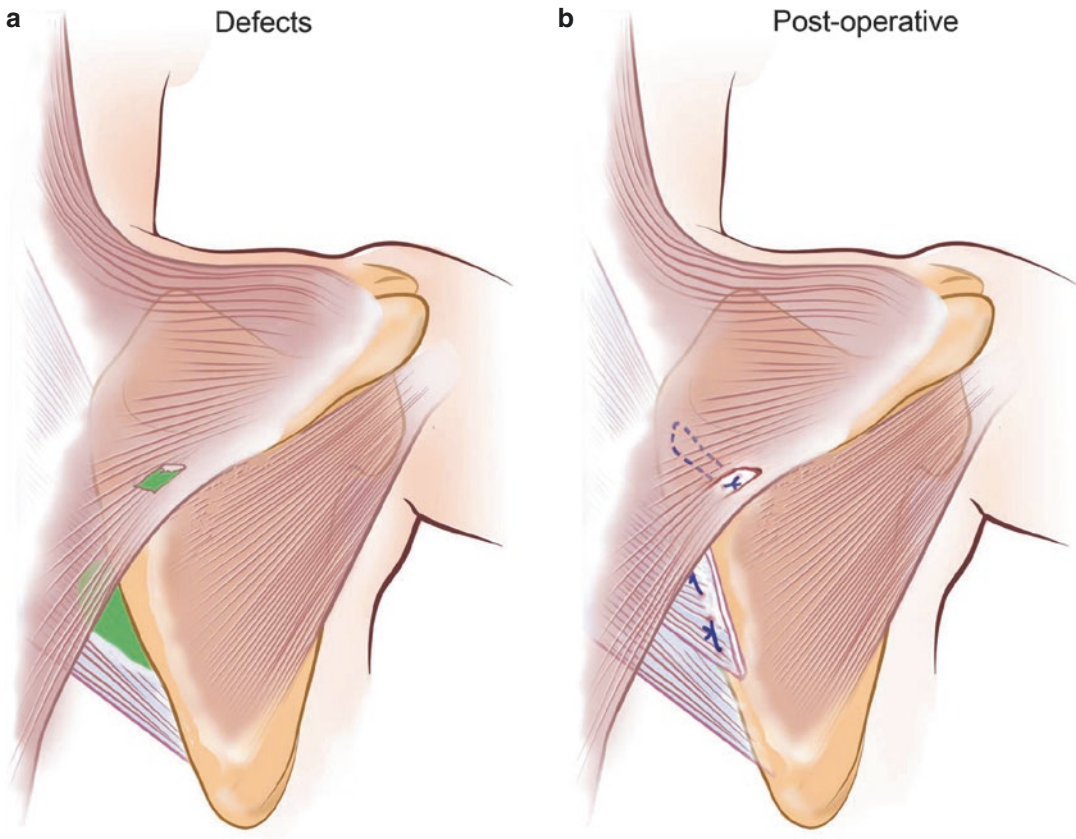

Fig. 11.4 (**a**, **b**) A comparative illustration of the presurgical injury (**a**) to the postsurgical repair (**b**)

Postsurgical Outcomes

In the original report describing the outcomes of surgical treatment in a large group with long-term follow-up, the entire group did well, achieving significant improvements in pain and function [11]. At discharge from active care, the ASES pain scores improved from 18/50 to 35/50, function scores improved from 20/50 to 28/50, and total ASES scores improved from 38/100 to 62/100 ($p < 0.001$). These results were maintained at minimum of 2-year follow-up. While the entire group did well, there was variation in the patient-reported outcomes. To look closely at the outcome variation, a subsequent evaluation subclassified the group based on achieving minimal detectable change for the total ASES score (>10 points) [13]. Fifty-eight of 78 patients (74%) did report greater than 10-point change and were considered significantly clinically improved. Twenty of 78 patients (26%) did not achieve at least a 10-point change on the ASES and were considered not significantly improved (Table 11.1).

Table 11.1 American Shoulder and Elbow Surgeons (ASES) scores between responders and nonresponders to scapular muscle reattachment surgery

	Responders ($n = 58$)	Nonresponders ($n = 20$)
	Presurgery	
ASES pain	16 ± 11	27 ± 12*
ASES function	18 ± 11	24 ± 9
ASES total	34 ± 16	50 ± 12*
	Post-surgery	
ASES pain	38 ± 10*	26 ± 12
ASES function	31 ± 11*	21 ± 10
ASES total	69 ± 18*	42 ± 16
Change from presurgery	**35 ± 18***	**−8 ± 14**

*Significantly greater score $p < .001$

Table 11.2 American Shoulder and Elbow Surgeons (ASES) scores between pain non-catastrophizers and pain catastrophizers following scapular muscle reattachment surgery

	Non-catastrophizers (n = 21)	Catastrophizers (n = 10)	P-Value
ASES pain	43 ± 8	27 ± 12	<.001
ASES function	40 ± 9	27 ± 12	.005
ASES total	83 ± 15	54 ± 18	<.001
Did not meet ASES MDC >10	1[a]	6	.047

[a]Patient initial ASES total = 90 and most recent follow-up ASES total = 88
MDC minimal detectable change

There may be several factors that contributed to this difference in patient-reported outcome in this group whose diagnosis, inclusion criteria, and surgical treatment are so homogenous. Among them are altered functional demands in the postoperative and return to activity phases, differences in implementation and completion of rehabilitation in the widely geographically dispersed group, chronicity of the injury with its effects on muscle strength and activation, and patient expectations and perceptions of the clinical problem and its effects on function.

One major patient-oriented effect that can impact reported outcomes is perception of pain. This factor has been noted following other types of shoulder procedures [14–18]. A pilot study assessed pain perception for a group of 31 postoperative patients with the #Pain Catastrophizing Scale (PCS) [19]. This 13-item self-reported scale estimates the patient's attitude toward the effect of pain on their function in their daily lives, which has a maximum scope of 65 (lower score = less pain catastrophizing characteristics). Twenty-one patients were classified as non-catastrophizers (PCS scores <30), while ten patients were classified as catastrophizers (PCS scores ≥30) meaning that they were prospectively and constantly aware of pain which they perceived would negatively impact their function. This patient-perceived effect was shown to impact reported outcome in several ways.

Only 3/10 patients in the catastrophizing subgroup reported satisfaction with the surgical outcome, while 7/10 were not satisfied or unsure of the outcome. Catastrophizing also had a differential effect on the total ASES scores. The average total ASES scores for the non-catastrophizers were 29 points greater than the patients identified as having pain catastrophization characteristics (Table 11.2). Patients who were not satisfied with the surgery and were catastrophized had 12–17 points less on the ASES pain component compared to non-catastrophizers, while there was only a 4–6-point difference between the catastrophizers and non-catastrophizers on the ASES function component.

These findings indicate that pain perception can be a significant factor in collecting data for patient-reported outcomes. It appears that pain perception is different in patients reporting a less satisfactory outcome. This patient-specific characteristic may have existed prior to the injury, could have been affected by a multitude of other factors (e.g., stress/anxiety, previous experiences, etc.), or be due to neuroplastic changes in the nociceptors, spinal cord, and brain known to contribute to chronic pain [15]. This may be very important in this group, since the diagnosis is frequently delayed, leading to long duration of the injury and pain.

Several clinical implications arise from these findings. First is the recognition that the patient's general response to pain, and the specific effect on catastrophizing, may have a large effect on treatment, and the patient-reported outcome should be evaluated in the diagnostic process and, if appropriate, treated as part of the comprehensive treatment plan. Second, it appears from clinical experience that rebalancing muscle tension by the reattachment procedure is a major source of relief of the pain felt along the medial scapular border and that this relief can be felt early in the postoperative period. Therefore, intensive efforts should be made to identify patients with this injury early to minimize the deleterious effects of the detachment on pain and

muscle inhibition. Most patients report satisfaction with the operative result because of the reduction in pain levels and the consequent ability to resume daily activities.

Summary

Scapular muscle detachment appears to be a clinically identifiable syndrome with a relatively homogeneous set of history and physical findings that can be used for the diagnosis and treatment. Its exact incidence is unknown but may be relatively common as better recognition is achieved. Surgical treatment can result in significant reduction in pain in almost all cases, but total functional capability will vary and may not return to normal. Factors impacting the functional capability may include the sequelae of chronic pain, long-term muscle atrophy, altered muscle activation patterning, and sequelae from other operations. Patient-reported factors, especially perception of pain, may have a large impact on reported outcomes. Awareness of this condition can allow earlier recognition, evaluation, and treatment, with shorter periods of disability, less functional decompensation, and hopefully better functional outcomes.

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Neurologic-Based Injuries and Scapula Winging

12

John E. Kuhn

Introduction

The scapula serves as a foundation for upper extremity function and strength. Like a crane on a construction site, the scapula must be secured to its foundation in order for the arm to lift and move heavy objects. If the cab of the crane does not have the footings secure, it will tip. If the scapula is not secured to the chest wall, it too will tip. Of the 17 muscles that have their origin or insertion on the scapula, those that serve like feet on a crane and secure the scapula to the torso include the pectoralis minor, omohyoid, levator scapulae, serratus anterior, trapezius, and rhomboideus major and minor (Table 12.1).

There are many causes of scapular winging [1], including static sources (most commonly an osteochondroma (Fig. 12.1)), dynamic sources (scapular dyskinesis), traumatic muscle avulsions [2], and, most commonly, from a neurologic injury. This chapter will focus on neurologic sources of scapular winging.

Table 12.1 Muscles of the scapula

Scapulothoracic muscles
Levator scapulae
Omohyoid
Pectoralis minor
Rhomboideus major
Rhomboideus minor
Serratus anterior
Trapezius
Scapulohumeral muscles
Rotator cuff
Infraspinatus
Subscapularis
Supraspinatus
Teres minor
Other
Biceps long head
Biceps short head
Coracobrachialis
Deltoid
Teres major
Triceps long head

Of the 17 muscles that are attached to the scapula, the seven scapulothoracic muscles provide the foundation which stabilize the scapula to allow upper extremity function. If these muscles are not functioning normally, scapular winging can occur

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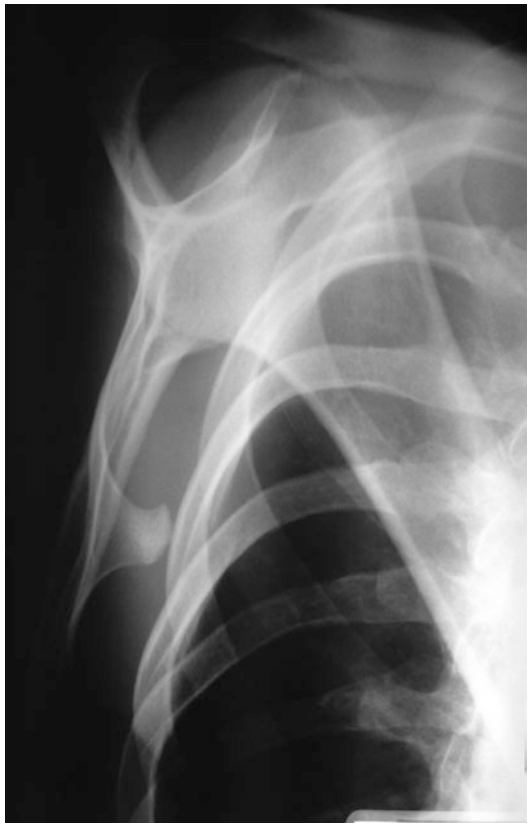

Fig. 12.1 Osteochondroma of the scapula. This is the most commonly seen tumor of the scapula and may be a source of static scapular winging

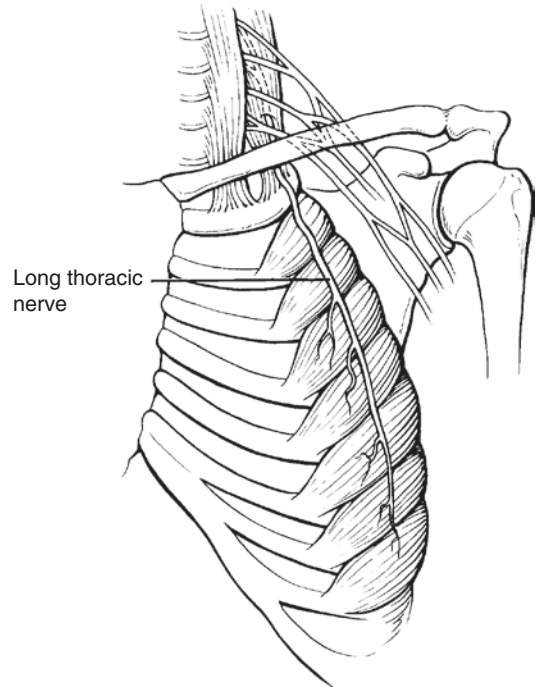

Fig. 12.2 Anatomic position of the long thoracic nerve (From Kuhn JE, Hawkins RJ. Evaluation and treatment of scapular disorders. In: Warner JP, Iannotti JP, Gerber C, eds. *Complex and revision problems in shoulder surgery.* Philadelphia: Lippincott-Raven Publishers, 1997:357–375)

Long Thoracic Nerve Injury and Serratus Palsy

The long thoracic nerve innervates the serratus anterior. This nerve typically arises from the anterior rami of C5, C6, and C7. It is important to note that the C5 and C6 roots of the nerve perforate the scalenus medius muscle and may be tethered here. The nerve then descends behind the brachial plexus and axillary vessels and runs along the side of the thorax innervating the slips of the serratus anterior (Fig. 12.2). The length and superficial position make the long thoracic nerve susceptible to neuropraxic stretch injury.

History

Injury to the long thoracic nerve typically occurs as a result of a stretching mechanism [1], especially in sports, although the nerve can be injured by compression, or very rarely penetrating trauma. Patients may not notice symptoms until days or weeks later. Usually a loss of upper extremity strength is the chief complaint, although prolonged symptoms may lead to pain from other periscapular muscles, especially the pectoralis minor and levator scapulae, as these muscles are excessively active in an attempt to compensate for the weak serratus anterior.

Physical Examination

With serratus anterior weakness, the levator scapulae, rhomboids, and trapezius will dominate pulling the scapula medial and superior (Fig. 12.3a, b). Tenderness may be found at the origins of the levator scapulae (superomedial angle of the scapula) and pectoralis minor (medial aspect of the coracoid). Winging may be accentuated by resisted flexion of the extended arm.

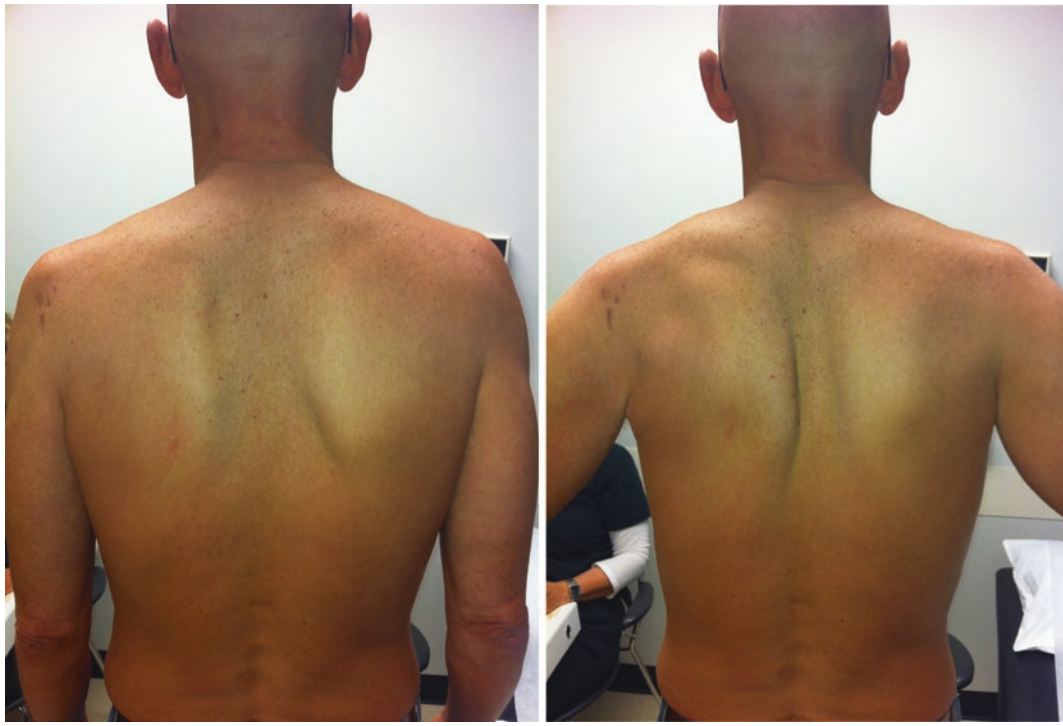

Fig. 12.3 Winging due to left serratus palsy. (**a**) At rest. Note the trapezius and rhomboids dominate pulling the scapula superior and medial. (**b**) With abduction. Elevation or abduction of the arm will accentuate the winging

Imaging

For neurologic injury, radiographs and MRI are not particularly helpful. These studies are more useful for structural sources of static winging (osteochondroma, malunions of rib or scapula fracture) or muscle avulsion.

EMG Analysis

Injury to the long thoracic nerve can be detected by EMG [3]. Findings in the injured nerve include increased latency, with fibrillations and sharp waves in the involved serratus anterior. In addition, a decreased number of motor unit action potentials are noted with voluntary contraction.

Differential Diagnosis

The differential diagnosis for scapular winging due to serratus palsy includes static causes of winging (scapular osteochondroma, malunited scapula or rib fracture), muscle injury (serratus or other), or other neurologic injury, including the spinal accessory nerve, the dorsal scapular nerve, and/or the brachial plexus or cervical nerve root injury.

Treatment

Conservative treatment is recommended as most cases of long thoracic nerve injury are neuropraxic and will recover spontaneously. Because the nerve is so long, however, the recovery may be up to 2 years. Recovery can be followed clinically or via serial EMG studies conducted no more frequently than every 3 months. Approximately 80% of patients do well in the long term with resolution of the winging and normal flexion and abduction; however many patients still have pain at long-term follow-up [4].

Surgical neurolysis of the long thoracic nerve in the supraclavicular region has been reported as a treatment with successful outcomes [5]. Release of the distal part of the nerve has also been reported [6]. It is important to recognize that if

neurolysis is performed early, it is unknown if these patients would have recovered spontaneously. The outcomes of neurolysis suggest relatively rapid recovery of the nerve.

In patients in whom the serratus palsy does not recover after 18–24 months, or in those in whom no recovery is noted after 12 months on serial EMG studies, muscle transfer surgery may be offered. Marmor and Bechtol [7] described transfer of the pectoralis major with a fascia lata extension to the inferior angle of the scapula (Fig. 12.4). There has been some concern with the potential for failure with indirect transfers of the tendon, leading some to recommend transfer of the tendon directly to the scapula (direct transfer) [8, 9]. In addition, because the muscle orientation is closer to the serratus, many authors recommend using only the sternal head of the pectoralis major, which reduces scarring and improves cosmesis in the axilla [8, 10]. Elhassan and Wagner [11] has described a variation of this transfer where a portion of the humeral bone is retained on the tendon of the sternal head of the pectoralis major, which allows bony union to the scapula.

Outcomes

Transfer of the sternal head of the pectoralis major to the scapula is helpful, and one can expect good to excellent results in approximately 90% of patients [8, 10, 12, 13]. Failure and recurrence of winging is a known complication and may be less common when the direct transfer is employed [12].

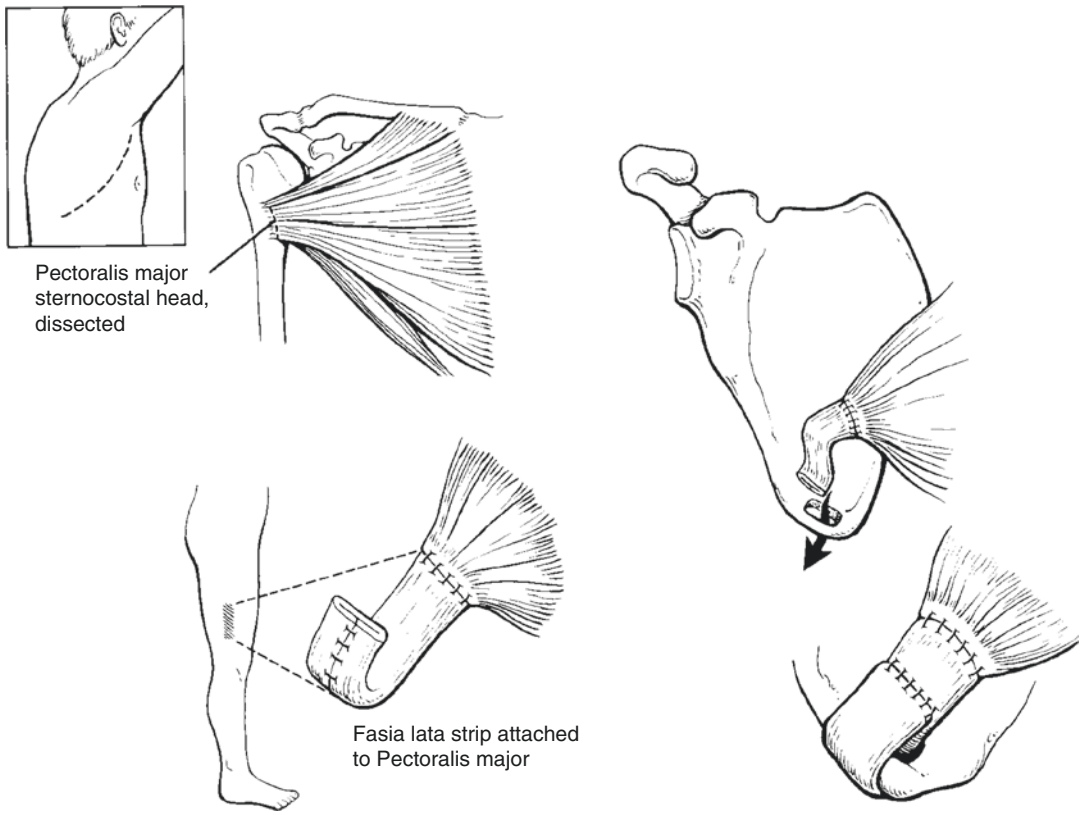

Fig. 12.4 Transfer of the pectoralis major muscle. The drawing depicts an indirect transfer of the pectoralis major with a fascia lata extension as described by Marmor and Bechtol [7]. Direct transfers would attach the tendon directly to the scapula (From: Kuhn JE, Hawkins RJ. Evaluation and treatment of scapular disorders. In: Warner JP, Iannotti JP, Gerber C, eds. *Complex and revision problems in shoulder surgery.* Philadelphia: Lippincott-Raven Publishers, 1997:357–375)

Spinal Accessory Nerve Injury and Trapezius Palsy

The spinal accessory nerve (cranial nerve XI) passes along the internal jugular vein, crossing it to innervate the sternocleidomastoid muscle. It then enters the posterior triangle of the neck to supply the trapezius (Fig. 12.5). It is fairly superficial and located near the posterior aspect of the sternocleidomastoid muscle, making it susceptible to iatrogenic injury.

History

Spinal accessory nerve injuries are almost always iatrogenic [14], typically as a result of a lymph node biopsy or other surgery in the posterior cervical triangle. The diagnosis and treatment are often delayed [14], and injury to this nerve is a common source of malpractice claims [15].

Physical Examination

With a palsy of the trapezius, inspection of the patient will demonstrate a loss of the usual webbing of the neck and often a surgical scar over the posterior cervical triangle. The serratus will dominate, and the scapula will rest in a depressed and lateral position (Fig. 12.6a, b). With elevation the medial border of the scapula will wing substantially. This can be accentuated by resisted flexion of the arm.

Two special physical examination tests have been described by Levy et al. [16]. The *Active Forward Elevation Lag Sign* in which elevation of the affected side requires increased lumbar lordosis and the triangle sign, which is maximizing forward elevation while the patient is lying prone on an examination table. The affected side will require elevation of the torso from the table creating a triangle formed by the arm, trunk, and exam table.

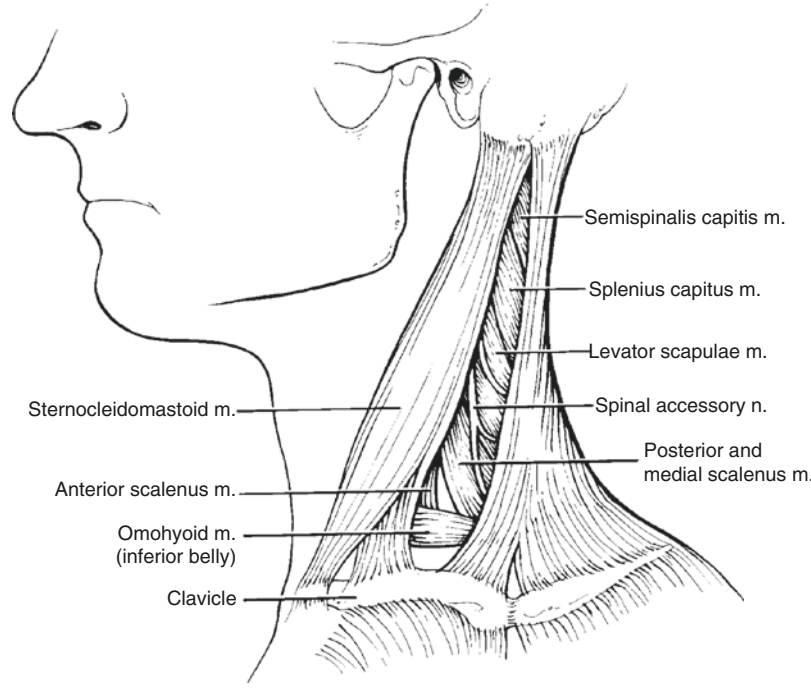

Fig. 12.5 Anatomy of the spinal accessory nerve. This nerve is superficial in the posterior cervical triangle, making it susceptible to iatrogenic injury during surgery (From: Kuhn JE and Hawkins RJ. Evaluation and Treatment of Scapular Disorders. In Warner JP, Iannotti JP and Gerber C. Eds. Complex and Revision Problems in Shoulder Surgery. Lippincott-Raven publishers, Philadelphia 1997, 357–375)

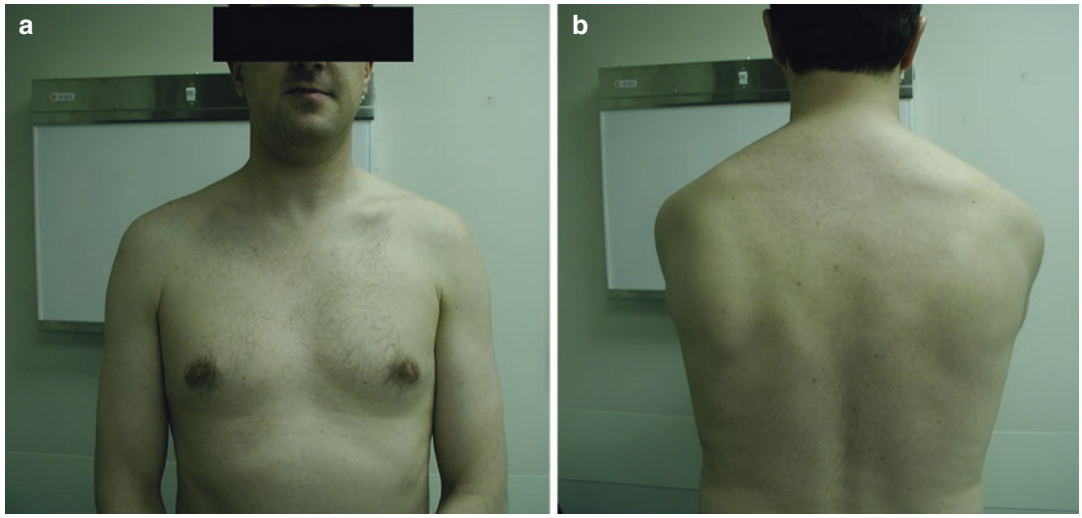

Fig. 12.6 Winging due to left trapezius palsy. (**a**) At rest. Notice the webbing of the neck on the patient's left is diminished. (**b**) With elevation. The serratus will dominate pulling the scapula lateral and inferior

Imaging

For neurologic injury, radiographs and MRI are not particularly helpful. These studies are more useful for structural sources of static winging (osteochondroma, malunions of rib or scapula fracture) or muscle avulsion. As expected, chronic denervation of the trapezius will produce abnormalities on MRI that include trapezius muscle atrophy and signal hyperintensity in the STIR images. Scarring may be detected around the nerve in postsurgical patients [17].

EMG Analysis

Electromyography will demonstrate a low-amplitude SAN compound muscle action potential (CMAP) that requires a higher stimulus intensity to obtain it than on the unaffected side. Upper trapezius needle electromyography shows dense fibrillation potentials, with voluntary motor unit potentials (MUPs) in about half of injuries [18].

Differential Diagnosis

While there are many causes of scapula winging to consider [1], in a patient with a cervical lymph node biopsy or surgery in the posterior cervical triangle, it would be unusual to see winging from another source.

Treatment

Surgical repair of the lacerated nerve has met with some success, particularly if performed early, ideally within 1 year [19–23]. Transfer of the lateral pectoral nerve [24] to the spinal accessory nerve has been described. Unfortunately, injury to the spinal accessory nerve is often diagnosed late, and surgery on the nerve may not be successful.

Some patients with this condition can be treated conservatively; however patients with difficulty raising the arm over shoulder level or who have the dominant arm affected may not do well with conservative treatment [25]. In these patients a transfer of the levator scapulae and rhomboid muscles as described by Eden and Lang may be employed [26, 27] (Fig. 12.7). In this operation the levator scapula is detached from its insertion on the superomedial angle of the scapula and transferred laterally on the spine of the scapula, while the rhomboid major and minor are detached from the medial border of the scapula and transferred laterally in the infraspinatus fossa.

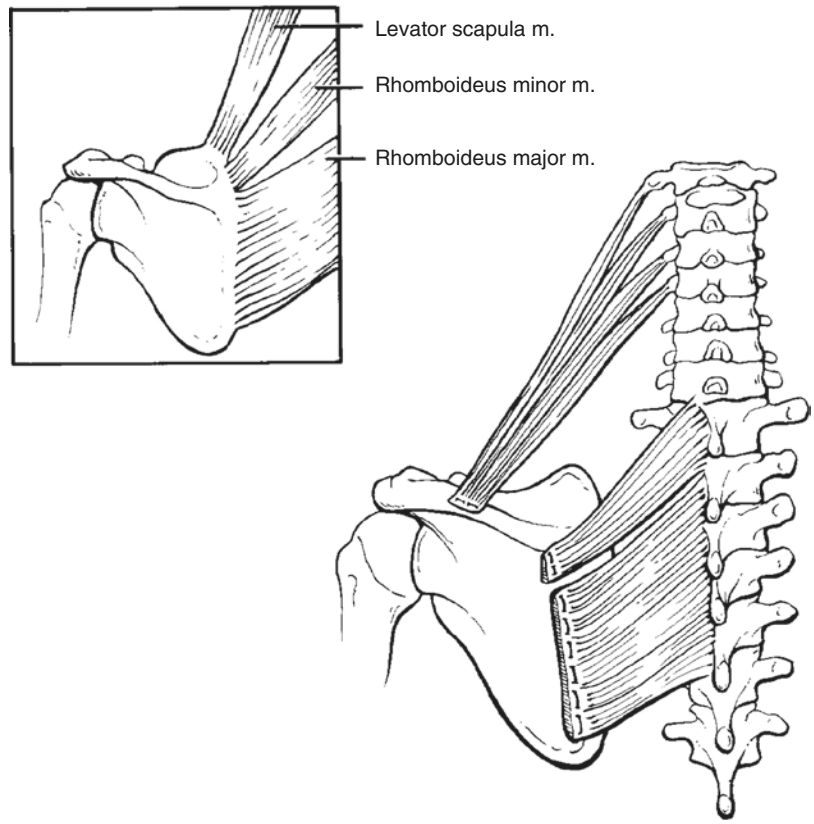

Fig. 12.7 Eden lange transfer for trapezius palsy. The levator scapula is transferred laterally on the spine of the scapula; the rhomboids are transferred lateral in the infraspinatus fosse (From: Kuhn JE. Chapter 34: The Scapulothoracic Articulation: Anatomy, Biomechanics, Pathophysiology, and Management. In: Disorders of the Shoulder: Diagnosis and Management. JP Iannotti, and GR Williams eds, Lippencott Williams & Wilkins Publishers, Philadelphia, PA, 1999, pp. 817–846)

Elhassan and Wagner described a variation of this technique where the rhomboid muscles are separated and transferred to different parts of the scapular spine [13].

Outcomes

Good to excellent results for these transfers can be expected in approximately 75% of patients [22, 23, 28, 29]. Poorer outcomes may be seen in patients over 50 years of age and patients with other shoulder disorders [22, 29].

Dorsal Scapular Nerve Injury and Rhomboid Paralysis

The dorsal scapula nerve arises from C5 and C4 ventral rami then pierces the middle scalene muscle before running deep to the levator scapulae and descending along the medial border of the scapula to innervate the rhomboid muscles.

Dorsal scapular nerve injury is rare and has been described in only a few case reports [30, 31, 32] but can occur as a complication of interscale blocks during shoulder surgery [33].

Patients with injury to the dorsal scapular nerve may have pain along the medial border of the scapula. Specialists in chronic pain have recognized dorsal scapular nerve syndrome, which is characterized by medial scapula pain that can radiate to the lateral arm and forearm, with accompanying functional impairment of different distress [34].

Winging will occur at the medial border and inferior angle of the scapula and can be accentuated by having the patient put the hands on his or her hips and pushing the elbows back against resistance [35]. The differential diagnosis includes C5 radicular injury as this, too, will produce rhomboid weakness.

As this condition is extremely rare, little in the literature exists regarding surgical treatment. Conservative treatment is often recommended; however in complete nerve injuries, an exploration and repair of the nerve may be attempted. The author has performed one case where the teres major was transferred from the humerus to the thoracic spinous processes with a good result.

Summary

Scapular winging has many causes; however neurologic injury creating muscle palsy is the most common. The long thoracic nerve creating serratus palsy is the most common, followed by the spinal accessory nerve creating trapezius palsy, and the least common is injury to the dorsal scapular nerve creating rhomboid paralysis. It is important to recognize these injuries early and begin treatment quickly. While most cases of serratus palsy will recover spontaneously, injuries to the spinal accessory nerve are usually iatrogenic and may require early surgical intervention. A variety of approaches to these injuries exist, and most produce significant improvement in pain and function when they are employed.

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Rehabilitation for Neurological Issues

13

Martin J. Kelley and Michael T. Piercey

Long thoracic nerve palsy (LTNP) and spinal accessory nerve palsy (SANP) can each provide a great challenge for the scapular specialist to rehabilitate due to loss of serratus anterior or trapezius function, respectively. Injury to either nerve may occur in isolation or in combination, due to blunt trauma, penetrating trauma, compression, stretch, traction, viral infection, or iatrogenic trauma [1–16]. Injury to the dorsal scapular nerve, affecting the rhomboid muscles, does occur but will not be directly discussed because of its rarity. Patients presenting with LTNP or SANP may report cervical, thoracic, shoulder, or scapular pain; sensation of upper extremity weakness or instability; and limited shoulder active range of motion (AROM), most notably shoulder flexion in patients with LTNP and abduction in patients with SANP [1, 2, 5–12, 15–18]. However, in the majority of cases, the patients will experience 1–3 weeks of scapular region pain and then only have pain-free weakness. These impairments usually result in some functional limitation of the involved extremity, especially involving lifting or resistance; however, most can function with surprisingly little difficulty for most activities. The severity of symptoms may vary but will often resolve within 24 months through neural regeneration in cases of neuropraxia and axonotmesis [9, 11, 12, 14–18]. In order to appropriately guide patients with LTNP or SANP, the scapular specialist must possess a strong understanding of scapular anatomy, mechanics, and normal muscular activation, as well as the appreciation to complete a thorough scapular evaluation.

The long thoracic nerve arises from the fifth, sixth, and seventh cervical nerve roots; crosses the middle scalene; passes behind the brachial plexus; enters the axilla; angles around the second rib; and descends on the anterolateral surface of the chest wall to exclusively innervate the serratus anterior superficial to the muscle [4, 8, 9, 12–14, 16, 19, 20]. The serratus anterior is a large, fan-shaped muscle with multiple digitations, divided into three components, originating at the lateral portion of ribs 1–9 and attaches on the medial scapular border [6, 9, 12, 16, 18–21]. Collectively, the components of the serratus anterior are responsible for protracting and upwardly rotating the scapula to allow for proper glenoid position during shoulder motion while also maintaining scapular contact with the thoracic wall [4, 6, 9, 12, 14, 16, 18, 22–27]. Ekstrom et al. [21] suggest the lower portion of the serratus anterior to be more heavily involved with scapular upward

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rotation, while the upper portion is more active with protraction. Given the sensitive course of the long thoracic nerve and multiple digitations of the serratus anterior, it is important to consider that one portion of the serratus anterior may be affected by LTNP, while the remaining portions can still be intact. This may occur during reinnervation when the upper fibers are active while the lower fibers are not, resulting in more protraction than upward rotation.

The spinal accessory nerve descends from the posterior cervical triangle, innervates the sternocleidomastoid, and continues superficial to the levator scapulae but deep to the medial portion of the trapezius along the medial scapular border to exclusively innervate the trapezius [1, 2, 7–9, 12, 15, 28]. The trapezius originates from the spinous processes of C7 through T12 and fans into three parts [7, 9, 12, 15]. The upper part inserts posterior to the lateral third of the clavicle, the middle part inserts on the medial acromion and superior to the scapular spine, and the lower inserts inferior to the scapular spine [7, 9, 12, 15]. In collaboration, the trapezius is crucial in providing stabilization to the scapula, as well as elevating, retracting, and rotating the scapula during overhead shoulder motion, especially abduction [1, 7, 10, 12, 15, 22, 24–26, 28–30].

Under normal circumstance, muscular force couples are required for proper scapulohumeral mechanics, as established by Inman [24]. Forces from the upper trapezius, levator scapulae, and serratus anterior are necessary to counteract the weight of the shoulder girdle by upwardly rotating the scapula [21, 22, 24, 25, 30–32]. Rotary forces are provided by the middle trapezius, lower trapezius, and rhomboid to pull the acromion medially, and the serratus anterior pull the inferior scapular angle anterolaterally [22–25, 30, 32]. When impaired motor control of the scapular rotators is present, such as with the serratus anterior during LTNP or the trapezius with SANP, imbalance of the normal scapular force couples results in abnormal kinematics, such as scapular dyskinesis, and resultant tensile overload of normally functioning tissues [23, 25, 31–33].

Assessment

Often, LTNP or SANP results in static scapular malposition due to the disruption of serratus anterior or trapezius tone and activity [8, 15, 16]. First, in a seated or standing position, posture should be assessed since the thoracic and cervical spine orientation will have a direct relationship on resting scapular position and scapular motion [25]. Specifically, the presence of scoliosis must be recognized in order to avoid confusion and falsely identifying the presence of both static and dynamic abnormalities. An individual with a commonly seen right thoracic curve will have right thoracic rotation causing accentuation of the rib angle (rib hump). This changes the scapula's resting position by creating a medial border prominence and often displaces the scapula slightly higher. Because the scapula is now on a differently shaped "platform" from the other side, it will move asymmetrically. This is not to be confused with true dyskinesis.

In patients with LTNP, the medial border will appear prominent, and typically the scapula is lower. Patients with a SANP demonstrate significant atrophy of the trapezius. Visually, this is most notable of the upper trapezius. The scapula (and whole shoulder girdle) of patients with SANP will droop, abduct, and downwardly rotate [1, 5, 8–10, 12, 15, 16, 25, 28].

The Scapular Muscle Examination Algorithm can be used to determine the presence of nerve palsy, motor control, dyskinesis, or some other pathology (Fig. 13.1). The specific tests can also help track reinnervation over time. The examiner must first determine if significant or obvious dyskinesis is present. The scapular dyskinesis test [34, 35] can determine if either "yes," an abnormal pattern/dyskinesis is observed, or "no," normal scapular motion is observed. Dyskinesis occurring in the sagittal plane may suggest LTNP. Medial winging often occurs near 90°, especially on descent. Often the lower trapezius becomes very active and visible toward available active end range where it attempts to compensate by retracting the scapula. Whereas dyskinesis or painless inability to elevate beyond 90° in the coronal plane may be indicative of SANP, either presentation will warrant further consideration of LTNP or SANP (Fig. 13.2).

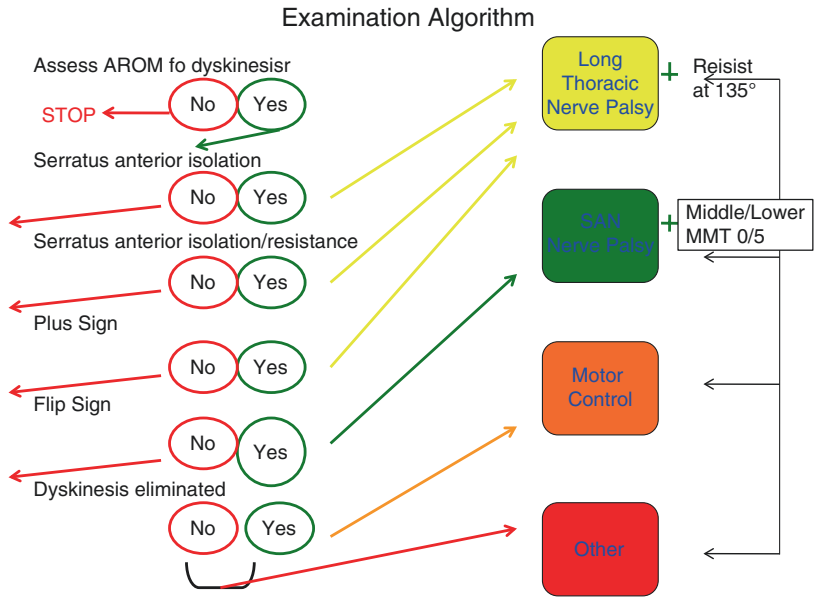

Fig. 13.1 Scapula muscle examination algorithm

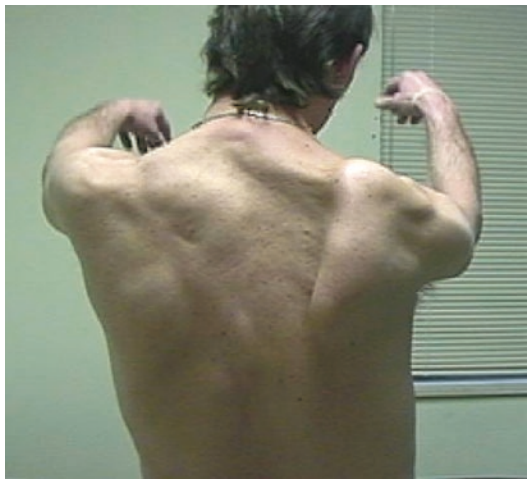

Fig. 13.2 Patient with both LTNP and SANP

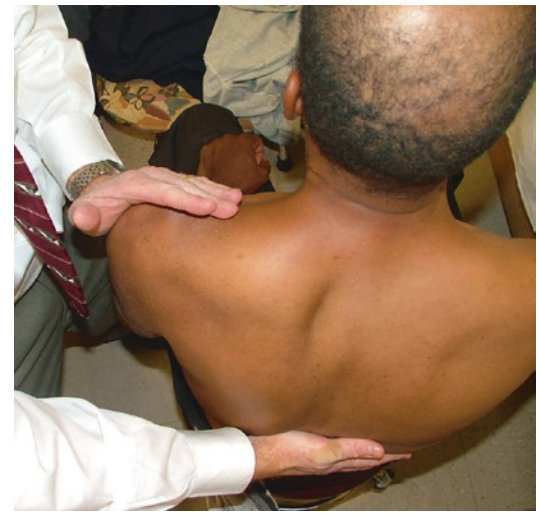

Fig. 13.3 Serratus anterior isolation with resistance

In order to assess for the presence of LTNP, the serratus anterior isolation test, without and with resistance, plus sign, and manual muscle testing should be assessed. The serratus anterior isolation test is completed with the patient's arms at the side in humeral external rotational (ER) while the patient actively protracts and elevates the scapula [19, 25, 32, 36]. A positive result is considered if the patient is unable to displace the scapula equally to the uninvolved side or inferior angle/medial border prominence still exists. To further challenge the serratus anterior, the clinician may add a posterior-directed resistance to the anterior shoulder to assess for the ease, and amount, of posterior displacement of the medial scapular border (Fig. 13.3). A plus sign is found if scapular

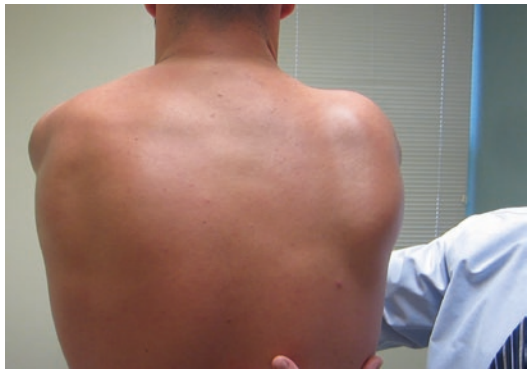

Fig. 13.4 Plus sign. Negative (*left*), positive (*right*)

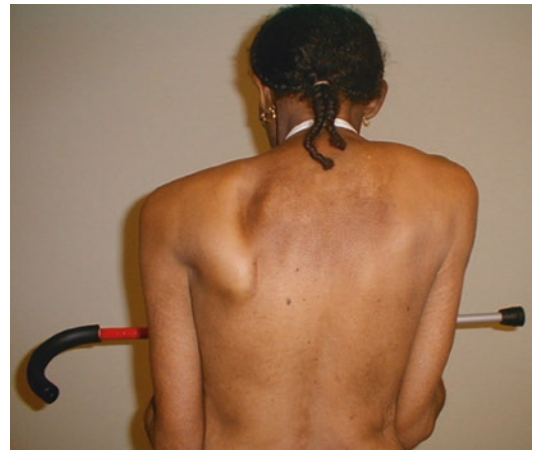

Fig. 13.5 Flip sign

winging is present when the patient reaches his arms forward at 90° in the sagittal plane. When doing so, the compensatory trapezius activity is forced to shut off due to antagonistic inhibition, and the scapular position is completely dependent on the serratus activity (Fig. 13.4). Plus sign may also be present when the patient completing a push-up against a wall with the shoulders flexed to 90° [8, 9, 16, 19, 25]. Manual muscle testing of the serratus anterior is completed with the patient supine and the tested extremity placed in 90° of shoulder and elbow flexion while the arm is protracted [37, 38]. The tester's force is placed through the ulna at the olecranon process along the axis of the humerus [37, 38]. Attention must be given to the pectoralis minor to recognize potential compensatory usage while completing this test. Muscle testing specific to the lower portion of the serratus anterior may be completed with either a force applied to the humerus and lateral scapular border into adduction while the shoulder is elevated to 125° of scaption or an extension force while the shoulder is flexed to 125° [21, 36]. When completing any of these muscle tests, the use of a handheld dynamometer may help quantify their result [38]. The scapula must be watched or palpated to determine if winging occurs. Each of these positive tests may provide suggestion of the presence of serratus anterior dysfunction, but if winging occurs with the plus sign, an LTNP is present.

The presence, or absence, of SANP is suggested by flip sign and manual muscle testing of the middle and lower portions of the trapezius. Flip sign is performed with the patient standing, arm at the side, and elbow flexed to 90°, as the examiner manually resists glenohumeral joint (GHJ) external rotation while observing the scapula [1, 7, 9] (Fig. 13.5). A positive test is found if the medial scapula border "flips" off of the thoracic wall while the resistance is being applied [1, 7]. Visible atrophy of the trapezius and a depressed shoulder girdle are indicative of SANP; however, this must be correlated with muscle activity in order to fully examine trapezius function [1, 5, 7, 8, 12, 15]. For this, manual muscle testing of the middle and lower trapezius is completed in accordance with the standard procedures described by Kendall [7, 37] with or without use of a handheld dynamometer [38]. When testing the middle and lower trapezius, the examiner must palpate for muscle activation. In a complete SANP, both the middle and lower trapeziuses are completely flaccid, and no activation is noted of the rhomboid since it does not activate in appropriate test positions. The examiner must retract and posteriorly displace the scapula and then cue the patient to keep the arm in full external rotation. In cases affecting the proximal portions of the spinal accessory nerve, involvement of the sternocleidomastoid may be present in addition to the trapezius [7]. Manual muscle testing of the sternocleidomastoid should always be completed [7, 37].

As one moves through the examination algorithm and neural integrity is determined, the examiner attempts to correct the dyskinesis by hand placement, cueing, or "teaching." Often individuals with glenohumeral instability will initiate shoulder elevation with scapular depression/anterior tilt either by activation of the pectoralis minor or selective deactivation of the serratus. The patient is cued to bring the scapula/shoulder girdle into slight elevation and protraction. This will activate the serratus and deactivate the pectoralis minor eliminating the dyskinesis.

Principles of Rehabilitation

Once the scapular specialist recognizes the presence of LTNP and/or SANP, an objective baseline should be established with the results of the tests described above. Intervention should begin immediately and will vary based on the extent of the injury, stage of neural regeneration, and associated limitations. It is critical of the scapular specialist to educate patients with LTNP and/or SANP that the neural regeneration process cannot be expedited and a great amount of patience will be required. If we permit attempts to force recruitment of affected muscles, unnecessary stresses will be placed on the healing nerve, inappropriate compensatory strategies will develop, associated tissues will become irritated, and patients are likely to become frustrated. Instead, we must recognize our key principle to create an environment that encourages usage of available musculature to improve symptoms and increase function without placing excessive stresses on all involved structures.

Management of Associated Symptoms/Limitations

Due to the roles of the serratus anterior and the trapezius as static scapular stabilizers, the patient with LTNP and/or SANP should be educated about scapular, shoulder girdle, and spinal postures related to this injury. The orientation of the cervical, thoracic, and lumbar spine should be considered and emphasized with the use of a lumbar roll as appropriate. Specifically, excessive scapular protraction, anterior tilting, and internal rotation should be avoided due to decreased serratus anterior and trapezius activity [39, 40]. Often poor posture positioning results in overactivity of the pectoralis minor or levator scapulae/upper trapezius [8, 9, 26, 41]. Positions should be adjusted to minimize the effects of scapular malposition and shoulder drooping [2, 23, 25, 31, 32]. When in an upright position, resting the affected extremity on a pillow or supporting in a coat pocket may easily decrease the weight of the extremity to decrease gravitational stresses [6]. It is important to teach the patient how to replicate the nuances of his/her optimal positioning for consistent practice. In chronic, severe cases, an orthotic may be fabricated that allows support of the involved extremity, yet permits freedom of available movement [9, 16, 18] (Fig. 13.6).

Fig. 13.6 Orthotic for upper extremity support

In addition to postural adjustments, overactivity of accessory musculature may be addressed with thermal modalities, soft tissue techniques, and/or stretching [31]. Techniques such as cervical retraction, spinal mobilization, thoracic extension, and supine pectoralis minor stretching may be beneficial; however, care must be practiced to avoid separation of the involved side head and shoulder as to minimize the potential for negatively impacting neural regeneration. Activity modification will further assist in symptom management [6, 9, 12, 14, 18]. Movements, such as reaching or lifting, may result in excessive stresses to the affected, healing nerve and the associated involved muscle. Inappropriate compensatory strategies are more likely to develop, placing uninvolved structures at risk for developing symptoms of impingement or tendinosis [14, 31]. The scapular specialist should ensure full shoulder passive range of motion (PROM) is present [3, 5, 8, 9, 12, 14, 16]. If not, manual techniques, including GHJ and scapulothoracic mobilizations, should be administered. While doing so, caution should be practiced to avoid the possibility of nerve stretch during manual techniques like inferior glides of the GHJ. To improve PROM, the patient may also be provided a home exercise program (HEP) that includes chair stretch, supine passive forward elevation (FE), and passive ER stretch with a wand [9].

Table 13.1 Staged rehabilitation exercises for long thoracic nerve palsy

Stage 1
Scapular retraction
Scapular retraction with curls and triceps
Sidelying forward elevation
Prone, horizontal abduction exercise performed at 90°
Scapular retraction with external rotation using elastic band
Scapular retraction with row using elastic band
Scapular retraction with backhand using elastic band
Scapular retraction and elevation (toward the plane of the scapula)
Scapular retraction with forehand using elastic band
Stage 2
Serratus anterior isolation
Supine plus
Sidelying plus, supported arm lift with palm up
Standing plus with palm up on ball
Prone closed chain plus into ball
Serratus anterior isolation and plus with arm lift, palm up, and elbow bent
Serratus anterior isolation and plus with arm lift, palm up, and elbow straight
Serratus anterior exercise on wall
Serratus anterior exercise with elastic band
Quadruped plus
Flexion D1 pattern

Compensatory Strategies

An important aspect of rehabilitation for complete paralysis or significant weakness of isolated scapular muscles is improving compensatory and primary scapular muscle activity. In other words, improve the patient's ability to recruit the unaffected (compensatory) scapular muscles then target the primary affected muscle when activity is noted. Therefore, we can consider two stages of muscle activity training, one related to compensatory (unaffected) muscle activation and the other to primary (affected) activation (Tables 13.1 and 13.2). The exercises may be the same or similar when targeting compensatory and primary muscles depending upon reinnervation, and there may be overlap as to whether the focus is on the compensatory or primary muscles.

For patients with LTNP, the rhomboid and trapezius should be trained to maximize scapular stabilization in the absence of the serratus anterior [6, 9, 12, 16]. Simply teaching the patient to retract is essential and can be used to gain proximal stabilization during simple functional activities such as picking up a gallon of milk. Progressing to distal loading such as during row exercises may be included for generalized trapezius activation [31, 42]. The rhomboid can be best activated when tested with the shoulder at 90° of abduction and slight extension with humeral internal rotation and a force applied in adduction and flexion [43]. These muscles can each be further targeted with manual resistive

Table 13.2 Staged rehabilitation exercises for spinal accessory nerve palsy

Stage 1
Rhomboid isolation
Serratus anterior isolation
Scapular elevation
Rhomboid isolation/retraction with curls and triceps
Rhomboid isolation/retraction with row using elastic band
Rhomboid isolation/retraction with external rotation using elastic band
Standing plus with palm up on ball
Serratus anterior isolation and plus with arm lift, palm up, and elbow bent
Serratus anterior isolation and plus with arm lift, palm up, and elbow straight
Serratus anterior exercise with elastic band
Stage 2
Retraction
Supine middle trapezius isometric
Supine lower trapezius isometric
Scapular retraction with external rotation using elastic band
Scapular retraction with row using elastic band
Prone, overhead raise
Scapular retraction with backhand using elastic band

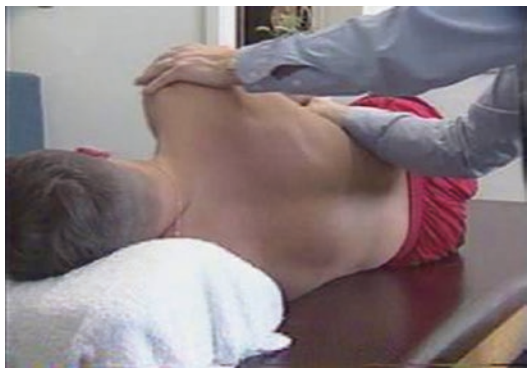

Fig. 13.7 Manual resistance to facilitate activity of the middle trapezius and rhomboid

techniques [32] (Fig. 13.7). Several authors have found favorable activation of the middle and lower trapezius with minimal upper trapezius or serratus anterior usage when completing sidelying ER, sidelying FE, prone horizontal abduction with ER, prone extension exercises, and prone

Fig. 13.8 Prone, horizontal abduction performed 90°

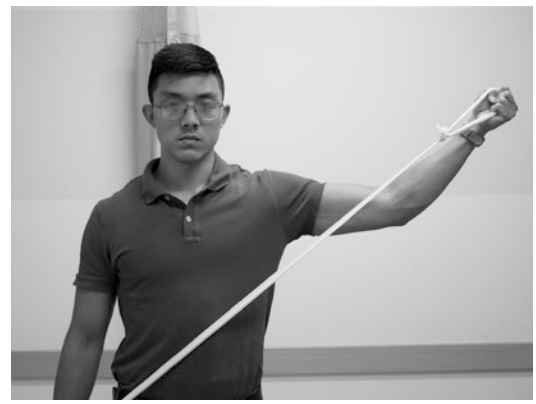

Fig. 13.9 Scapular retraction with backhand using elastic band

overhead raise [27, 29–31, 41, 42, 44–47] (Fig. 13.8). Once the rhomboid and trapezius are easily controlled by the patient, as evident by maintaining the corrected neutral scapular position can be held for at least 5 s, their usage can now be gradually integrated into larger, more challenging movement patterns [48] (Fig. 13.9).

Patients with SANP will require training of the rhomboid and serratus anterior to compensate for the trapezius [5, 10]. Serratus anterior isolation exercises should also be included, supine scapular protraction with plus, serratus anterior isolation and plus with arm lift, palm up and elbow straight, and quadruped push-up with plus exercises [42, 44, 47, 49] (Fig. 13.10). Manual resistance can be utilized, with specific attention to rhomboid activation. Once the patient can demonstrate proper control of the serratus anterior and rhomboid, larger and more complex movement patterns may

Fig. 13.10 Quadruped push-up with plus

Fig. 13.11 Serratus anterior exercise with elastic band

be also implemented provided the patient is maintaining a stable scapular base with rhomboid and serratus anterior (Fig. 13.11).

At this point, if pain is appropriately managed, full PROM is achieved, and if the patient is properly implementing compensatory movement strategies, the scapular specialist must wait for neural regeneration to occur with an observed return of activity of the affected muscle. This will be evident by reassessing the tests described above in 3–6 week intervals to monitor progress as the signs/symptoms improve [8]. Signs of neural regeneration should be evident within 12 weeks from onset of LTNP and/or SANP. In cases in which complete absence of serratus anterior and/or trapezius persists after 12 weeks, surgical intervention may be considered, especially if neurotmesis is suspected, but often a conservative approach is chosen for 6–12 months [8, 9, 15, 16]. It is important to reiterate that most cases of LTNP and SANP resolve within 24 months, without surgical intervention [9, 12, 14–18]. In order for the patient to be successful, a thorough education must be provided, understanding must be received, consistency with the prescribed HEP must be practiced, and patience must be observed as the time-dependent process of neural regeneration occurs.

Retraining Affected Musculature

Once signs of neural regeneration are present, primary training of the serratus anterior and/or trapezius can gradually begin. Exercises should be selected in which contraction of the affected muscle can be successfully achieved in order to foster proper motor learning [32]. These exercises should not be progressed until the appropriate completion of the activity is consistently demonstrated. To facilitate this motor control, a specific HEP is prescribed to be completed as often as every other hour throughout the day [32].

Activation of the serratus anterior with resolving LTNP is started with serratus anterior isolation exercise by asking the patient to protract and slightly elevate the scapula while keeping arms at the side in humeral ER when in a standing position [36] (Fig. 13.12). As greater motor control of the serratus anterior is gained, FE with a plus may be performed while lying on the uninvolved side then supine, first with a bent elbow, then

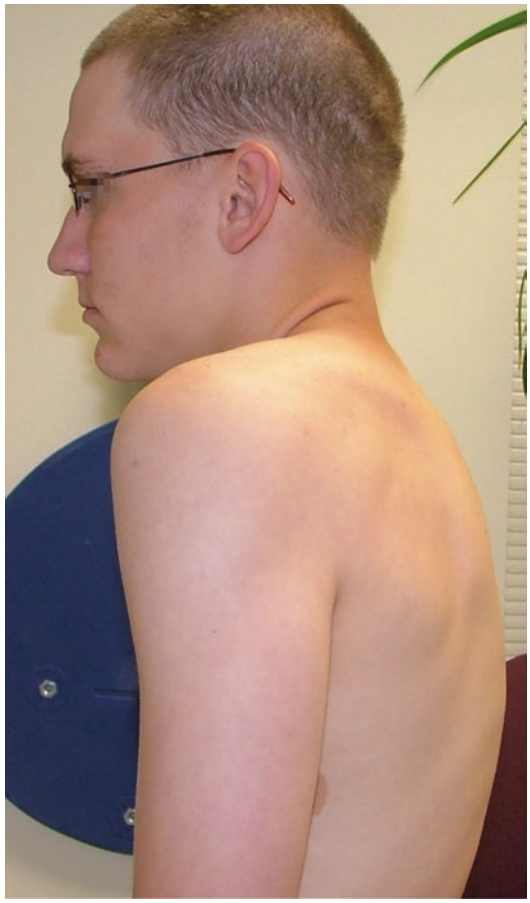

Fig. 13.12 Serratus anterior isolation

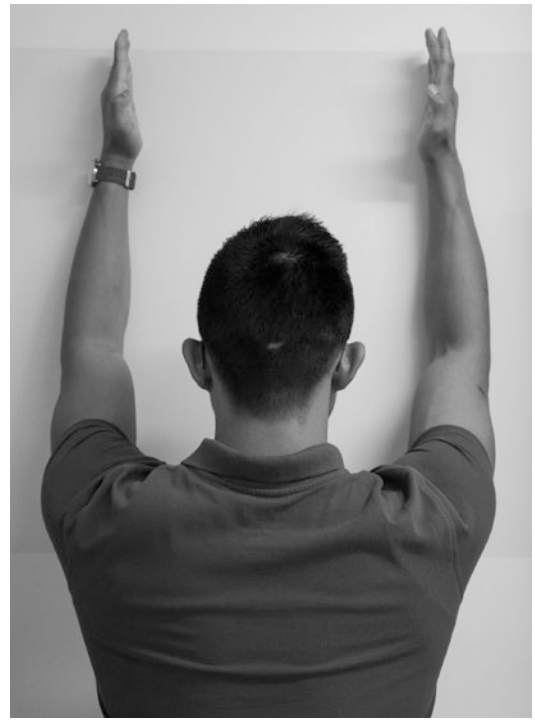

Fig. 13.13 Serratus anterior exercise on wall

Fig. 13.14 Flexion D1 pattern

with the elbow straightened to increase the lever arm. Rolling a ball forward with a plus on a table can be progressed to an inclined surface, then finally up a wall. Sliding the arm up the wall in the sagittal plane with ER will significantly activate the serratus (Fig. 13.13). Overhead FE with a plus is progressed to the vertical against gravity then with resistance. This can be progressed to supine scapular protraction, FE with a plus, D1 flexion, shoulder scaption to 125°, and push-up exercises with a plus [21, 42, 44, 49] (Fig. 13.14). As patients with SANP demonstrate neural regeneration, training of the trapezius may be started. Rowing can be completed as general trapezius exercise [42]. This can then be progressed to supine middle and lower trapezius isometric exercises, sidelying ER and sidelying FE with the focus of encouraging trapezius activity [29–31, 42, 44] (Fig. 13.15). Lastly, prone horizontal abduction with ER, prone extension exercises, and prone overhead raise against gravity may be completed [29–31, 36, 42, 44] (Fig. 13.16).

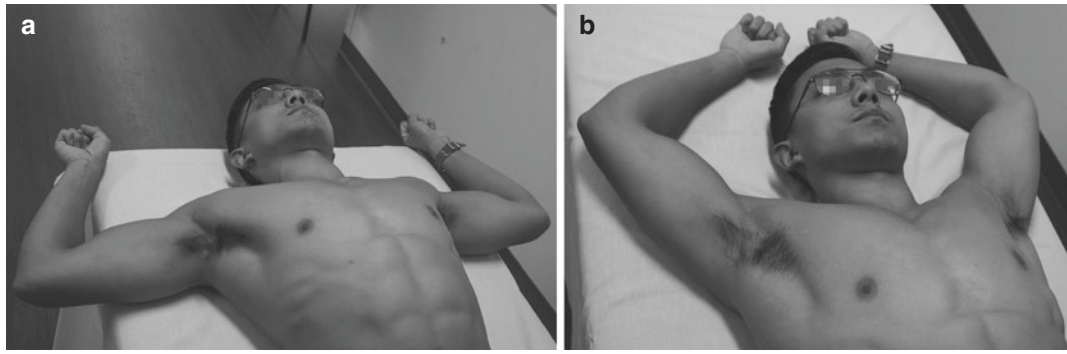

Fig. 13.15 Middle (**a**) and lower (**b**) trapezius isometric exercises

Fig. 13.16 Prone, overhead raise

Conclusion

Rehabilitation of patients with LTNP and/or SANP requires a systematic, algorithmic approach. Although we cannot accelerate the process of neural regeneration, scapular specialists can create an environment that encourages usage of available musculature to improve symptoms and increase function without placing excessive stresses on all involved structures. A specific, thorough assessment and regular reassessments are necessary to identify the involved structures and monitor the progress of neural regeneration. As signs of neural regeneration and muscle reactivation become apparent, gradual training of affected muscles may occur.

Appendix

See Tables 13.3, 13.4, 13.5, and 13.6.

Table 13.3 Compensatory strategies for patients with long thoracic nerve palsy

–	Exercise
Phase I	Prone row
–	Prone shoulder horizontal abduction with external rotation
–	Prone shoulder horizontal abduction with internal rotation
–	Prone arm raised above head in line with lower trapezius
–	Standing shrug
–	Shoulder extension at 30° of abduction
–	Seated row
–	Shoulder adduction with extension
–	Sidelying shoulder external rotation
–	–
Phase II	Prone extension
–	Prone shoulder abduction to 90° with external rotation
–	Sidelying shoulder flexion
–	Seated shoulder scaption to 80°
–	Low row
–	Seated shoulder abduction to 90°
–	Lawn mower
–	–

Rhomboid and trapezius should be trained to improve scapular stabilization in the absence of the serratus anterior

Table 13.4 Retraining affected musculature in patients with recovering long thoracic nerve palsy

–	Exercise
Phase III	Seated shoulder flexion to 125°
–	Seated shoulder scaption to 125°
–	Seated diagonal pattern (Shoulder flexion/horizontal adduction/ external rotation)
–	Seated high row
–	Wall push-up
–	Supine shoulder flexion to 90° with protraction
–	Elevation with resisted shoulder external rotation
–	–
Phase IV	Military press
–	Wall push-up with plus
–	Table push-up with plus
–	Elbow push-up with plus
–	Knee push-up with Plus
–	Floor Push-up with plus
–	–

Activation of the serratus anterior with resolving LTNP

Table 13.5 Compensatory strategies for patients with spinal accessory nerve palsy

–	Exercise
Phase I	Wall push-up with plus
–	Table push-up with plus
–	Elbow push-up with plus
–	Knee push-up with plus
–	Floor push-up with plus
–	Supine shoulder flexion to 90° with scapular protraction
–	–
Phase II	Seated shoulder flexion to 125°
–	Seated shoulder scaption to 125°
–	Military press
–	Shoulder extension at 30° abduction
–	Shoulder adduction with extension
–	Seated shoulder abduction
–	–

Training of the rhomboid and serratus anterior to compensate for the trapezius

Table 13.6 Retraining affected musculature in patients with recovering spinal accessory nerve palsy

–	Exercise
Phase III	Seated diagonal pattern (Shoulder flexion/horizontal adduction/ external rotation)
–	Lawn mower
–	Seated row
–	High row
–	Low row
–	Sidelying shoulder flexion
–	Sidelying shoulder external rotation
–	Elevation with resisted shoulder external rotation
–	–
Phase IV	Standing shrug
–	Prone shoulder abduction to 90° with external rotation
–	Prone extension
–	Prone shoulder horizontal abduction with internal rotation
–	Prone shoulder horizontal abduction with external rotation
–	Prone arm raised above head in line with lower trapezius
–	Sidelying shoulder external rotation
–	–

SANP demonstrate neural regeneration, training of the trapezius may be instigated

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Snapping Scapula Syndrome

14

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Abbreviations

ASES	American Shoulder Elbow Surgeons shoulder score—patient self-report section
CT	Computed tomography
et al.	et alii (and colleagues)
MRI	Magnetic resonance imaging
SANE	Single Alpha Numeric Evaluation
SSS	Snapping scapula syndrome
WORC	Western Ontario Rotator Cuff Index

Pathophysiology

Snapping scapula syndrome has previously been described as the pathological motion of the concave scapula over the convex thorax during movement of the shoulder girdle. Sound and palpable crepitus from the abnormal biomechanics of the scapulothoracic articulation may be amplified by the thoracic cavity [1]. The abnormal contact can occur not only because of predisposing anatomic abnormality but might also be the result of overuse in setting of a normal scapulothoracic articulation. Three main categories of etiology are thought to contribute to SSS: chronic bursitis, muscular dysfunctions, or anatomical abnormality [2].

Chronic overuse, especially in overhead athletes, in the absence of a predisposing anatomic abnormality produces inflammation of the bursa and surrounding musculature, which can cause reactive bursitis and subsequent scarring. There are six recognized bursae described about the scapulothoracic articulation, two major, which are generally considered physiologic, and four minor (or adventitial) bursae that are not consistently found and likely reflect pathologic motion [3]. The two major bursae are the infraserratus bursa between the serratus anterior and the chest wall and the supraserratus bursa between the serratus anterior and the subscapularis (Fig. 14.1). These bursae are well-recognized and have been repeatedly identified in arthroscopy as well as cadaveric studies. The four adventitial bursae are the supra- and infraserratus bursae at the superomedial angle of the scapula, a bursa at the inferomedial angle of the scapula, and a more superficial bursa between

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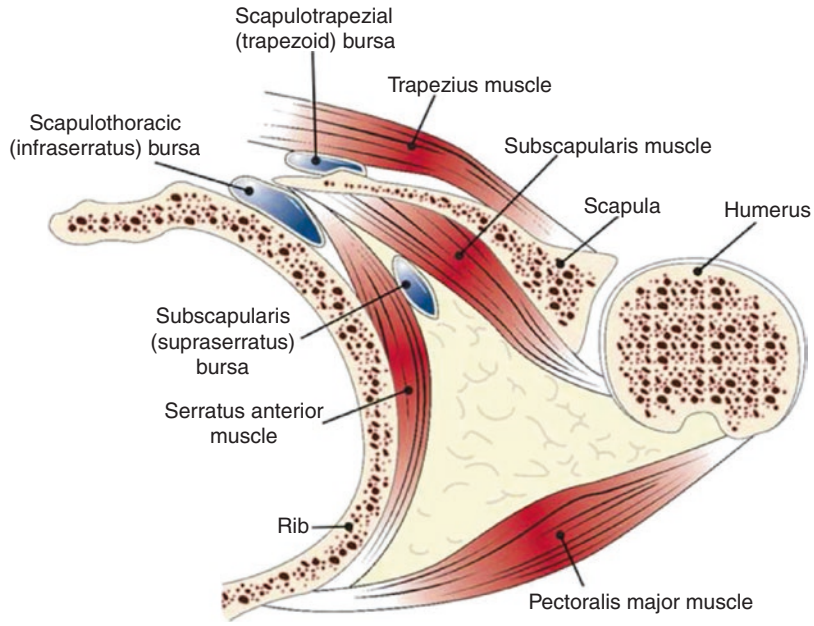

Fig. 14.1 Schematic demonstrating scapula anatomy in the axial plane. Two major bursae, the supraserratus and infraserratus bursae, the source of pathology in the majority of cases in the scapulothoracic bursitis, can be identified. (Reproduced with permission from Gaskill T, Millett PJ, Snapping Scapula Syndrome: Diagnosis and Management. *J Am Acad Orthop Surg.* 2013; 21(4): 214–224)

the medial scapular spine and the overlying trapezius. The most commonly involved bursae in SSS are those at the superomedial angle of the scapula [4]. In a chronic inflammatory setting characteristic of SSS, irritation causes bursitis and scarring in a cycle that is self-propagating leading to impingement, pain, and further inflammation. Bursal fibrosis with secondary scarring accounts for the pain and can even lead to mechanical and audible "snapping" symptoms in the absence of overt bony or soft tissues anatomic abnormalities [2].

Muscular dysfunction can also be attributable to SSS [5] and can take the form of either abnormal biomechanics, anatomical variants, or both. The synchrony of the periscapular muscles may be disrupted by muscle weakness, glenohumeral pathology, trauma, or iatrogenic nerve injury leading to a disruption of the force couples about the scapula and abnormal scapula motion, which can subsequently cause abnormal contact between the scapula and the thoracic cavity [3, 6]. In this setting, arm forward flexion may induce a posterior tilting of the scapula, thereby compressing the space between the inferior pole and the rib cage [7]. Conversely, abnormal anterior tilt due to a pathologically tight pectoralis minor muscle, for example, may compress the space between the superior scapula and the thoracic wall [8]. Atrophied or fibrotic muscle or anomalous insertions can produce abnormal scapular biomechanics potentially leading to painful "snapping."

Finally, soft tissue anatomic variations, including thinner muscle bulk of the subscapularis on the medial border of the scapula, may lead to a predisposition to abrasive forces against the rib cage [9, 10]. A superomedial bare area on the costal surface of the scapula between the origin of the subscapularis and the insertion of the serratus anterior has recently been described in a cadaveric study and may play a role in some cases [6, 11]. Skeletal abnormalities that contribute to SSS include anatomic variations, posttraumatic conditions, and mass lesions. A review of 89 cases of snapping scapula syndrome determined that a skeletal abnor-

mality was present in 43% of cases [12]. Anatomic variations are the most common subgroup and involve the bony structures of the scapula, thoracic cage, or the spine. A recent imaging analysis demonstrated that a "curved-type" scapular morphology, anteriorly angled medial border of the scapula, and decreased scapulocostal distance might be associated with SSS [13]. Luschka's tubercle, which is a prominence of bone or fibrocartilage at the superomedial angle of the scapula, has also been shown to contribute [14]. Additionally, removal of the first rib for treatment of thoracic outlet syndrome has led to SSS in some cases [15]. With regard to spinal abnormalities, excessive thoracic kyphosis or scoliosis may be implicated as the cause of an abnormal scapulothoracic articulation [4]. Despite these associations, many patients with anatomic variations are asymptomatic, so the clinical context of their presentation must be taken into account [16]. Posttraumatic conditions including malunion of the scapula or underlying ribs and reactive bone spurs from repetitive periscapular muscle trauma can also disrupt the normal articulation [9, 17–19]. Furthermore, musculoskeletal tumors such as osteochondromas, elastofibroma dorsi, and rarely chondrosarcoma can be the cause and must be excluded [20]. Osteochondromas in particular are the most common benign tumor of the scapula [21] and have been well-documented as a cause of SSS in the literature with one report accounting 16% of cases as due to these mass lesions [12]. Elastofibroma dorsi may specifically affect the ventral surface of inferomedial angle of the scapula causing a mass effect and abnormal biomechanics [22].

Clinical Presentation

Patients with SSS can present with a spectrum of complaints from mild discomfort to severely painful pseudoparalysis of the shoulder with an audible crepitus. This wide variety of presentations is largely due to the diversity of underlying causes.

History

Patients with scapulothoracic bursitis or snapping scapula typically complain of pain, palpable crepitus, and/or audible noise with arm movement, especially with overhead activities. These symptoms can significantly vary between individuals. As such, the patient should be questioned on the precise location, quality, and intensity of the associated pain or discomfort along with its chronicity, associated symptoms, and aggravating and alleviating factors. A family history of similar symptoms may be important as Cobey et al. suggested that there may be an inherited predisposition for scapular crepitus [18]. In addition, the patient's prior as well as desired type and level of activity should also be noted for appropriate goal setting and management of expectations.

Physical Examination

Physical examination should begin with a visual inspection of posture because significant kyphoscoliosis is known to reduce scapulothoracic congruity and may induce scapular snapping with or without painful bursitis [4]. Evaluation of the cervical spine should be performed in all patients to exclude a referred pain syndrome resulting from nerve compression between the C5 and C8 nerve root levels [23, 24]. Dynamic evaluation of both scapulae is then undertaken, noting any evidence of asymmetry, dyskinesis, winging, or audible snapping as the arms are moved through a range of active and passive motion. It is important to note that overhead athletes will often have depression, protraction, and downward rotation of their dominant scapula, which may be unrelated to their primary complaint [25].

Additionally, scapular dyskinesis is a common finding in patients with scapulothoracic bursitis and may be the result of unbalanced periscapular muscle kinematics such as weakness or tightness of the serratus anterior, trapezius, levator scapulae, or pectoralis minor muscles. Scapular winging can result from serratus anterior muscle weakness, most commonly caused by a long thoracic nerve palsy, or weakness or atrophy of the

trapezius muscle, which may be caused by a spinal accessory nerve palsy. Superomedially, tightness of the trapezius and levator scapulae muscle may present with neck stiffness and can be diagnosed via muscle length testing. Anteriorly, pectoralis minor tightness, which can result in scapular depression and protraction, can be diagnosed by visualization of the difference in the height of the shoulders off the examination table with the patient in a supine position. The affected shoulder girdle will rise higher off the table than the unaffected shoulder [26, 27]. In addition, an alternative method to assess pectoralis minor tightness in the same position is to place a hand on the anterior aspect of the affected shoulder and apply a moderate anteroposterior force. Significant resistance in flattening the shoulder against the examination table likely indicates a shortened pectoralis minor muscle-tendon complex. The presence of SICK (scapular malposition, inferomedial border prominence, anterior coracoid pain, and scapular dyskinesis) scapula in overhead athletes should alert the clinician to other associated diagnoses such as a glenohumeral internal rotation deficit (GIRD), posterosuperior impingement, or superior labral anterior to posterior (SLAP) tears, which may be contributing to snapping through scapular malpositioning or a dyskinetic pathophysiology [1].

Palpation of the periscapular region may reveal areas of localized tenderness consistent with adventitial infraserratus or supraserratus bursal inflammation. The superomedial angle and the inferomedial angle of the scapula are the most common locations for painful bursae [28]. Deeper palpation of these sites may be achieved by placing the arm in the "chicken-wing" position, in which the humerus is internally rotated and the dorsum of the hand is placed over the lumbosacral junction, a movement which tilts the scapula laterally [28, 29]. Some patients may be able to reliably produce scapulothoracic crepitus with provocative movements. In these cases, palpating the scapula while the patient performs these movements may help localize the site of pathology [30]. Additionally, applying posterior-to-anterior compressive forces over the scapular body during range of motion testing may also precipitate or accentuate crepitation between the scapula and the posterior thorax and exacerbate the patient's symptoms [31].

Periscapular muscle strength testing should also be performed on individual muscle groups to identify any weakness that may result in biomechanical force imbalance, scapular dyskinesia or winging, and subsequent snapping. The examiner should apply varying levels of resistance, and all resistance testing should be compared to the contralateral side. The trapezius musculature is evaluated by having the patient shrug the shoulders against resistance, while the levator scapulae and rhomboid musculature are best examined with the patient's hands on the ipsilateral iliac crests and subsequently having the patient force the elbows posteriorly against resistance. The serratus anterior muscle is tested by having the patient perform a wall push-up while the examiner simultaneously visualizes and palpates the medial border of the scapula. Weakness will exacerbate medial border prominence. The latissimus dorsi muscle can be isolated by having the patient push posteriorly against resistance with the arm at the side while the examiner palpates the inferomedial angle of the scapula.

Imaging

Radiographs

Standard radiographs should always be obtained when a diagnosis of snapping scapula syndrome is suspected. A combination comprising of true anteroposterior, tangential Y, and axillary views gives the clinician the best chance to exclude skeletal abnormalities. Despite adequate plain radiographs, bony anatomic abnormalities may still be missed [32].

Computed Tomography

When a skeletal lesion is identified on plain radiographs or suspected based on clinical exam, a CT scan, ideally including three-dimensional optimization, should be obtained to further characterize the lesion [32]. Routine CT scanning should be avoided in young patients unless indicated by radiographic evidence of an osseous or cartilaginous lesion that alters the congruency of the scapulothoracic articulation.

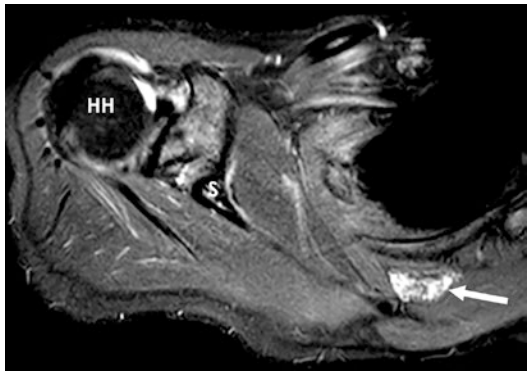

Fig. 14.2 Preoperative MRI (T2 weighted) of a patient with SSS showing inflamed fibrotic scar tissue between the superomedial scapula angle and the thorax (*white arrow*). *HH* humeral head, *S* scapula

Magnetic Resonance Imaging

Magnetic resonance imaging (MRI) is most useful to identify soft tissue structures that may be responsible for scapulothoracic crepitus or bursitis such as fibrotic scar tissue (Fig. 14.2), inflamed tissue, or musculotendinous disease. Additionally, tissue component analysis can be used to distinguish between malignant and benign soft tissue lesions [33].

Electromyograms

An electromyogram is indicated to evaluate the integrity of the nervous supply to the musculature in patients with unexplained scapular winging or periscapular muscle weakness. In particular, medial scapular winging may be caused by atrophy or weakness of the serratus anterior muscle following long thoracic nerve injury, and lateral scapular winging may be caused by trapezius muscle atrophy or weakness or spinal accessory nerve dysfunction. Whereas the majority of long thoracic nerve palsies are posttraumatic, the majority of spinal accessory nerve palsies are iatrogenic following neck or facial surgery [34]. Aberrant arthroscopic portal placement superior to the level of the scapular spine is an extremely rare cause of spinal accessory nerve dysfunction but should be considered in the appropriate circumstances [2].

Diagnostic Injections

In general, injections with local anesthetic agent and steroid can be both diagnostic and therapeutic. The temporary resolution of pain after the injection confirms the diagnosis of bursitis while also precisely localizing the pathological bursa. Despite a high likelihood of immediate success in these patients, the effect is rarely long-lasting although there have been reports of extended success [35]. The patient is positioned prone with the shoulder extended, internally rotated, and adducted in a "chicken-wing" position. The skin overlying the medial scapula is prepared in a sterile fashion, and the needle is inserted parallel to the anterior border of the scapula at the spot of maximal tenderness. Clinicians must be aware of the potential risk of intrathoracic penetration with an inappropriate vector of injection. Use of ultrasound has been described to aid localization of scapulothoracic injection with good results [36].

Nonoperative Treatment

With the exception of the situation in which there is a malignant mass lesion, a trial of nonoperative therapy is warranted regardless of the underlying of etiology of SSS. When caused by chronic overuse in the absence of anatomic abnormalities, nonoperative treatment should be attempted for 6 months to 1 year prior to considering surgery and can be expected to have high success rates [37, 38]. If symptoms are caused by an anatomic lesion, a trial of conservative treatment is still warranted [4]; however, the threshold for considering surgery is lower as surgical excision or correction of the abnormality has a high cure rate [14, 39]. A nonoperative protocol consists of activity modification, nonsteroidal anti-inflammatory medications, physical therapy, and therapeutic injections of steroids and/or local anesthetic into the inflamed bursae. With overuse and biomechanical imbalances being the major etiologic factors, the patient must initially modify activities to abate the cycle of bursitis and scarring.

Physical therapy should focus on periscapular muscle strengthening and improving shoulder girdle biomechanics. In cases where poor posture is contributory, training to minimize kyphosis, promote upright posture, and strengthen upper thoracic musculature is indicated. Because the scapula is responsible for static stability of the shoulder girdle, endurance training is crucial for scapular stability. This type of training comprises of low-intensity exercises with high repetitions. Strengthening of the subscapularis and serratus anterior reduces anterior tilt of the scapula alleviating bursal compression. Scapular adduction and postural shoulder shrug exercises are critical to strengthen the scapular stabilizers, including the serratus anterior, rhomboids, and levator scapulae. Specific beneficial exercises include scaption, press-up and push-up plus, rowing and machine rowing, and ball isometric scapular stabilization exercises. Abduction and elevation of the scapula cause increased pressure and strain on the underlying musculature and therefore should be avoided [40].

Operative Treatment

Indications

Surgical treatment is considered in patients who have failed nonoperative therapy. Surgery may provide more reliable results in patients who experience temporary relief with injections or in those patients with anatomic abnormalities contributing to their symptoms [38, 41]. In most cases, an arthroscopic approach may be successful; however, with larger mass lesions, open techniques may offer superior visualization and direct access and prevent the inadvertent spread of malignant cells. Arthroscopic treatment offers a quicker postoperative recovery and rehabilitation process [30, 42]. Specific surgical methods are variable depending on the individual patient's complaints and anatomic abnormalities, but typically surgery entails bursectomy of the pathologic bursa with or without partial scapulectomy of the superomedial scapula. Good results have been demonstrated with bursectomy alone in some reports [43]; however, partial scapulectomy with bursectomy is more commonly performed, particularly in the setting of mechanical crepitus [29, 44].

Arthroscopic Technique

Prior to surgery, the most painful areas should be confirmed with the patient to maximize success of surgery. These can be indicated with an indelible marker prior to induction of anesthesia in counsel with the patient. With regard to positioning, the patient is positioned prone with the nonoperative arm tucked to the side (Fig. 14.3a). The posterior thorax is draped widely, and the operative extremity is placed into a sterile stockinette. The dorsum of the operative hand is positioned on to the small of the back, effectively placing the glenohumeral joint into extension and near maximal internal rotation in the "chicken-wing" position. This position aids portal placement by increasing the potential space between the scapula and the chest wall. Additional separation may be accomplished by placing a medially directed force on the lateral shoulder to cause bayonet apposition of the scapular body. Bony landmarks are marked including the medial border and the spine of the scapula. Portals (Fig. 14.3b) are established 3 cm medial to the medial scapular border and kept inferior to the scapular spine to reduce the risk of injury to the main branches of dorsal scapular nerve and artery. This medial portal placement also allows a trajectory into the bursae that is more parallel to the chest wall, thereby decreasing the risk of thoracic penetration.

An initial viewing portal is made 3 cm medial to the inferomedial angle of the scapula, and a 30° arthroscope is introduced (Fig. 14.3b). Fluid pressure is routinely maintained at or below 50 mmHg. A second medial portal (Fig. 14.3b) is placed by triangulation, located 3 cm medial to the scapula just inferior to the medial confluence of the scapular spine. Once adequate visualization is established, a diagnostic bursoscopy is performed. The intercostal muscles and ribs are visualized inferiorly, the subscapularis is visual-

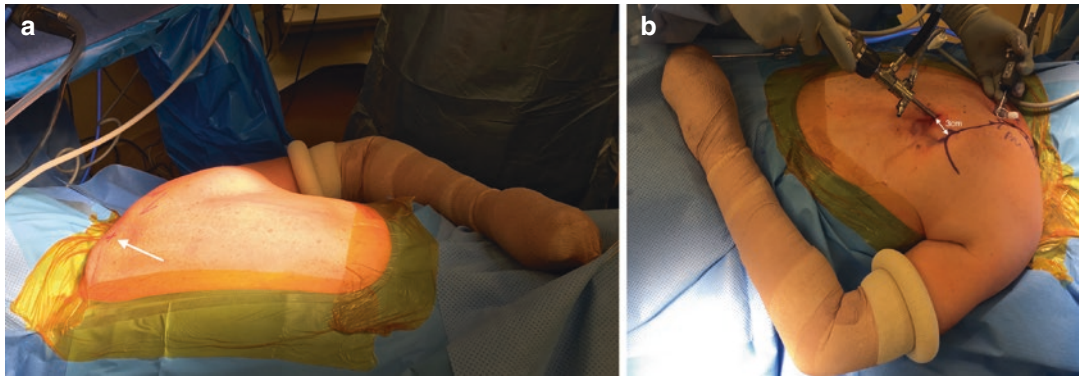

Fig. 14.3 (**a**) Intraoperative photograph of a right scapula and arm, placed in the "chicken-wing" position. Preoperatively, the point of maximum tenderness (*white arrow*) is marked. (**b**) Intraoperative photograph of the right scapula. The bony landmarks including the medial border of the scapula are marked. Portals are placed 3 cm medially to the scapula to minimize the risk of injury to neurovascular structures

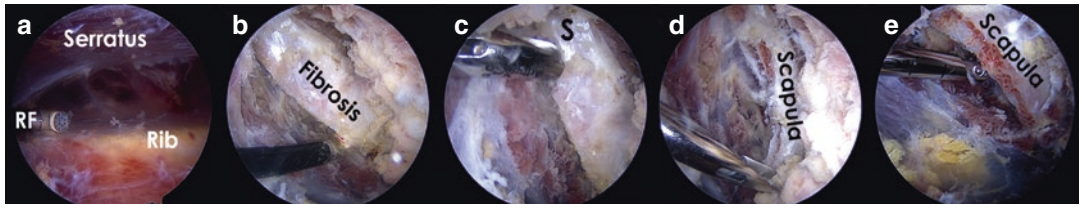

Fig. 14.4 (**a**) Arthroscopic image showing a radiofrequency (RF) device removing inflamed bursal tissue in the scapulothoracic space. Cranial is the serratus posterior superior muscle (serratus) and caudal the rib cage (rib). (**b**) Fibrotic scar tissue is resected with a radiofrequency device. (**c**) Next, an arthroscopic shaver is used to resect further soft tissue to release the margin of the scapula (S). (**d**) After completion, the superomedial angle of the scapula is visible and is partially resected. (**e**) Final picture after resection of the superomedial angle of the scapula

ized laterally, and the rhomboid and levator muscles are identified medially. A spinal needle is placed along the superomedial scapular border for additional orientation. Red muscle fibers of the subscapularis are not resected because a shaver or radiofrequency (RF) ablator is used to clear bursal tissue and fibrous bands in order to skeletonize the superomedial scapular border. Next, the supraserratus bursa is accessed similarly by bluntly penetrating the serratus posterior superior (Fig. 14.4a).

The superomedial angle of the scapula is exposed by removing the underlying muscular attachments with a radiofrequency probe or arthroscopic shaver (Fig. 14.4b, c). If crepitus or snapping of the scapula remains clinically evident after the superomedial angle of the scapula is exposed, spinal needles are placed to mark the extent of the planned resection. The arthroscopic scapuloplasty (Fig. 14.4d) is then performed with a high-speed bur, removing a triangular section of bone of approximately 2 cm (superior to inferior) by 3 cm (medial to lateral). The appropriate extent of resection is determined by removing the scapular border convexity as determined arthroscopically. A dynamic examination of the scapula should be routinely performed with the patient still under anesthesia to ensure adequate clearance and that residual mechanical crepitation does not persist. The suprascapular nerve can be at risk if this resection is taken too far laterally, and therefore arthroscopic instruments should proceed no further than the spinal needle placed to mark the extent scapular resection. The resection is visualized from both portals to ensure that it is smooth and adequate clearance has been achieved (Fig. 14.4e). Because the scapular bone is quite thin, a rasp is typically used to contour

resected edges. The arm is tested dynamically in a full range of motion, to ensure that no mechanical crepitation remains [31].

A superior accessory portal can be used to aid the resection of the superomedial scapula. This portal should be made at the junction of the medial one-third and lateral two-thirds of the distance between the superomedial scapular angle and the lateral acromion in order to protect the suprascapular nerve and artery. The trocar is advanced in a medial and caudal direction. The surgeon should stay in close proximity to the anterior aspect of the scapula to avoid thoracic penetration [44]. Finally, portals are closed routinely, and a sling is applied postoperatively (Table 14.1).

Table 14.1 Pearls and pitfalls of arthroscopic technique for scapulothoracic bursectomy and resection

	Pearls	Pitfalls
Portals	Place portals 3 cm medial to the medial border of the scapula	Placing portals too far laterally will risk injury to the dorsal scapular nerve and artery
	Place portals inferior to the scapular spine	Placing portals too far superiorly will risk injury to the spinal accessory nerve
Resection of bursitis	Perform resection at an angle that is roughly parallel to the chest wall	Too perpendicular of an angle may result in penetration of the thorax
Bony resection	Mark the most lateral end of the intended bony resection with a spinal needle and clear the bone prior to resection for adequate visualization	Carrying the resection too far laterally will put the suprascapular nerve at risk. Too much bony resection may result in disruption and dysfunction of the muscle
Bleeding	Decrease pump pressure and obtain good hemostasis prior to conclusion of procedure	Inadequate hemostasis can result in painful postoperative hematoma

Open Technique

Prior to surgery, the precise location of the patient's pain should be localized and marked for surgical planning, because the procedure may vary depending on the location of the patient's pain. The most common location for scapulothoracic bursitis and crepitus is the superomedial angle of the scapula [28], and as a result, open surgery will usually entail a vertical incision over the superomedial border of the scapula for the majority of patients (Fig. 14.5a). The patient is positioned prone, and dissection is carried down to the trapezius fascia and muscle which is split transversely in line with its fibers (Fig. 14.5b). Retraction of the trapezius muscle fibers cranially reveals the underlying rhomboids and levator scapulae inserting on the medial border of the scapula. Care is taken to subperiosteally elevate these muscles, as well as the supraspinatus and the subscapularis, from the superomedial angle of the scapula, thereby revealing the site of bony resection (Fig. 14.5c). The superomedial angle of the scapula is then excised with an oscillating saw and bursectomy subsequently performed (Fig. 14.6a, b). Typically, bone resection (Fig. 14.6c) totals 2 cm (superior to inferior) by 3 cm (medial to lateral). Following bony resection, the scapula is dynamically evaluated intraoperatively to confirm that there is no impingement between the scapula and thorax. Once the resection is deemed adequate, care is taken to repair the rhomboids and levator scapulae muscles through bone tunnels to the medial border of the scapula (Fig. 14.7a, b). The wound is then closed (Fig. 14.7c) in a standard, layered fashion [4, 30], and the patient is placed in a sling postoperatively.

For patients with bursitis at the inferomedial angle, which is the second most common site of scapulothoracic bursitis, incision is made obliquely over the inferior aspect of the scapula. Dissection is carried down to fascia after which an incision is made in the fascia and muscle in line with the fibers of the lower por-

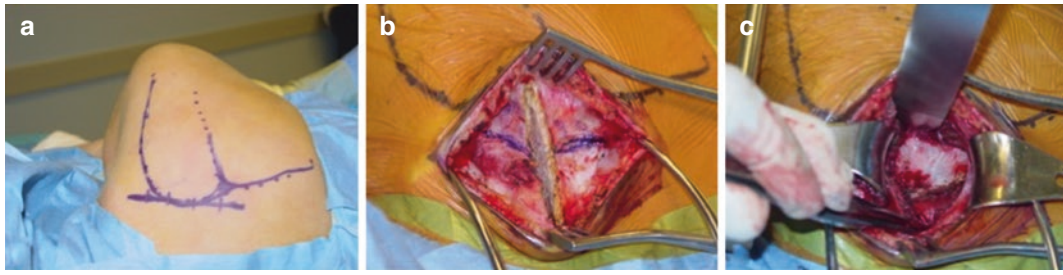

Fig. 14.5 (**a**) Intraoperative photograph illustrating bony landmarks of the superomedial border, spine of the scapula, and the medial border are marked on the skin. The site for a vertical incision over the superomedial border, for an open procedure to treat scapulothoracic bursitis, has also been marked. (**b**) The skin incision is retracted, and a dissection is performed down to the trapezius fascia and muscle, which is split transversely in line with its fibers. (**c**) After retraction of the trapezius muscle fibers cranially, and subperiosteal elevation of the underlying rhomboids, levator scapulae, supraspinatus, and infraspinatus, the site of bony resection is revealed

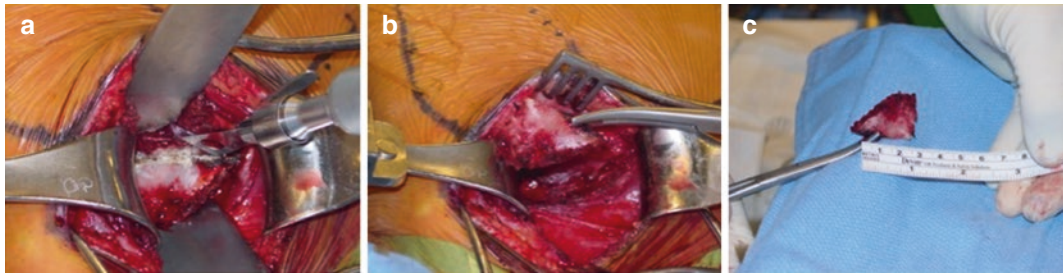

Fig. 14.6 (**a**) An oscillating saw is used to resect the superomedial angle. (**b**) The resected bony segment is retrieved. (**c**) A bony segment of typically 2 cm (superior to inferior) by 3 cm (medial to lateral) is removed

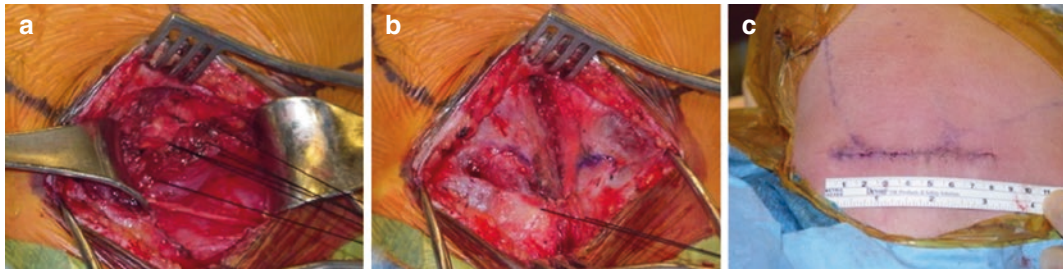

Fig. 14.7 (**a**) Following the resection, the rhomboids and levator scapulae muscles are carefully repaired through bone tunnels to the medial border of the scapula. (**b**) The wound is closed in a standard, layered fashion. (**c**) A closed incision of 8 cm in length is shown

tion of the trapezius and the latissimus dorsi. The bursa is thereby exposed and excised. Care is taken to remove any bony prominence at the inferior margin of the scapula as well. The wound is then closed in a standard fashion as above [4].

Postoperative Rehabilitation

The course of postoperative rehabilitation depends on whether the procedure was performed arthroscopically or open. For patients following an open resection requiring bone resection and

muscle repair, the shoulder is typically immobilized for up to 4 weeks to allow muscular healing. Passive motion is started shortly thereafter with emphasis on scapulothoracic mobilization; this is followed by active motion at 8 weeks and strengthening at 12 weeks [30]. Patients who have open surgery that does not require muscle repair through bone tunnels have a quicker rehabilitation course with passive motion started immediately postoperatively and active motion at approximately 3–4 weeks followed by strengthening as the patient tolerates [42]. Patients undergoing arthroscopic surgery have the fewest limitations following surgery and the quickest recovery. They wear a sling for 24–48 h and then begin both passive and active motion of the upper extremity as tolerated; early scapulothoracic mobilization is essential. Physical therapy immediately focuses on thoracic posture, scapular coordination, and strengthening. Full active motion following the arthroscopic procedure is expected by 1 week. Full recovery can be expected by 2–4 weeks postoperatively; however, return to sports and overhead activities should be delayed to 2 or 3 months postoperatively to enhance healing, even if the patient has seemingly achieved a full recovery prior to this point [30, 42].

Outcomes After Operative Treatment

Arthroscopic Techniques

Several studies have reported similar clinical outcomes after arthroscopic techniques when compared with open or mini-open approaches. In 1999, Harper et al. [45] were among the first investigators to use a technique for arthroscopic partial scapulectomy, where they reported excellent improvement in pain and function at a mean follow-up of 7 months in seven patients. Lehtinen et al. [43] evaluated 16 patients with either open or arthroscopic treatment of scapulothoracic bursitis; at 3-year average follow-up, 81% of patients were satisfied, SST was 9.8, and no statistical difference was found between the two techniques [43]. Later, Pearse et al. [39] reported the outcomes of 13 patients after arthroscopic bursectomy for scapulothoracic bursitis or osseous impingement and three of whom had an additional superomedial scapular resection. At a mean follow-up of 18.5 months, 9 of the 13 patients (69.2%) demonstrated improvement in pain and function with a median postoperative constant score of 87 (range, 58–95). Millett et al. [31] demonstrated an improvement in pain and function after arthroscopic bursectomy with or without scapuloplasty in a large series of 23 shoulders with a minimum 2-year follow-up. However, despite these improvements, median patient satisfaction was only 6 of 10 in this series. Two patients in this series did not undergo scapuloplasty, and although these two patients improved, they were less satisfied than those patients who had bony resection in addition to bursectomy. The authors postulated that arthroscopic bony resection may allow a more complete bursectomy to be performed. More recently, Blønd and Rechter [46] also showed measurable improvement in outcomes after arthroscopic bursectomy and scapuloplasty. At a mean follow-up of 2.9 years, 18 of 20 patients (90.0%) reported an improvement in pain and function over preoperative baseline values citing a median Western Ontario Rotator Cuff Index (WORC) improvement from 35.0 preoperatively to 86.4 postoperatively. Most recently, Menge et al. [47] demonstrated excellent results in 60 out of 74 shoulders (81%) that underwent scapulothoracic bursectomy and scapuloplasty. All outcome scores significantly improved from pre- to postoperatively: SF-12 PCS from 39.2 to 45.4, ASES score from 52.6 to 75.8, and QuickDASH from 40.2 to 24.2. Lower preoperative mental status score, longer duration of symptoms, and greater age were associated with lower postoperative outcome scores [47].

Open Techniques

Milch was the first to document the surgical technique and results of partial scapulectomy in three patients with snapping scapula syndrome

in 1950 [48]. Since this time, there have been numerous studies showing good outcomes after superomedial angle resection, especially in those with a predisposing anatomic variation or distinct skeletal lesions [7, 12, 43, 49–53]. Indeed, Arntz and Matsen [7] reported excellent results in 12 of 14 shoulders (86%) that underwent open superomedial angle resection for an abnormal bony shape or scapulothoracic incongruity. Of note, the investigators also histologically examined the resected bone and found no abnormalities consistent with the findings of other authors [28, 41, 50].

Symptomatic patients without radiographic or surgical evidence of an osseous abnormality may be candidates for bursectomy alone without resection of the superomedial angle. McCluskey and Bigliani [54] reported excellent outcomes in 8 of 9 shoulders (89%) after isolated supraserratus bursectomy. Nicholson and Duckworth [41] followed 17 patients for a mean of 2.5 years after open bursectomy, where 5 of the 17 patients (29.4%) received additional superomedial angle resection. The authors noted that superomedial angle resection allowed for a more complete bursectomy while also relieving osseous impingement. Symptom resolution occurred in all patients with significant improvement in American Shoulder and Elbow Surgeons (ASES) scores; however, the low numbers limited a comparative analysis between the groups.

Although less common, inflammation of the bursa at the inferomedial angle of the scapula can also occur and be the most problematic site for patients. Sisto and Jobe [55] reported excellent outcomes on four professional baseball pitchers who underwent open bursectomy at the inferomedial angle of their dominant scapulae and were able to return to pitching at the professional level without further problems.

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Scapula Fractures

15

Donald Lee and Schuyler Halverson

Scapula fractures constitute only 1% of all reported fractures, with less than 5% of shoulder fractures involving the scapula [1, 2]. The scapula provides a complex scaffold for a variety of muscular attachments, with each of the differing regions of the osteology presenting with its own fracture incidence and clinical significance. The primary planes of scapula fractures most commonly involve the scapular body (45%), followed by glenoid neck (25%), glenoid fossa (10%), acromion (8%), coracoid process (7%), and scapular spine (5%) [3]. As the majority of scapula fractures are adequately treated nonoperatively [4], the prevalence of fracture patterns requiring operative intervention is distinctly different than the overall prevalence, with operative fractures typically involving multiple fractures in the same periscapular region. An analysis of 90 operative scapula fractures showed 71% of operative fractures involve the superior-medial border of the scapular body, 68% involve the glenoid neck, 22% involve the spinoglenoid notch, 17% are intra-articular, 23% involve the scapular spine, and no isolated fractures of the acromion or coracoid were operative, although a series of operative acromion and coracoid fractures have been reported [5–8]. Of note, the superior-medial border of the scapular body is the most prevalent location for operative fractures as it is a common fracture exit point, not that a fracture through that location lends itself to any particular operative indication. These anatomic locations provide the framework for the subsequent report of fracture classification, treatment, and outcomes.

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Classifications

Scapular Body

The original 1996 OTA classification system for scapular fractures, along with a revised 2007 version, followed the OTA format of describing fractures as A, B, or C based on articular involvement [9, 10]. Due to the complex osseous anatomy of the scapula, with multiple articulations and processes, this classification failed to achieve widespread understanding, familiarity, and use. In order to address the limitations of their previous classification system, the OTA joined with the AO Foundation to develop a comprehensive system for in-depth classification of all scapular fractures by separating the scapula into three regions, the fossa, the processes, and the body, denoted F, P, or B, respectively (Fig. 15.1) [10–12]. The fossa includes the glenoid and the adjacent glenoid rim and neck lateral to the suprascapular notch. The processes include the acromion, which is defined

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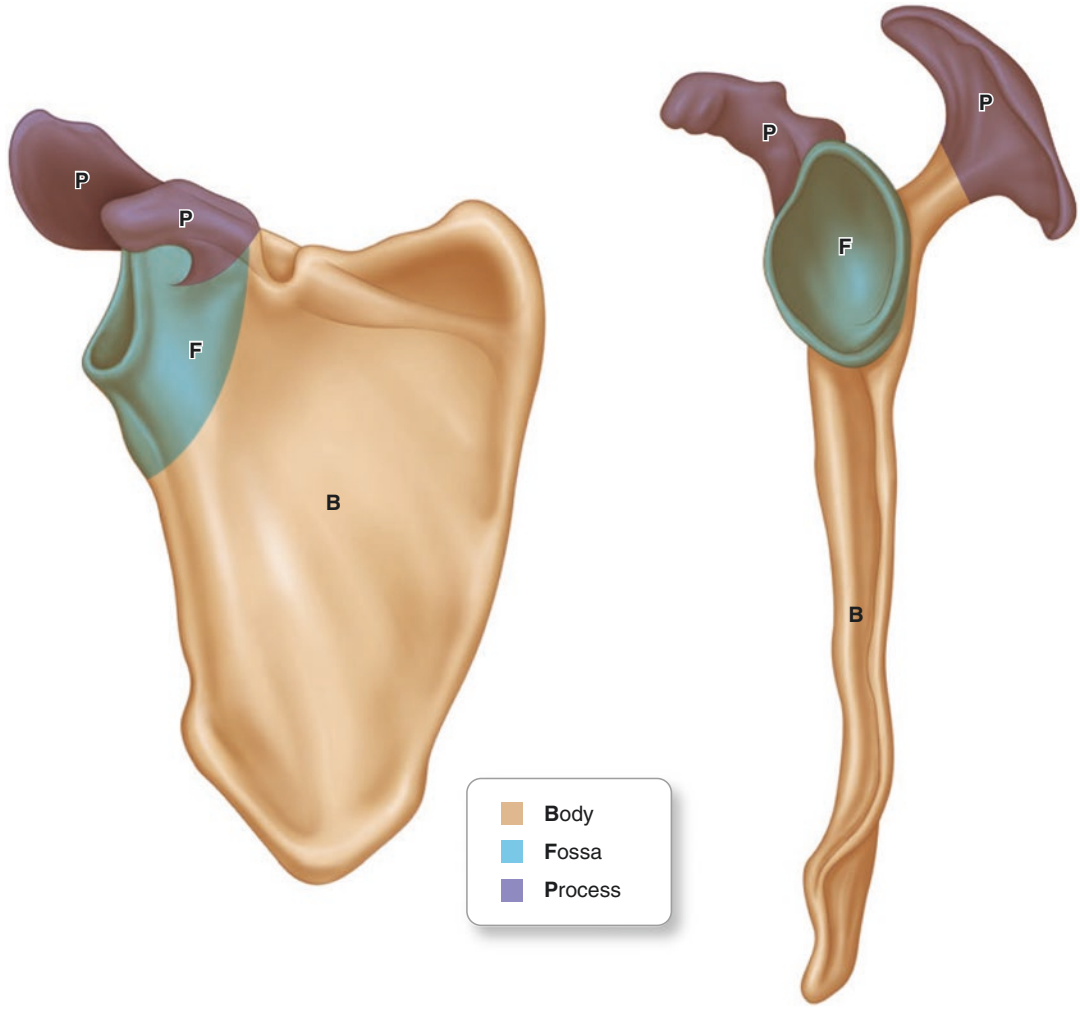

Fig. 15.1 OA/OTA classification system of fractures of the scapular body, fossa, and processes

as lateral to the plane of the glenoid, and the coracoid, defined beyond the superior limit of the glenoid. The body involves all scapula medial to a line parallel to the plane of the glenoid, starting cranially at the lateral border of the suprascapular notch. Body and fossa fractures are described by both a basic and focused classification.

In the basic classification system, body fractures are coded as B1 for simple fractures with two or less body fracture exit points and B2 for complex body fractures with three or more fracture exit points. In the focused system, the body is separated into the lateral (*l*) border (between the inferior articular rim and the inferior scapula angle), the superior (*s*) border (between the scapular notch and the superior scapular angle), and the medial (*m*) border (between the superior and inferior scapular angles), with the central (*c*) body having no border involvement, and the small area between the superior glenoid rim and lateral to the coracoid base designated as (*g*), glenoid side. The focused classification codes all involved sides in parenthesis following the scapular body code of B. For example, a fracture traveling between the medial and lateral border is coded B(ml).

Fossa involvement is classified in the basic system as F0 for an extra-articular fracture where the fossa is no longer attached to the scapular body. F1 fractures are intra-articular simple patterns of rim, transverse, or oblique fractures through the glenoid fossa. F2 fractures are intra-articular multifragmentary fractures. The focused classification system further describes fossa involvement based on the fracture pattern and location of involved articular

quadrants, as defined with respect to an equatorial line and the intertubercular line between the infraglenoid and supraglenoid tubercles. Simple rim fractures are denoted 1 for anterior, 2 for posterior, and 3 for simple transverse or short oblique. Fractures deemed 1 or 2 are further classified as "a" if infra-equatorial, "b" if involving superior and inferior quadrants, or "c" if involving both the anterior and posterior infra-equatorial quadrants, with 1 or 2 having been defined by the side containing the majority of the fracture fragment. Fractures deemed 3 are further classified as "a" if infra-equatorial, "b" if equatorial, and "c" if supra-equatorial. Complex fossa fractures are classified as 4 if there are more than two fracture line exit points and 5 for central fracture dislocations without exit points on the rim.

Fractures involving the processes are coded as P1 for coracoid, P2 for acromion, and P3 for both.

Fractures are then coded as a combination of all involved aspects of the fracture plane. For example, if the previously described B(ml) fracture had an additional fracture plane separating the glenoid from the scapular body, the coding would change to F0.B(ml). Adding fracture extension to the superior scapular body with a comminuted intra-articular fragment and coracoid fracture would be F2.B(mls).P1.

The AO/OTA system resulted in an 82% agreement overall between surgeons when discussing scapular body fractures with the basic system, with an overall kappa coefficient of 0.75. The simple classification has been shown to have interrater reliability kappa coefficient and overall agreement, respectively, of 0.57–0.59 and 49–82% for scapular body fractures, 0.79 and 90% for fossa fractures, and 0.49–0.53 and 72–81% for process fractures [11, 13, 14]. When the focused system was used to describe fractures involving the inferior, medial, or superior body borders, kappa coefficients were 0.73, 0.71, and 0.62, with overall agreement of 72%, 61%, and 5% [12]. The focused system for fossa fractures showed 86–100% agreement of intra-articular fractures and has been shown with three-dimensional CT analysis to adequately categorize clinically prevalent fracture patterns [11, 12, 15]. When comparing the new AO/OTA system with the previous OTA classification, it showed superior overall agreement and kappa coefficient of 81% and 0.53 versus 57% and 0.47, respectively [14].

Glenoid Fossa

The Ideberg classification [16], later modified by Heggland [17], classifies intra-articular scapula fractures (Fig. 15.2). Type 1 fractures involve the anterior glenoid rim, with type 1A having a fracture fragment of 5 mm or less and type 1B fragments greater than 5 mm. Type 2 through 6 all involve complete fractures through the glenoid, as opposed to just a rim fracture, and they differ based on where the fracture plane exists. Type 2 fractures exit inferiorly at the glenoid neck. Type 3 fractures exit superiorly at the base of the coracoid process. Type 4 fractures involve both the scapular neck and body, with a fracture plane running inferior to the spine of the scapula, ultimately exiting on the medial border. Type 5 combines the horizontal fracture plane of type 4, with the fractures of (a) type 2, (b) type 3, or (c) type 2 and type 3. Type 6 describes severely comminuted fractures of the glenoid fossa or a combination of type 1A and 1B. Type 1 fractures constitute the vast majority of glenoid fractures (85%, 50% type 1A, 35% type 1B) and have a strong association with dislocation (66%), subluxation (22%), additional skeletal injury (44%), or an additional nerve lesion (6%). The frequency of fracture types 2 through 5 is 3%, 1%, 6%, and 5%, respectively, with an additional skeletal injury rate of 66%, 100%, 0%, and 60%, respectively, a 33% subluxation rate for type 2 and no other reported dislocations or nerve lesions [18].

Glenoid Neck

Glenoid neck fractures are classified based not on a specific fracture pattern but rather on displacement of the fracture fragment (Fig. 15.3) [19]. Type 1 fractures are minimally or nondisplaced, while type 2 fractures are displaced greater or equal to 1 cm of translation and/or 40° of angulation [20, 21]. Description of glenoid neck fractures may describe fracture patterns as "anatomic neck," exiting superiorly lateral to the coracoid; "surgical neck," exiting medial to the coracoid; or "inferior neck," coursing inferior to the scapular spine, exiting along the medial scapular border.

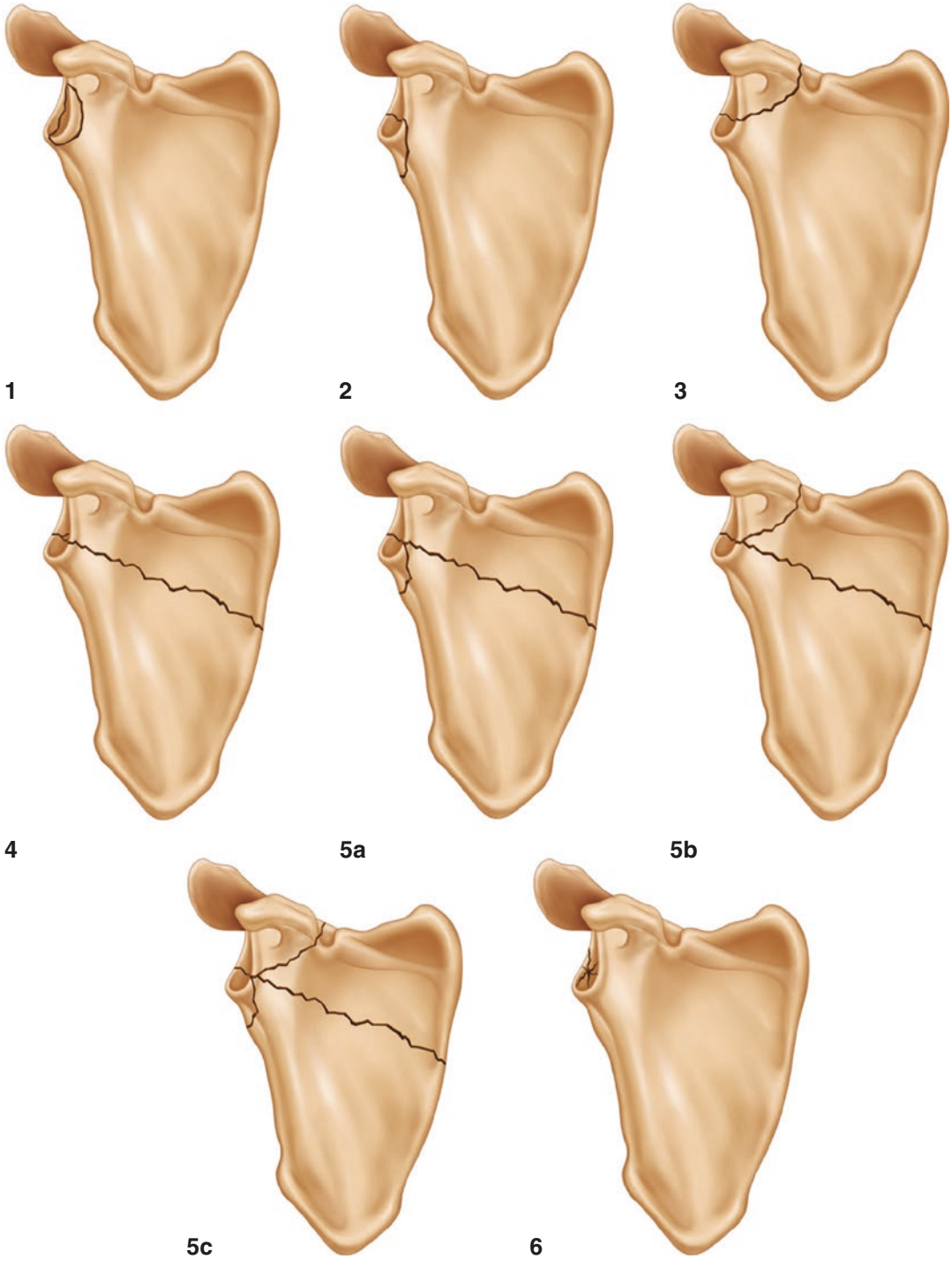

Fig. 15.2 Ideberg classification of glenoid fossa fractures

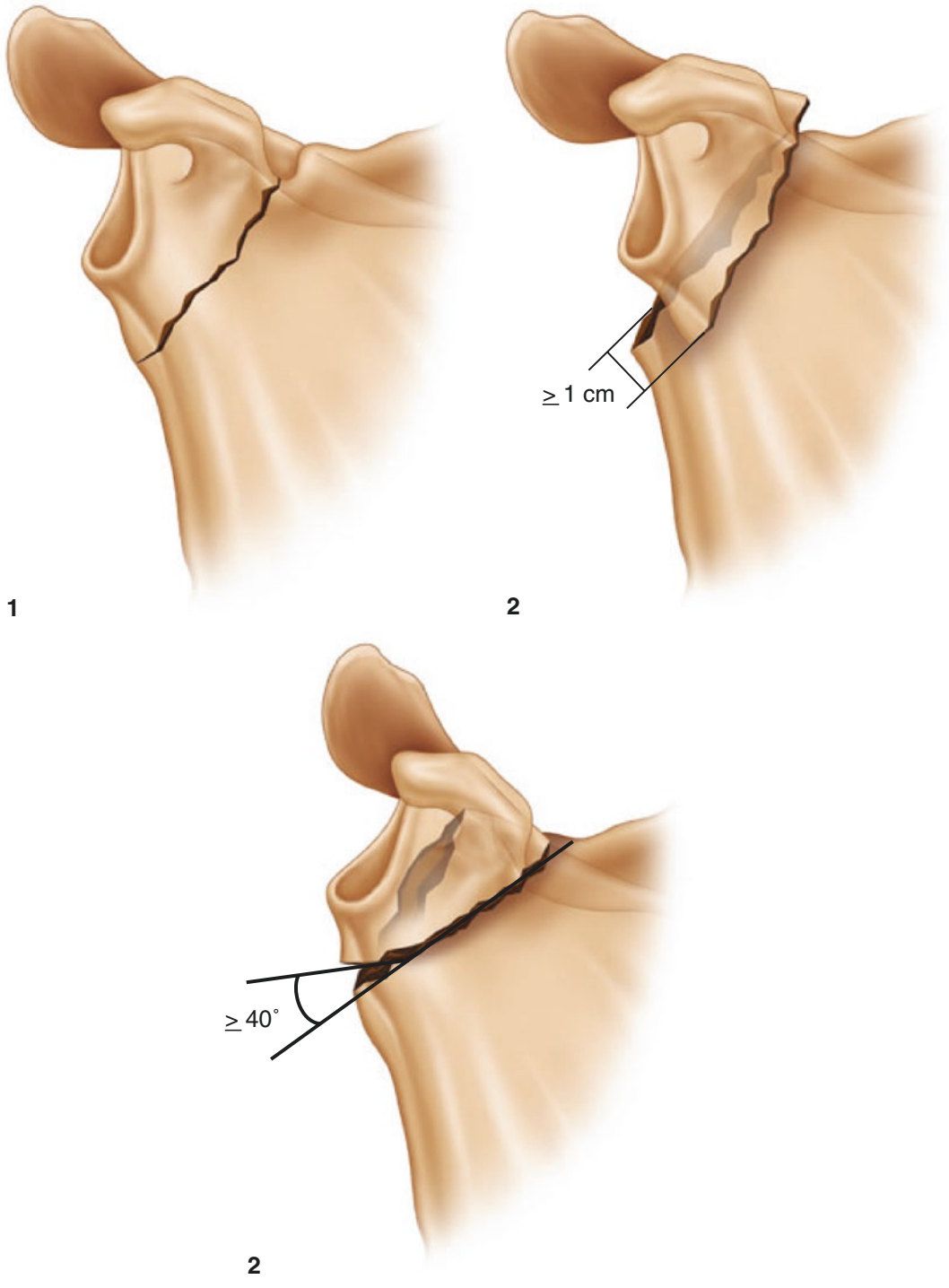

Fig. 15.3 Glenoid neck fracture classification

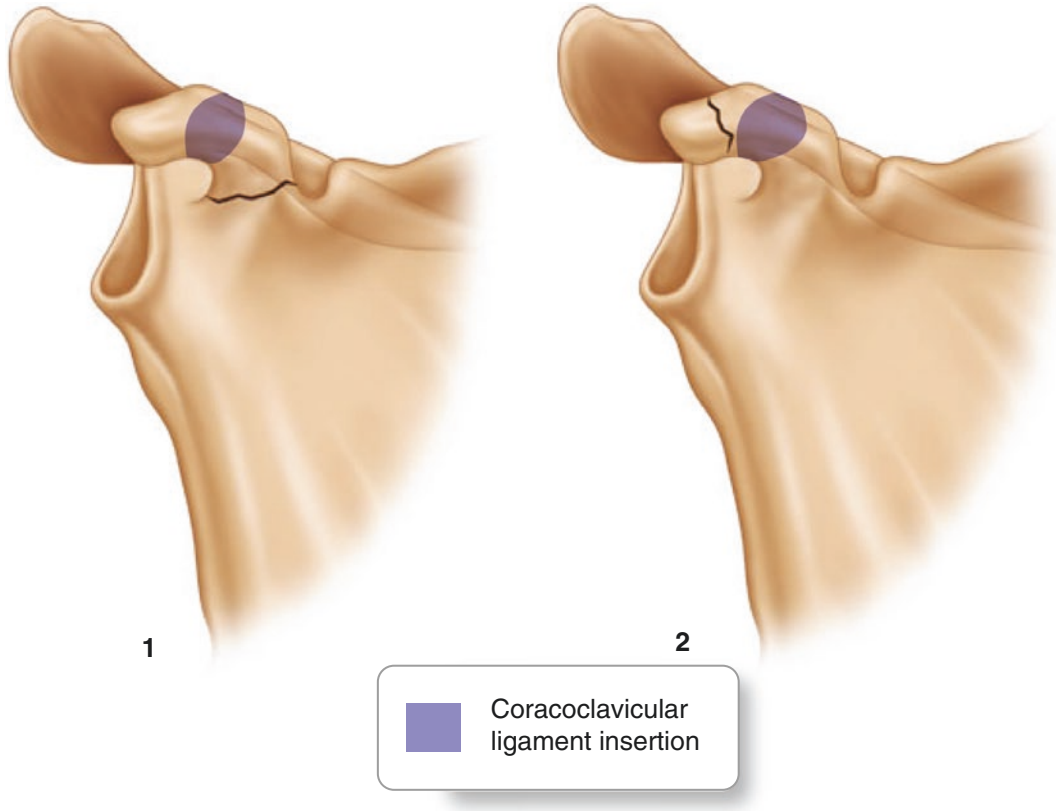

Fig. 15.4 Ogawa classification of coracoid process fractures

Coracoid Fractures

Isolated fractures of the coracoid have been classified by Ogawa et al., who originally proposed a five-type fracture classification, which was later simplified to have two fracture types based on their relation to the coracoclavicular ligaments [7, 22]. Type 1 fractures were located posterior to these ligaments, while type 2 fractures are anterior (Fig. 15.4). Type 1 fractures were notable for 32% association with the upper glenoid fractures and strong associations with shoulder dislocation and rotator cuff injuries, while type 2 had a strong association with distal clavicle fractures. Roughly 90% of coracoid fractures, regardless of type, were associated with acromioclavicular dislocation.

Acromion Fractures

Isolated acromion fractures were classified by Kuhn et al. into three groups (Fig. 15.5) [23]. Type 1 fractures are minimally displaced fractures, with type 1A being avulsion fractures resulting from muscle strain and type 1B resulting from direct trauma. Type 2 and type 3 fractures are displaced in any direction and differ in that they are without and with narrowing of the subacromial space, respectively. The original description of this classification also noted stress fractures as a possible injury, but this was not assigned its own type due to the significantly different mechanism of injury and due to the strong association between rheumatoid arthritis and acromial stress fracture, which is not applicable to the general population.

Using the Kuhn classification system, type 1A fractures usually resolve quickly without operative intervention, although nonunions were reported. Type 1B universally healed without deficits in shoulder function, although ipsilateral shoulder injuries may delay the healing process. Type 2 and type 3 fractures showed strong association with other injuries of the shoulder girdle or brachial plexus. Type 2 still did not require

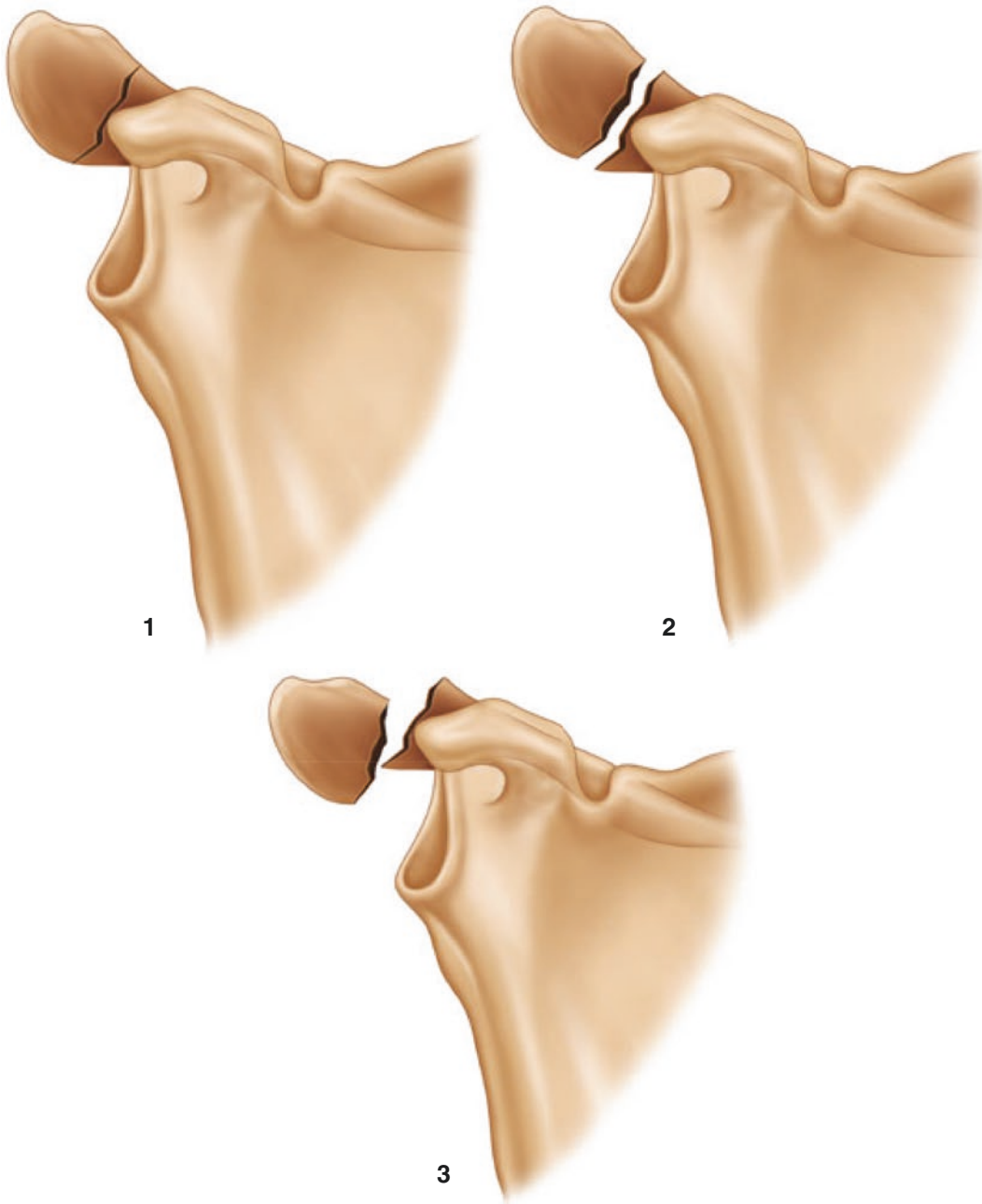

Fig. 15.5 Kuhn classification of acromion fractures

operative intervention, regardless of associated injuries, and all resulted in good clinical outcomes. Type 3 fractures are the only acromial fracture types with suboptimal expected outcomes, with all patients doing poorly due to a mechanical block to range of motion. Stress fractures, although not classified as their own type, typically fail conservative treatment and develop into painful nonunions.

Superior Shoulder Suspensory Complex Injuries and Lateral Scapular Suspension System Fractures

Fractures of the scapula are associated with clavicle fractures in up to 50% of reported cases [24]. The term "floating shoulder" was coined to

describe ipsilateral clavicle and glenoid fractures to imply an inherent instability. After progress in understanding the importance of the coracoacromial (CA), coracoclavicular (CC), and acromioclavicular (AC) ligaments, the definition of a floating shoulder was modified to require a double disruption of the superior shoulder suspensory complex (SSSC) (Fig. 15.6) [25, 26]. The SSSC describes multiple areas of the shoulder acting as a unit to provide stability. The SSSC is a ring composed of the distal clavicle, the AC ligament, the acromion, the glenoid process, the coracoid process, and the CC ligament. A formal classification system for the SSSC is not widely used, and diagnosis of SSSC disruptions is clinically difficult.

Recognizing that the fracture classifications described in previous sections rarely occur

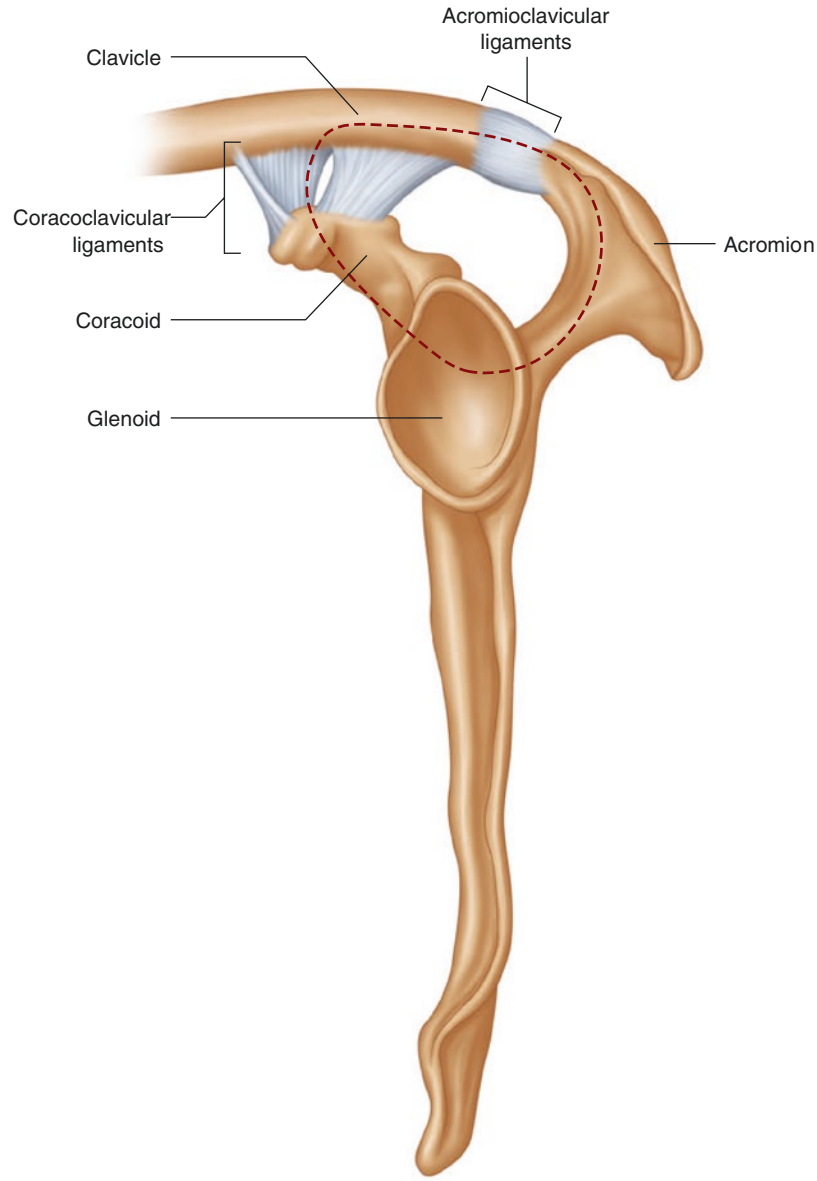

Fig. 15.6 Diagram of the superior shoulder suspensory complex (SSSC) classification

alone, the cumbersome nature of classifying a single patient's shoulder injury with multiple simultaneous classifications, and most importantly the inadequacies of the SSSC in describing the possible options for disruption of the distal clavicle versus AC ligament, Lambert et al. developed a focused classification system to address injuries to the lateral scapular suspension system (LSSS) (Fig. 15.7) [27]. The LSSS is composed of the distal clavicle, acromion, coracoid, scapular spine, and glenoid and is classified into three types. Type S0 injuries have an intact LSSS without failure of the overall support structure. Type S1 has incomplete failure of LSSS, simplified as an injury to a single component of the LSSS. This is further classified as S1a for a clavicle fracture lateral to the CC ligaments, S1b for an incomplete AC separation, and S1c for acromion fractures, scapular spine fractures, or a fracture at the base of the

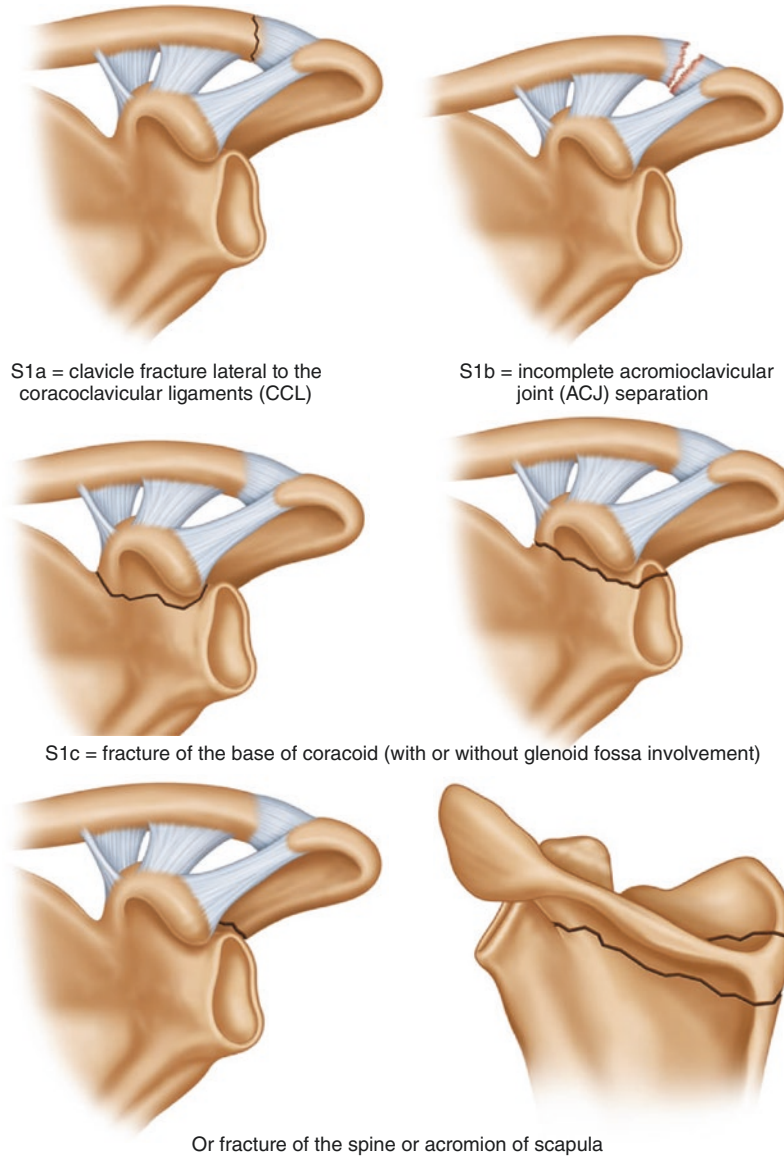

Fig. 15.7 Diagram of the lateral scapular suspension system classification

coracoid (with or without glenoid involvement). Type S2 injuries have complete failure of the LSSS or injuries at multiple locations. This is further classified as S2a for a clavicle fracture medial to the CCL, S2b for complete AC separation with CCL disruption, and S2c for a fracture at the base of the coracoid (with or without glenoid involvement) and a fracture of the acromion or scapular spine.

The LSSS classification system has shown overall agreement between shoulder specialists of 47%, with a kappa coefficient of 0.54 [27]. Use of this system to accurately distinguish between shoulder injuries with intact versus deficient LSSS was much better with an accuracy of 93% and a kappa coefficient of 0.63.

Associated Injuries

With the exception of rare avulsion injuries, a high-energy mechanism is required to cause a scapula fracture, which most commonly occurs from motor vehicle accidents in men aged 35–42 [3, 28–32]. Given the mechanism of injury, associated injuries have been reported in up to 95% of scapula fracture patients, who average 3.9 other major injuries [29, 30]. Multiple studies have investigated associated injuries, and results are often conflicting. Scapula fractures have been shown to be associated with injuries of the upper extremity, thorax, pelvic ring, neurovascular injury, chest and abdomen Abbreviated Injury Scale (AIS), and Injury Severity Score (ISS) [29–37]. Despite conflicting case series, the most comprehensive studies have shown several notable findings. Scapula fractures are consistently associated with a higher ISS, but when comparing to other patients with equal ISS without scapula fractures, besides an increase in ipsilateral upper extremity injuries and thoracic trauma, there is no difference in other injuries, hospital stay, or intensive care unit admission [29–31]. Additionally, scapula fractures may be associated with lower mortality compared to other multiply injured trauma patients, with a proposed mechanism that the scapula and its extensive muscular encasing provides protection for the underlying vital organs that is not present if the scapula isn't involved in absorbing direct trauma [38]. When mortality does occur in scapula fracture patients, the most common etiology is from pulmonary complications, typically underlying pulmonary contusions and respiratory splinting from rib fractures leading to a fatal pneumonia [30].

Radiographic Assessment

Assessment for suspected scapula fractures involves three primary radiographic views. A true anteroposterior view shows the glenoid, scapular neck, lateral scapular body and margin, and the scapular spine. The lateral view, or scapular Y view, shows the scapular body. The true axillary view shows the acromion, the AC joint, the coracoid process, and the anterior and posterior borders of the glenoid. A Velpeau view, although an acceptable replacement of the axillary view for assessing shoulder dislocations, does not visualize scapular anatomy as well as the axillary view. A standard chest film allows visualization of the medial scapular body and border, but was shown to not be an adequate independent screening tool for scapula fractures in children [39]. Although many additional named radiographic views have been published, few have proved to be clinically relevant. Bhatia views are an exception to this trend and help to visualize complex fractures of the coracoid via orthogonal views of the superior and inferior coracoid pillars [40].

The role of computed tomography (CT) in diagnosing scapula fractures is controversial. A comparison between surgeons' ability to diagnose scapula fractures based on standard radiographic views versus CT showed that inter-rater reliability of scapular fracture classification is not improved and in some fracture patterns even worsened when relying on CT as the primary imaging modality [41]. In the setting of complex fracture patterns or potentially operative scapula fractures, CT has been shown to provide useful information for both diagnosis and

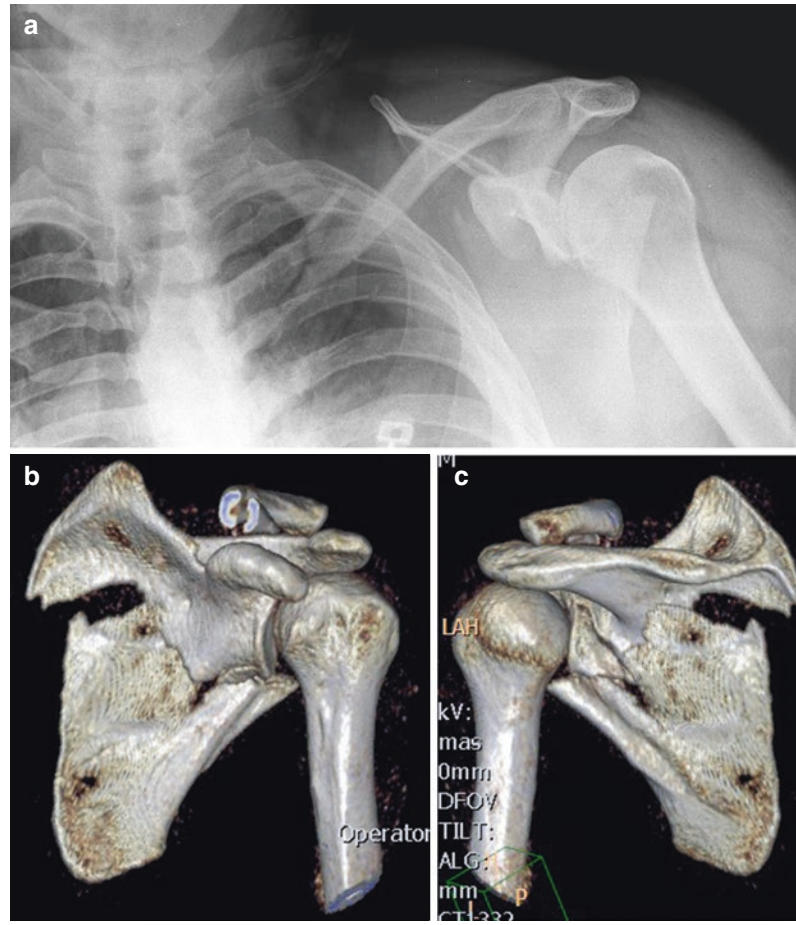

Fig. 15.8 (**a**) Anterior-posterior radiograph of a complex scapular fracture with associated mid-shaft clavicle fracture. (**b**) Anterior view of three-dimensional computed tomogram of same scapular fracture. (**c**) Posterior view of three-dimensional computed tomogram of same scapular fracture

surgical planning (Fig. 15.8a–c) [5, 42–46]. Considering the widespread use of CT scans for initial evaluation of high-energy trauma patients, this debate has grown to be somewhat irrelevant.

Surgical Indications

While the first operative fixation of a scapula was performed in 1910, it wasn't until roughly the 1990s when operative fixation indications became established and pursued, which was largely due to an improved understanding of fracture patterns and use of CT imaging [42, 47]. Nonoperative management of scapula fractures still remains an appropriate treatment for the majority of cases and typically constitutes sling immobilization for 2 weeks followed by active shoulder range of motion. Excellent functional outcomes have been shown in greater than 90% of scapular fractures, regardless of the anatomic areas involved [48–55]. Conservative management is not always successful, as symptomatic malunions, nonunions, and fracture displacement do occasionally occur [24, 56–59].

Multiple authors have worked to develop criteria for surgical interventions, but few go beyond expert opinion to established global indication guidelines with substantial evidential backing [1, 24, 60–62]. Accordingly, assessments of common practice patterns show poor correlation with published indications [63]. The only absolute indications for operative intervention are intrathoracic penetration of the scapular body between

Table 15.1 Indications for ORIF of scapula fractures

Intra-articular glenoid fractures	>25% glenoid involvement with humeral subluxation [70] or >5 mm articular step-off [71]
Extra-articular scapular neck fractures	40° angulation or 1 cm translation [24]
Extra-articular scapular body fractures	Significant displacement No consensus measurements
Acromial fractures	>1 cm inferior displacement, painful nonunion [23]
Coracoid fractures	>1 cm displacement, painful nonunion [72]
Disrupted superior shoulder suspensory complex	Double injury to the SSSC
Rare causes	Intrathoracic extension
	Open fractures

the ribs, and open fractures - both of which are exceedingly rare and will not be discussed below [64–68].

Bauer and colleagues appropriately summarize open reduction and internal fixation (ORIF) as the recommended treatment in grossly displaced fractures of the acromion, coracoid process, glenoid, and anatomic neck, or unstable fractures of the surgical neck, although they do not mention what defines "grossly displaced" [69]. Table 15.1 summarizes the best supported recommendations for operative fixation, with further discussion of each fracture type below.

Glenoid Fractures

As intra-articular fractures, glenoid fractures require operative intervention if they prevent congruent articulation (fossa fractures) or joint stability (rim fractures).

Operative fixation of fossa fractures serves to prevent posttraumatic stiffness and pain with shoulder motion [1]. Mayo [71] reported results in ORIF of glenoid fossa fractures with greater than 5 mm of articular displacement or any displacement resulting in humeral head subluxation. Included patients represented Ideberg I–V fractures, with 82% of patients reporting excellent or good long-term outcomes, whereas the other 18% of patients with unsatisfactory outcomes were largely related to associated injuries. These criteria have been validated by further clinical and biomechanical studies, while other sources state the criteria should be >2 mm or >3 mm [51, 61, 63, 73–77].

Rim fractures typically result from traumatic dislocation of the humeral head; therefore surgery is indicated when joint stability is threatened [1]. Measurements deemed to be indications for surgery include rim displacement greater than 10 mm, greater than one-fourth of anterior rim involvement, or greater than one-third of posterior rim involvement [70]. Although these indications were originally based on expert opinion, several studies have provided greater evidence to show acceptable outcomes using these criteria [1, 78–80].

Glenoid Neck Fractures

Fractures of the glenoid neck are inherently unstable, as the glenohumeral joint loses its suspension and accordingly no longer has a solid attachment to the axial skeleton. The debate of whether the glenoid medializes or the scapula lateralizes has largely been put to rest as several well-done studies have utilized axial imaging to show that the scapular body lateralizes [81, 82]. This scapular lateralization creates an impingement between the acromion and humeral head with arm abduction and alters the mechanics of the rotator cuff, resulting in near universally poor outcomes [1, 83, 84]. Multiple sources have independently proposed 1 cm as the maximum allowable glenoid neck displacement before debilitating impingement [20, 24]. Additionally, glenoid rotation greater than 40° in either the transverse or coronal plane resulted in significant pain and decreased range of motion and serves as another indication for operative intervention [24]. Assessing glenoid rotation on plane films is exceedingly unreliable and requires use of CT and potentially three-dimensional reconstruction.

Scapular Body Fractures

Isolated scapular body fractures are treated nonoperatively in 99% of cases, with 86% achieving a good to excellent functional outcome [48]. Multiple studies have identified no appreciable clinical difference between conservatively managed scapular body fractures and either the contralateral side or the general population, although symptomatic malunions do rarely occur [4, 51, 55, 85–88]. Consensus agreement on operative indications for scapular body fractures does not exist, but rather that they should be assessed on a case-by-case basis. Some sources cite 1 cm of displacement as an indication, while other sources show no functional deficits in conservative management of fractures displaced less than 2 cm and advocate for fixation of fracture displacement of >2.5 cm or angular deformity of >45° [20, 51, 89]. When operative scapular body fractures do arise, fortunately they have fantastic outcomes with the 1% of patients requiring ORIF in the previously mentioned series resulting in 100% achieving excellent functional outcomes [48]. Additionally, multiple rare indications exist for fixation of scapular body fractures, such as intrathoracic penetration, intra-articular penetration into the glenohumeral joint, malunion revision, and nonunion revision [64–67, 85–87].

Acromion and Coracoid Fractures

Few series describe operative management of fractures of the scapular processes, and no absolute set of indications has been validated. Proposed indications include extension of the fracture into the spinoglenoid notch, painful nonunion, displacement >1 cm in any direction, inferior displacement of acromion, or the presence of another ipsilateral scapula fracture requiring fixation [6, 7, 23, 62, 72]. The few studies providing clinical outcome data on operative process fractures report excellent outcomes, with all reported cases (excluding a single case study) going on to fracture union and recovery of painless full range of motion [6–8, 72, 90, 91].

Combined Fractures

All combined fractures are deemed operative if either one of the injuries is by itself operative or if in combination they disrupt the SSSC/LSSS. For example, combined glenoid neck and clavicle fractures require operative intervention when the CC ligaments are disrupted, or when the CC ligaments are intact, but the glenoid neck fracture meets the previously described operative indications of >1 cm displacement or >40° angulation. Isolated fixation of the clavicle is necessary only if the CC ligament is intact, and operative indications are met as described previously in Chap. 9 of this text. As another example, acromion and coracoid fractures in combination require fixation if the coracoid fracture is medial to the CC ligaments, making it an Ogawa type 2, as this would represent a double disruption of the SSSC.

Operative Intervention

Consistent among all scapular fracture types is the difficulty in finding cortical bone thick enough for operative fixation, as some portions of adult scapulae are less than 2 mm thick and not able to receive adequate screw fixation. Burke and colleagues mapped mean osseous thickness throughout the scapula, reporting bony thickness at the glenoid fossa of 25 mm, 9.7 mm at the lateral scapular border, 8.3 mm at the scapular spine, and 3.0 mm at the central scapular body [92]. Based on these measurements, the scapular regions with adequate bone stock for internal fixation are the glenoid neck, scapular spine, lateral scapular border, and coracoid process.

Method of internal fixation varies by fracture location. Glenoid neck fractures accept 3.5 mm pelvic contoured reconstruction plates or precontoured scapular plates along the posterior aspect of the glenoid and along the lateral border of the scapula. Another arrangement for neck fractures utilizes two separate plates, one along the lateral border of the scapular body and another along the scapular spine. Glenoid rim fractures utilize interfragmentary compression screws placed either percutaneously or via an open exposure, arthroscopic

soft tissue suturing, or fracture excision and bone grafting in the case of highly comminuted fracture not amenable to internal fixation. Glenoid fossa fractures are typically treated with interfragmentary compression screws or precontoured reconstruction plates. Double disruption of the SSSC may require precontoured clavicle plates along with abovementioned methods for the second area of disruption. Acromial fractures can be repaired via tension band technique using cannulated screws or via precontoured acromial plate fixation. Coracoid fractures can be treated with interfragmentary screw fixation or if the fragment is too small to tolerate fixation, via fragment excision.

Surgical Approaches

Although many modifications exist, the workhorse approaches for operative fixation of the scapula are the anterior, posterior, superior, and arthroscopic approaches. Table 15.2 lists recommended approach based on fracture location. In general, the patient is positioned in a lateral decubitus position, allowing for a combined anterior and posterior approach to the shoulder. Fluoroscopy can be positioned directly over the patient for intraoperative visualization of the fracture and hardware placement (Fig. 15.9). If only an anterior approach is needed, then the patient is placed in either a supine or beach chair position.

Table 15.2 Recommended approaches by fracture location

Intra-articular glenoid fractures	
– Anterior glenoid fossa	Deltopectoral
– Superior glenoid fossa	Superior deltoid split
– Posterior glenoid fossa	Posterior
Extra-articular scapular body fractures	
– Inferior glenoid fossa	Modified Judet
– Lateral scapular body	Modified Judet
– Central scapular body	Standard Judet
– Scapular spine	Posterior
– Multiple scapular borders	Standard Judet
Acromial fractures	Posterior extended toward acromion
Coracoid fractures	Deltopectoral
Clavicle fractures	Superior parallel to fracture

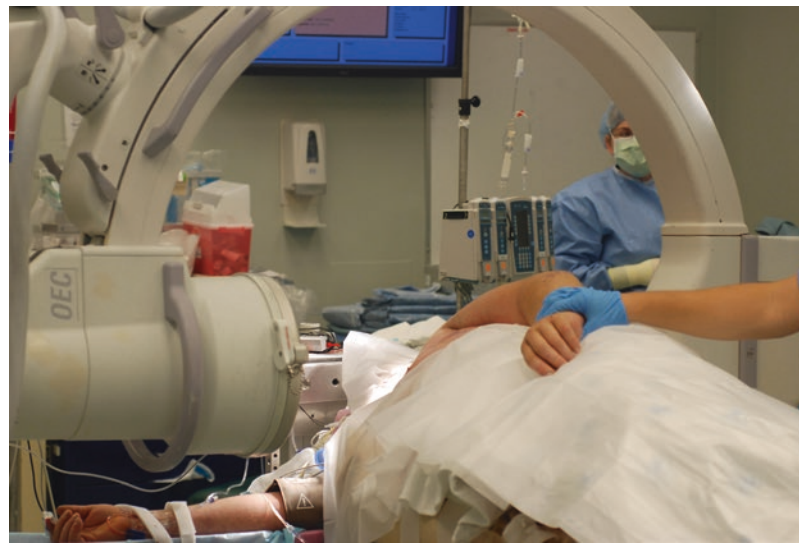

Fig. 15.9 The patient is positioned in a lateral decubitus position using a beanbag positioner. C-arm fluoroscopy can be brought over the patient for intraoperative visualization of the fracture and hardware placement

Anterior Deltopectoral and Transverse Clavicle Approach

The anterior deltopectoral approach (Fig. 15.10a) can be utilized for fractures of the anterior glenoid rim (Ideberg Ia), intra-articular glenoid fossa, superior glenoid fossa, coracoid fractures, and Ideberg III with associated clavicle fractures. The skin incision starts superior to the coracoid process, near the midclavicle. It then extends distally, laterally, and obliquely over the deltopectoral interval toward the deltoid insertion. As the deltopectoral intermuscular plane is developed, the cephalic vein is retracted medially or laterally, the latter of which is preferred due to the fracture work being directed medially. The clavipectoral fascia is incised along the lateral edge of the conjoined tendon proximal toward, but not through, the CA ligament. If needed, the coracoid is exposed at this point. The anterior-inferior humeral circumflex artery should be identified and ligated. Release of the subscapularis off of the lesser tuberosity exposes the underlying anterior joint capsule, which is elevated along with a sleeve of periosteum to expose the underlying glenoid. Visualization is improved by releasing the rotator interval [93]. For clavicle fractures, a transverse incision (Fig. 15.10b) is created just inferior to the long axis of the clavicle and center over the fracture site. Care is taken to protect the supraclavicular nerves, the platysma is incised, and a subperiosteal approach to the clavicle is performed (Fig. 15.10c).

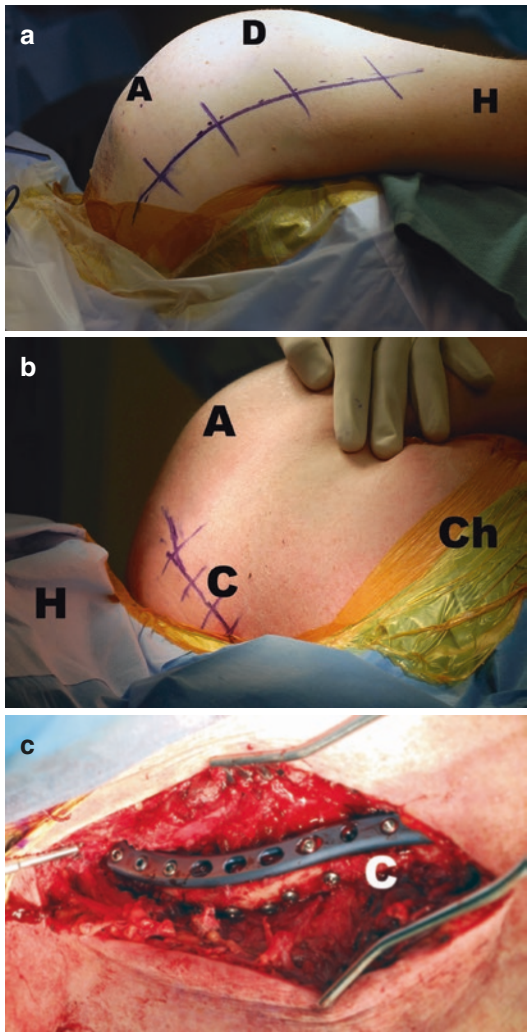

Fig. 15.10 (**a**) An incision for an anterior deltopectoral incision is outlined (*A* acromion, *D* deltoid, *H* humerus). (**b**) An incision for operative fixation of a clavicle fracture is outlined (*H* head, *C* clavicle, *A* acromion, *Ch* chest wall). (**c**) Intraoperative photograph following fixation of clavicle fracture (*C* clavicle)

Posterior Approach

The posterior approach (Fig. 15.11) allows access for posterior glenoid rim fractures (Ideberg Ib), intra-articular glenoid fossa fractures (Ideberg II–V), glenoid neck fractures,

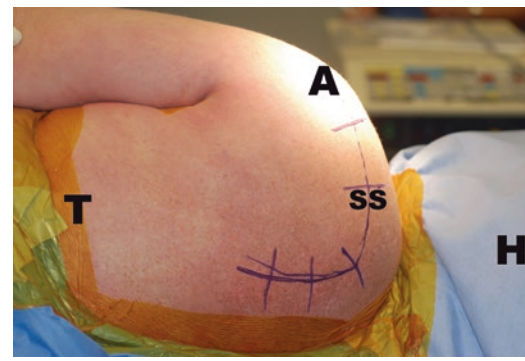

Fig. 15.11 An incision for a posterior approach to the scapula is outlined (*H* head, *A* posterior acromion, *SS* scapular spine, *T* thorax)

acromial fractures, and scapular body and scapular spine fractures. Incision is made from the posterolateral corner of the acromion horizontally, running parallel to the scapular spine before curving vertically to head along the medial scapular border. Elevation of the deltoid, trapezius, and their overlying fascia off of the scapular spine is performed carefully, as this is utilized for later repair. The medial border of the scapula is exposed by incising the fascia overlying the plane between the rhomboids and the infraspinatus/teres minor. Access to acromial fractures requires extension of the incision further anterior and lateral. The interval between the deltoid and the infraspinatus is developed inferior to the scapular spine [93].

The Judet approach elevates the infraspinatus and teres minor off of the infraspinatus fossa from medially to laterally, allowing visualization of the scapular body and scapular neck (Fig. 15.12) [94]. The modified Judet allows for exposure of the glenoid neck via the interval between the infraspinatus and teres minor [95]. Additionally, exposure of intra-articular glenoid fractures is possible via tenotomy of the infraspinatus and teres minor, if needed, and posterior capsulotomy. Direct comparison between the modified Judet and the standard Judet showed that both allow for exposure of the full medial and lateral borders, with the modified Judet exposing only 20% of the surface area exposed by the standard Judet [96]. The majority of this difference is due to the near complete dissection of the infraspinatus from its fossa in the standard Judet approach, which offers essentially no benefit due to the excessively thin nature of the underlying bone, which does not allow for screw fixation. A case example following operative fixation of a scapular body fracture (Fig. 15.8a–c) and clavicle fracture using a Judet approach combined with an anterior-superior approach (Fig. 15.13a–i) is shown.

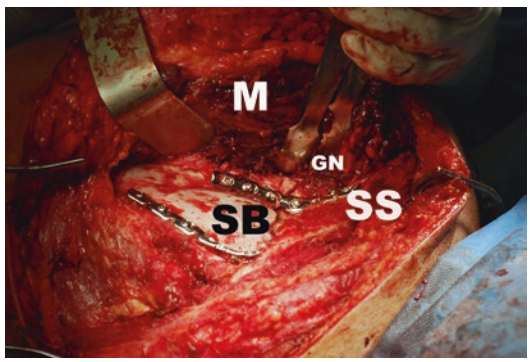

Fig. 15.12 Intraoperative photograph of a Judet approach to the posterior scapula. The infraspinatus and supraspinatus muscles (*M*) have been elevated (*SS* scapular spine, *SB* scapular body, *GN* glenoid neck). Contoured pelvic reconstruction plates have been used to stabilize the scapular body fracture

Superior or Anterior-Superior Approach

The superior or anterior-superior approach is used for coracoid fractures, superior glenoid fossa fracture involving the coracoid process, acromial fractures, and clavicle fractures. The skin incision can be a transverse incision inferior and parallel to the clavicle for clavicular fractures (Fig. 15.8b, c) or a saber-cut incision along Langer's lines over the area of injury. The deltoid is split between the anterior and middle thirds, and if needed, a portion of the deltoid can be reflected off the anterior aspect of the acromion and clavicle for exposure. The glenoid is exposed by opening the rotator interval [93].

Athroscopic

The use of arthroscopy has proved beneficial for glenoid rim fractures (Ideberg Ia, Ib, and VI) utilizing standard posterior and anterosuperior shoulder arthroscopy portals [97]. Given the capsuloligamentous structures typically attached to fracture fragments, nonabsorbable suture can be passed through the labrum to allow manipulation,

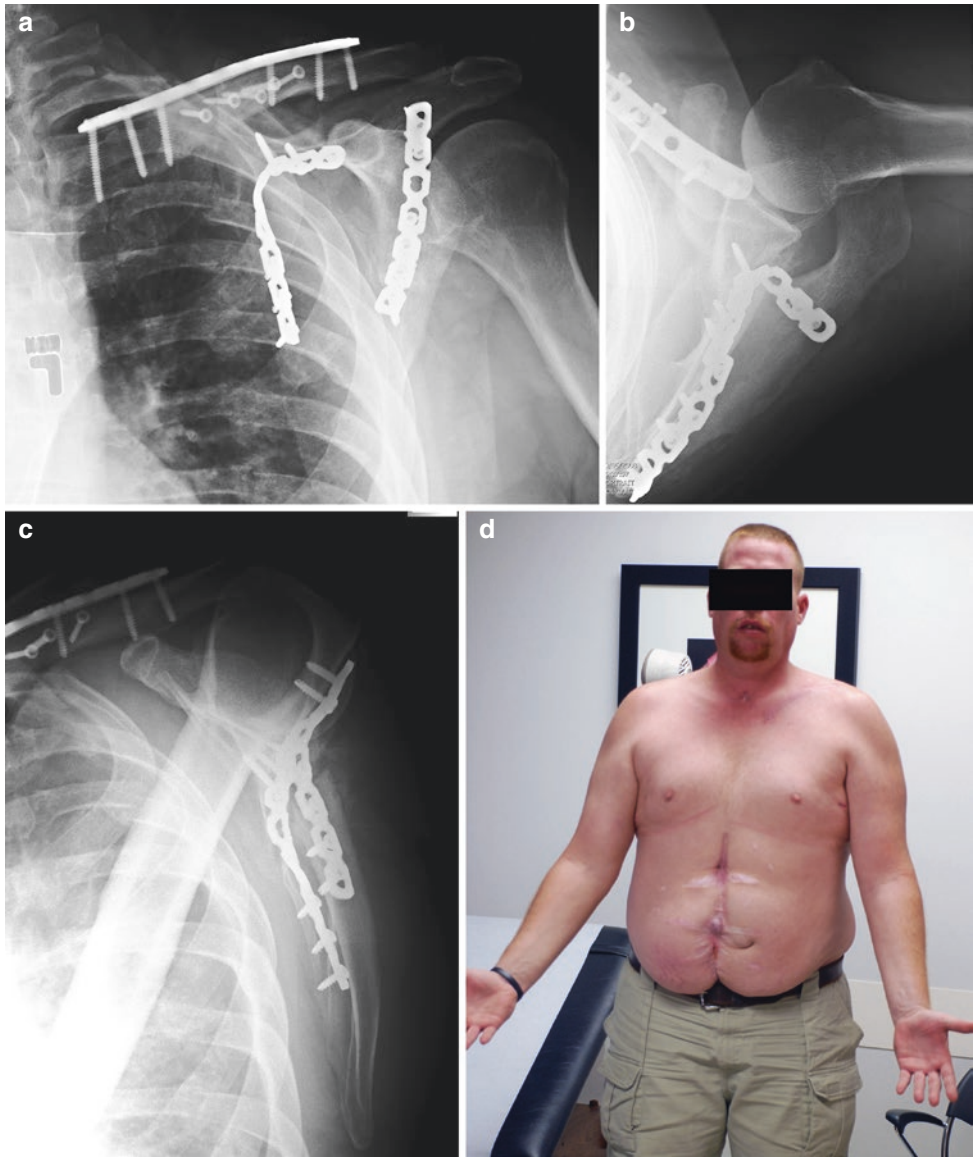

Fig. 15.13 (**a**–**c**) Anterior-posterior view (**a**), axillary (**b**), and scapular Y (**c**) views of shoulder following fixation of a scapular and clavicle fracture. (**d**–**g**) Postoperative range of motion following fixation of scapular and clavicle fracture. (**h**, **i**) Postoperative appearance of posterior (**h**) and anterior incisions (**i**) (*A* acromion, *S* sternal notch)

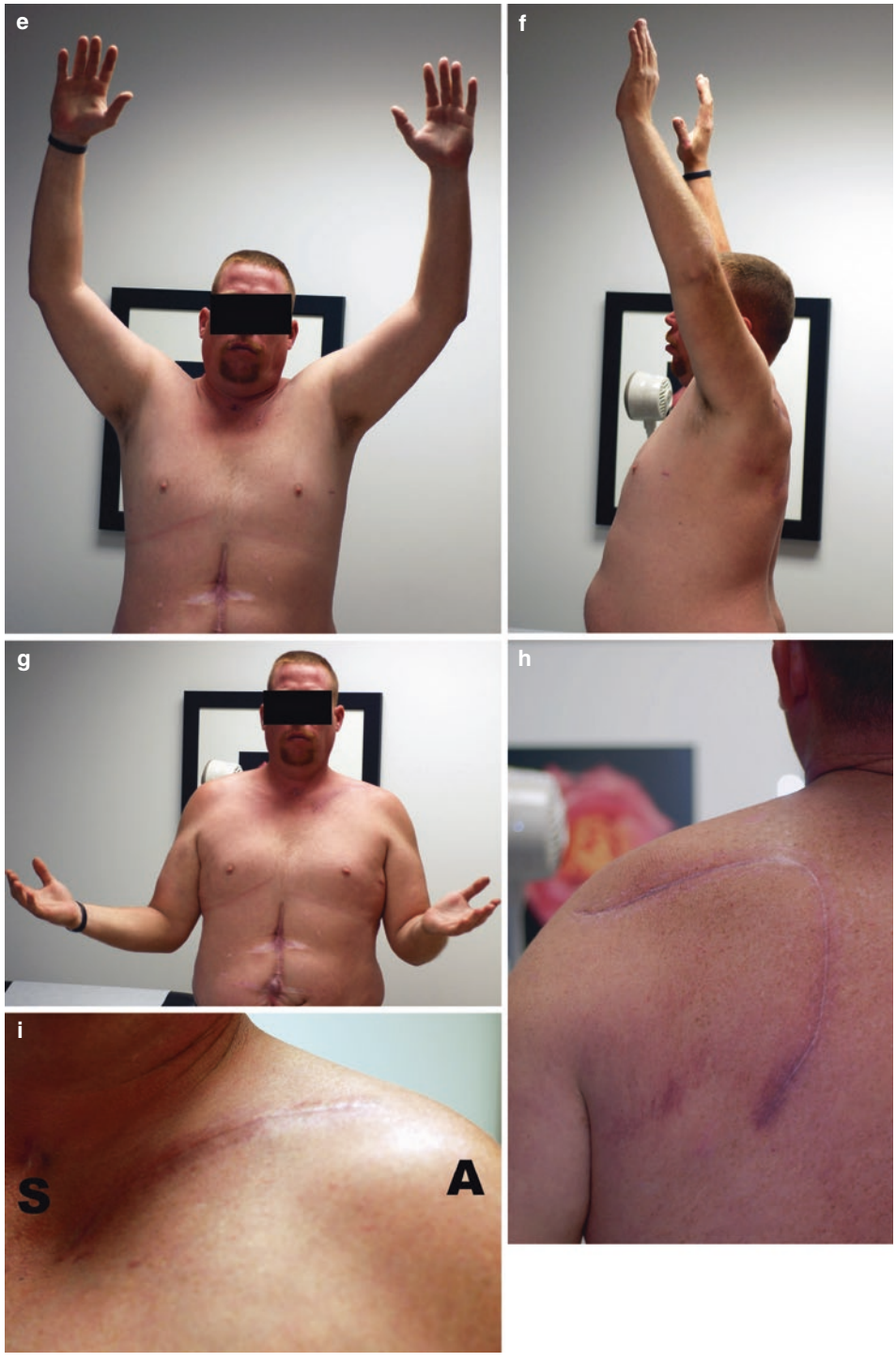

Fig. 15.13 (continued)

reduction, and fixation of the fracture. Suture anchors may be utilized if soft tissue attachments are of questionable integrity. Additionally, arthroscopy can be used to assist percutaneous screw fixation of larger intra-articular fracture fragments not amenable to suture fixation, such as minimally displaced Ideberg II and III fractures [98–102].

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Rehabilitation of Scapular Dyskinesis

16

Ann M. Cools, Todd S. Ellenbecker, and Lori A. Michener

Abbreviations

ASES	American Shoulder Elbow Surgeons Shoulder Score—patient self-report section
GRoC	Global rating of change
NMES	Neuromuscular electrical stimulation
NS	Not significant
SANE	Single Alpha Numeric Evaluation
SAT	Scapular assistance test
SDQ	Shoulder disability questionnaire
SRT	Scapular reposition test
SS	Statistically significant
SSMP	Shoulder Symptom Modification Procedure
WORC	Western Ontario Rotator Cuff Index

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Introduction

Scapular dyskinesis may have multiple causes including bony (e.g., clavicular fracture), neurologic (e.g., long thoracic or accessory nerve palsy), and muscular dysfunction (e.g., soft tissue inflexibility, muscle weakness, inhibition, or imbalance) [1]. The clinical reasoning process for scapular rehabilitation should be based on the clinical evaluation of the patient and should include a current understanding of the biomechanics of the upper quadrant, alterations in scapular kinematics and muscle function, biomechanical mechanisms inducing scapular dyskinesis, and chronic dysfunction in the painful shoulder and cervical spine. The development of a scapular rehabilitation program should be based upon the key impairments of scapular position and movement patterns, symptom alteration tests, and dynamic stability identified by both the objective clinical examination and additional clinical measurements. The clinical evaluation of the scapula should include all possible local and more distant contributors to dyskinesis. More proximal links in the kinetic chain, such as spinal mobility and stability as well as lower limb function are key points in shoulder rehabilitation—especially in those individuals who must rely on the lower extremities and trunk for the transfer of force to the upper extremity. In overhead athletes distal components like elbow strength and mobility and forearm pronation-supination should also be

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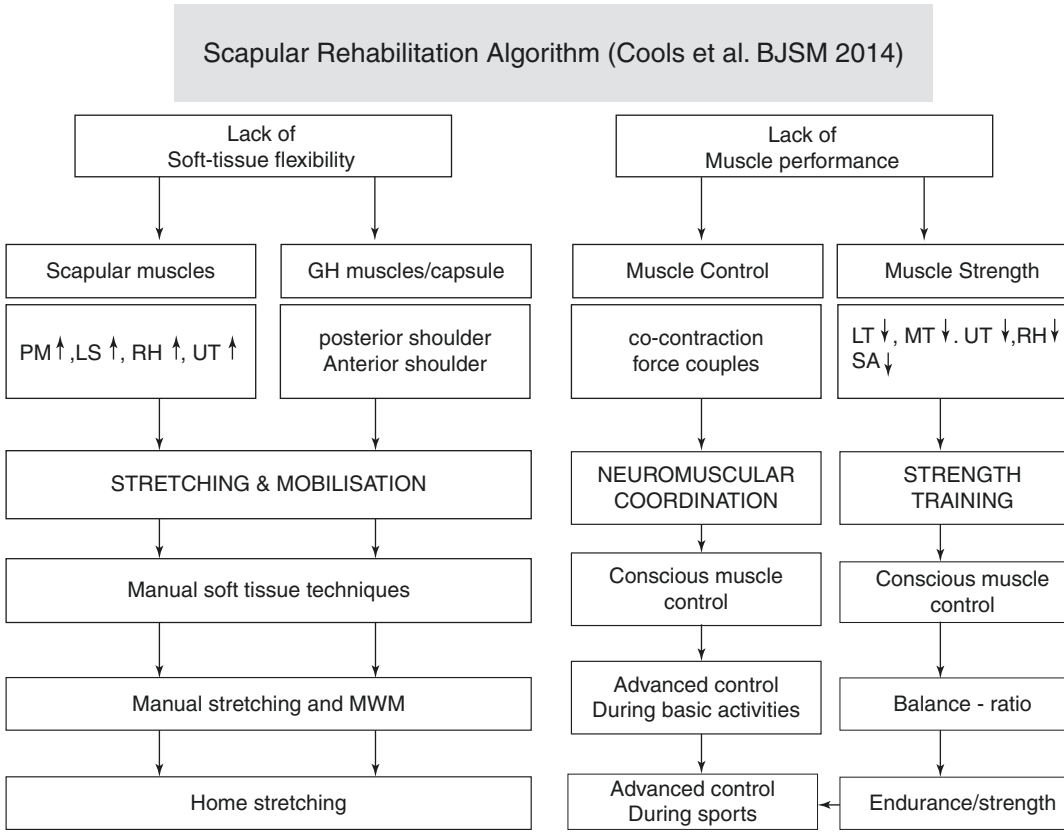

Fig. 16.1 Scapular rehabilitation algorithm [9]

addressed. Given the fact that the observed scapular dyskinesis might be the representation of normal movement variability [2, 3], it is imperative to explore the clinical relevance of this scapular dyskinesis with respect to the actual symptoms and complaints of the patient. Different types of corrective maneuvers, also known as symptom alteration tests, such as the scapular assistance test (SAT) or scapular reposition test (SRT), may be used to alter patient symptoms by correcting scapular position and motion [1, 4, 5]. The Shoulder Symptom Modification Procedure (SSMP) [6] also has corrective maneuvers that allow for the identification of the functional impairments in the scapulothoracic complex and may serve as a basis for treatment strategy. Additionally, the clinician should be aware of possible central sensitization mechanisms in patients with long-standing chronic shoulder pain and dysfunction [7].

Scapula-focused treatment aims to restore scapular position and movement patterns that are related to the patient's symptoms, which encompass a large part of the kinetic chain of the shoulder [8]. Flexibility deficits as well as muscle performance dysfunctions should be addressed (Fig. 16.1, adapted from Cools et al. [9]). The purpose of this chapter is to describe tactics and treatment strategies for flexibility deficits and muscular dysfunction around the scapula. Secondly, special attention is focused on the rehabilitation of scapular dyskinesis in the advanced stages of the rehabilitation of the overhead athlete. Finally, the effectiveness and outcomes after scapula-focused rehabilitation programs are discussed.

Treatment of Flexibility Deficits

The relationship between scapular position and glenohumeral joint range of motion has been studied with significant ramifications for

clinicians who treat patients with shoulder range of motion loss and scapular dysfunction. Laudner et al. [10] reported an association between posterior shoulder tightness (horizontal adduction range of motion loss) and scapular dysfunction characterized by anterior scapular positioning in 40 professional baseball pitchers. Additionally, a review of the literature by Ludewig and Reynolds [11] described findings of changes in scapular upward rotation and posterior tilt in patients diagnosed with adhesive capsulitis and shoulder stiffness. Vermeulen et al. [12] did show improvements in scapular biomechanics with physical therapy improving glenohumeral range of motion and resultant scapular mechanics. Therefore, evaluating patients with scapular dysfunction as mentioned earlier in this chapter should include objective quantification of glenohumeral joint range of motion to determine its potential role in scapular dysfunction. This includes the use of a goniometer or inclinometer to assess shoulder external and internal range of motion in 90° of abduction with scapular stabilization [13] (Fig. 16.2). Additionally, the measurement of horizontal crossarm adduction is also important given its potential relationship to scapular dysfunction in the overhead athlete [10] (Fig. 16.3). A plethora of studies are present in the orthopedic and sports medicine literature profiling the normative range of motion patterns in elite overhead athletes [14–18]. These studies can provide important framework for clinicians regarding normal shoulder range of motion patterns to identify individuals who have range of motion deficiencies that may affect scapular mechanics.

Additional measurements have been used to measure flexibility of the pectoralis musculature [19]. Kluemper et al. [20] and Lynch et al. [21] have used a double square method in standing to quantify bilateral differences in the anterior shoulder and scapular position in swimmers. This clinical technique allows for rapid identification of anterior shoulder posture and can be done in the supine position as well. Ellenbecker et al. [22] have identified increased anterior shoulder posture in the dominant arm of elite tennis players using this method. Individuals presenting with increases in anterior shoulder posture receive specific interventions to improve pectoralis flexibility such as supine scapular retraction stretches on a foam roll (with and without therapist assistance) (Fig. 16.4a, b) as well as corner stretches [21, 23]. To address posterior shoulder tightness, research has shown the sleeper stretch to produce acute and longer-term improvements in shoulder internal rotation range of motion [24, 25] and is recommended for clinical use (Fig. 16.5). Use of the crossarm stretch (horizontal adduction) is also supported in the literature by McClure et al. [26], Moore et al. [27], and Ellenbecker et al. [28], with the later study showing up to 8° of internal rotation range

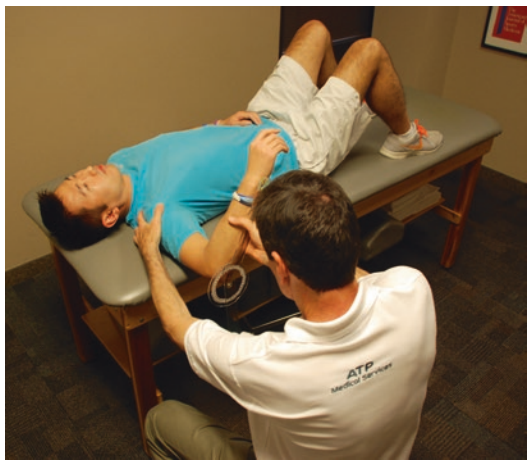

Fig. 16.2 Internal rotation measurement of the glenohumeral joint with scapular stabilization

Fig. 16.3 Measurement of horizontal crossarm adduction range of motion using a digital inclinometer and scapular stabilization

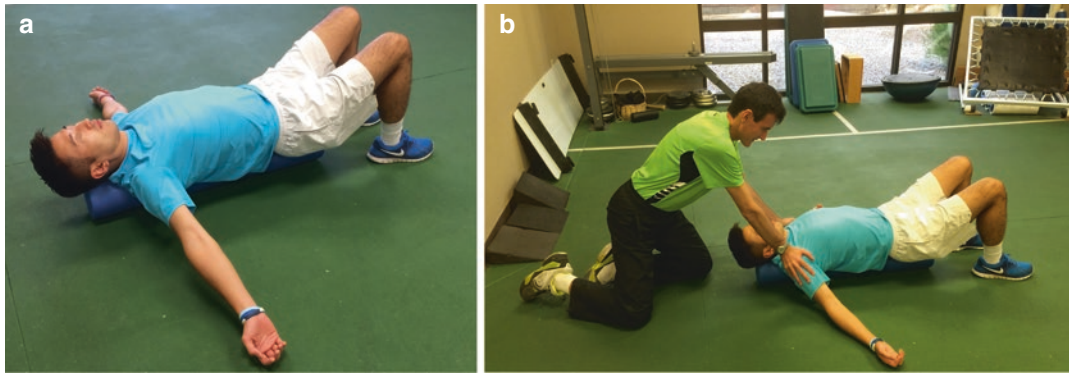

Fig. 16.4 Foam roll pectoralis stretch without (**a**) and with (**b**) physical therapist overpressure

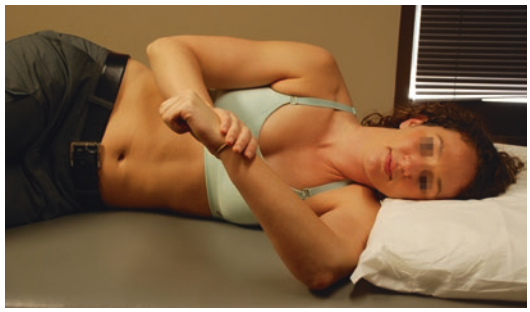

Fig. 16.5 Sleeper stretch

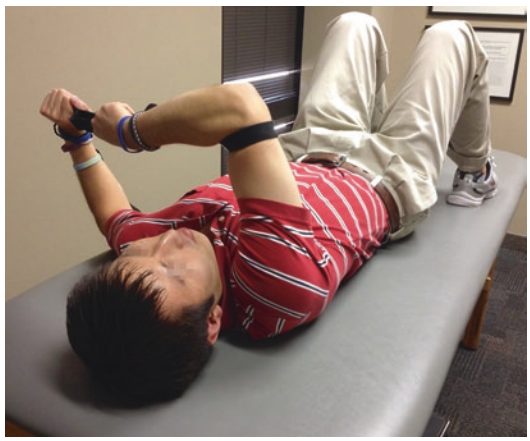

Fig. 16.6 Patient-independent crossarm adduction stretch with stretch strap

of motion improvement following three, 30-s stretches using a contract-relax format and stretch strap (Fig. 16.6). Clinically, research has recently been published [29] highlighting the importance of scapular stabilization during the crossarm stretch performed by a physical therapist showing superior results with the addition of a medially directed stabilization force on the lateral border of the scapula and shoulder during the application of the crossarm adduction movement (Fig. 16.7).

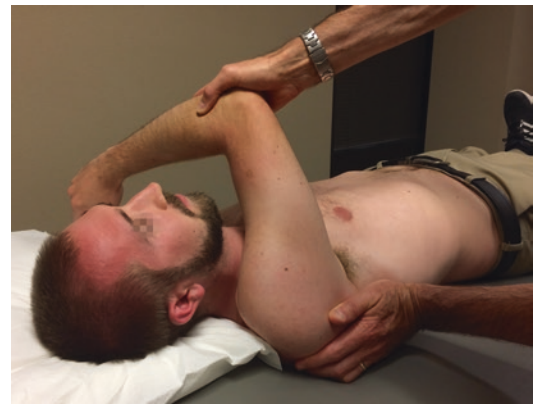

Fig. 16.7 Crossarm adduction stretch with scapular stabilization

Treatment of Muscle Performance Deficits

In the early stage of scapular training, conscious muscle control of the scapular muscles may be necessary to improve proprioception and to normalize scapular resting position. A priority is first given toward the patient attaining active control of scapular orientation, facilitated by the therapist and then practiced by the patient. Depending on the dominant type of scapular dyskinesis, attention is given to correcting the scapula toward more posterior

tilting, upward rotation, or external rotation. It was demonstrated in a three-dimensional scapular movement analysis study that it is possible to teach a normal subject to consistently reproduce movements of the scapula into posterior tilt and upward rotation [30]. In addition, De Mey et al. [31] showed higher electromyographical activity in the targeted muscles and in particular middle trapezius and lower trapezius during dynamic shoulder exercises when conscious correction of the scapular position was performed prior to the exercise. It is important to incorporate scapular orientation with spinal posture correction, especially in those patients who have a tendency to drift into a forward head posture, with an increasing thoracic kyphosis and protraction of the shoulder girdle. Patients are encouraged to perform this exercise repeatedly throughout the day, with the emphasis being on a change in postural habit. Memory joggers may be useful to ensure it becomes a habit [9].

Based on patient observation and the physical clinical exam, the clinician may decide to focus more on training of neuromuscular control (force-couple activation and co-contraction) during functional movements or on isolated muscle strength and balance training of the scapular muscles. The fundamental differences between both approaches might be the exercise selection. When focusing on motor control, low-load functional movement exercises should be chosen, mainly elevation exercises in variable planes, since the aim is to optimize neuromuscular control during daily functional activities. Moreover it has been suggested that high-load isolated training of individual muscles does not allow the nervous system to adapt to optimal movement strategies [2]. Motor control exercises should primarily focus on quality of movement and endurance, in more advanced stages of rehabilitation also on energy transfer and absorption [1, 2]. Scapular co-contraction may be trained in basic positions, movements, and exercises (e.g., simultaneous inferior glide, bilateral external rotation, and thoracic extension). As the shoulder girdle functions in both open- and closed-chain activities, the muscles should be trained to respond to both situations, by challenging the maintenance of the new scapular position under load, using weight-bearing and non-weight-bearing tasks of the upper limb. These should be consistent with the functional requirements of the patient. With respect to open-chain activities, Kibler et al. [32] described specific exercises for scapular control in the early phases of shoulder rehabilitation. The "low row," "inferior glide," "lawnmower," and "robbery" exercises, as described in their paper, activate the key scapular-stabilizing muscles without putting high demands on the shoulder joint, making these exercises appropriate to use in early stages of nonoperative as well as in postoperative rehabilitation. Closed-chain exercises are believed to improve dynamic glenohumeral and scapulothoracic stability through stimulation of the intra- and periarticular proprioceptors and enhance co-contraction of the rotator cuff and scapular stabilizers. It should be noted, however, that closed-chain positions such as the "push-up" position or the "wall slide" exercise mainly activate the anterior scapular muscles such as serratus anterior and also pectoralis minor. These exercises should be selected with caution, to avoid activation of anterior muscles that might be shortened or hyperactive [33]. In these early stages of rehabilitation, the progression may benefit from additional taping and bracing; however literature is inconclusive regarding the effects of these interventions [34]. The use of neuromuscular electrical stimulation (NMES) may also be considered. NMES on the serratus anterior and lower trapezius has been shown to increase the acromiohumeral distance in healthy shoulders, suggesting a beneficial effect on scapular position for patients with subacromial pain syndrome [35].

If substantial deficits in muscle strength or muscle imbalances are identified, selective muscle training restoring strength and inter- and intramuscular balance may be warranted, performing high-load exercises isolating specific muscle groups. These exercises are often less functional (e.g., in prone- or side-lying positions) as a consequence of the specific goal of the exercise, particularly aiming at activating one specific muscle group [36, 37]. Functional exercises such as elevation will always activate more or less all scapular muscles and will never be able to isolate one muscle group. If weakness in one muscle is accompanied by hyperactivity in another one, restoring muscle balance is necessary with minimal activity of the hyperactive muscles. After muscle balance is restored, more general strengthening exercises for the scapular muscles may be used.

The selection of exercises may be based on many criteria, such as general guidelines from the literature, outcome studies showing evidence of specific exercise protocols, personal preference of the clinician, and functional relevance in view of the patient's demands and expectations. Results from electromyographical studies on the activity of scapular muscles in healthy persons as well as in patients with upper quadrant pain and dysfunction have been a basis for recommendation for the choice of exercises during treatment of patients with shoulder or neck pain related to scapular dysfunction. Studies on patients illustrate specific scapular muscle dysfunctions in relation to shoulder or neck pain; studies on healthy persons justify the choice of exercises based on specific treatment goals. It is believed that performing exercises with the appropriate focus on specific muscle activation patterns may improve the quality of scapular motion and thus restore optimal movement patterns. Assuming the large variability in scapular dysfunction and abnormal scapular muscle recruitment patterns, exercises should always first focus on restoring the muscle balance. Based on current evidence and clinical experience, muscles that are often hyperactive are the upper trapezius, rhomboids, pectoralis minor, and levator scapulae, whereas a lack of activation has been found in the upper trapezius, rhomboids, middle trapezius, lower trapezius, and serratus anterior [38–40]. Indeed the role of upper trapezius and rhomboids in scapular dysfunction and shoulder pain is not clear yet, and it has been suggested that both hypercontracture and weakness might be present in a patient with upper quadrant pain and dysfunction [11, 41]. In view of these imbalances, exercises may be selected based on muscle balance ratios with lesser/more scapular muscle activity. Based on the available evidence [41, 42] and clinical experience, the clinically relevant balance ratios and most appropriate exercises are presented (Table 16.1).

Table 16.1 Exercise selection based on clinically relevant balance ratios

Hyperactive muscles	Hypoactive muscles	Clinically relevant balance ratios	Proposed exercises	Reference
Pectoralis minor	Serratus anterior	Pectoralis minor/serratus anterior	Serratus punch standing	[33]
	Middle trapezius	Pectoralis minor/middle trapezius	Elevation with external rotation	[43]
	Lower trapezius	Pectoralis minor/lower trapezius	Elevation with external rotation	[43]
	Rhomboids	Pectoralis minor/RH	Elevation with external rotation	[43]
Upper trapezius	Serratus anterior	Upper trapezius/serratus anterior	Elbow push-up	[37]
			Serratus punch supine	[44]
			Elevation with external rotation	[43]
			wall slide	[43]
	Middle trapezius or lower trapezius	Upper trapezius/middle trapezius or upper trapezius/lower trapezius	Elevation with external rotation	[43]
			Side-lying forward flexion	[36]
			Side-lying external rotation	[36]
			Prone Hor Abd with external rotation	[36]
			Prone extension	[36]
			Prone external rotation in 90° abduction	[45]
	Rhomboids	Upper trapezius/rhomboids	Elevation with external rotation	[43]
Levator scapula	Serratus anterior	Levator scapula/serratus anterior	Wall slide	[43]
	Upper trapezius	Levator scapula/upper trapezius	Overhead shrug	[46]
	Middle trapezius or lower trapezius or rhomboids	Levator scapula/middle trapezius or levator scapula/lower trapezius or levator scapula/rhomboids	Overhead retraction	[46]
Rhomboids	Serratus anterior	Rhomboids/serratus anterior	Wall slide	[43]

From a clinical perspective, motor control deficits, muscle imbalances, and hyperactivity are not easy to examine or measure in an objective way, and often the clinician relies upon his visual observation skills to define normal versus abnormal scapular position and motion. Using the medial border of the scapula as the landmark for scapular orientation, three types of scapular malpositioning may be defined. Predominant inferior medial border prominence (type I) reflects a scapula that is too much anteriorly tilted. Underlying mechanisms may be flexibility deficits in the pectoralis minor and/or a dysfunction in the lower trapezius and/or serratus anterior, the force couple responsible for posterior tilt of the scapula. Exercises for type I should therefore focus on activating serratus anterior and lower trapezius. Exercises with a glenohumeral external rotation component have shown to increase activity in lower trapezius [36, 43]. In case the entire medial border is visible (type II), the scapula is positioned in too much internal rotation. Exercises should focus on external rotation of the scapula, activating the force couple trapezius/serratus anterior. Exercises performing retraction in the horizontal plane (with the arms elevated 90°) are most likely to enhance scapular external rotation and activating the three trapezius parts together with serratus anterior. These exercises may be performed in open (e.g., "horizontal abduction with ER") as well as in closed (e.g., "from prone to side bridging") kinetic chain. When scapular malpositioning is characterized by superior medial border prominence (type III), the scapula is too much downwardly rotated. Underlying mechanisms are contracture of levator or rhomboids and dysfunction in the upward rotation force couple including upper trapezius and serratus anterior with stabilizing components coming from middle and lower trapezius. There should be a focus on promoting upward rotation by performing exercises with the arms in higher elevation angles to put the scapula in a maximal upward rotation, like overhead shrugging and retraction [46].

Throughout the exercise program, proximal and distal links of the kinetic chain should be implemented. Creating diagonal patterns in open and closed chain by standing on the contralateral leg or extending one leg in the four-point kneeling position has shown to positively influence serratus anterior and middle and lower trapezius activity [47, 48]. Adding trunk rotation to exercises also promotes proper scapular alignment into posterior tilt and external rotation and increases lower trapezius activity and decreases upper/lower trapezius ratio [49]. Implementing upper limb functional patterns into the exercises allows focus on elbow, forearm, wrist, and hand movements during scapula-focused exercises.

Although the relevance of the scapula in normal shoulder function is well documented and therefore any rehabilitation program for the upper quadrant should include scapula-focused interventions, there are still many uncertainties and questions, and therefore clinicians as well as researchers should remain critical in their interpretation of research and clinical guidelines. Firstly, the cause-consequence between shoulder pain and scapular dyskinesis remains unclear. It is unclear whether the alterations found in scapular kinematics are compensatory or contributory to neck/shoulder pathology. Results from prospective studies investigating scapular dyskinesis as a possible risk factor show conflicting results [50–52]. According to a recent theory of pain adaptation, it is suggested that during episodes of pain, there is a redistribution of activity within and between muscles, which may have some benefit in the short term (protection from further pain or injury), but there is the potential for adverse long-term mechanical consequences to pain-sensitive tissues [53]. In view of this theory, scapular muscle dysfunction might be secondary to painful shoulder or neck conditions. Secondly, since scapular dyskinesis seems to be present also in a large proportion of a healthy population [54] and scapular asymmetry is considered to be

"normal" in overhead athletes [1], the observed "abnormal" position and motion of the scapula may represent normal kinematic variability, reflecting the individual variety of coordination patterns to complete a task [2]. Therefore, it is imperative to put possible scapular dysfunction into the right perspective, linking the dyskinesis to the presence of symptoms [6]. Thirdly, when prescribing exercises "focusing on the scapula," we should take into account that scapular exercises always include glenohumeral components. In most exercises with target on the scapula, the rotator cuff muscles are highly activated as a result of an external or internal rotation component during the exercise. It is therefore nearly impossible but also not desirable to markedly differentiate between "scapular" and "glenohumeral" exercises, but rather to focus on the integration of both links in the upper limb kinetic chain in a coordinated manner in the exercise program.

Scapular Rehabilitation in the Overhead Athlete

Evidence of scapular dysfunction in the overhead athlete is present in the literature. Oyama et al. [55] studied overhead athletes and found the dominant extremity to have greater amounts of scapular upward rotation, anterior tilt, and internal rotation. Additionally, the dominant side shoulder girdle was consistently more protracted. Cools et al. [56] similarly found significant scapular position and scapular muscle strength differences or asymmetries between the dominant and non-dominant extremity in elite junior tennis players. Significant muscular demands have been profiled in electromyographical studies for the tennis serve and ground strokes [57, 58], as well as for the overhead throwing motion [59]. These studies show the inherent demands on the scapular musculature needed for optimal stabilization and positioning particularly during the phases of late cocking and deceleration. Exercises that specifically focus on the positions of 90° shoulder external rotation and abduction (late cocking) as well as during late acceleration and follow-through are recommended. Ellenbecker et al. [60] provided a descriptive study of two commonly used plyometric exercises that recruit the serratus anterior, posterior rotator cuff, and lower trapezius at moderate to high levels and utilize biomechanical movement patterns that simulate those utilized by overhead athletes. Figures 16.8 and 16.9a–d profile those exercises for rotator cuff and scapular stabilization. A low-resistance/high-repetition base for these exercises is again emphasized. Carter et al. [61] have demonstrated increases in shoulder strength and throwing velocity after an 8-week program of rotator cuff and scapular exercises using both elastic- and plyometric-based exercises. Figure 16.10 shows a follow-through specific exercise using both elastic resistance and rhythmic stabilization applied to the dominant extremity in a throwing athlete that can be utilized. Further research is needed in this area to better understand the effects of scapular stabilization exercise on both performance enhancement and injury prevention.

Fig. 16.8 90/90 plyometric drops to increase posterior rotator cuff and scapular muscle activation

Fig. 16.9 (**a**–**d**) 90/90 reverse catch plyometric exercise

Fig. 16.10 Scapular protraction exercise mimicking the follow phase of the overhead throwing motion with elastic resistance and rhythmic stabilization

Outcomes After Scapula-Focused Exercise Programs

The evidence for the effectiveness of scapular-focused rehabilitation is limited [34]. There is limited support for the superiority of scapular-focused programs over usual care or non-scapular-focused exercise therapy. There are five randomized clinical trials [62–66] that have used various types of scapular-focused exercise interventions in patients with shoulder pain. The interventions included some type of scapular-focused exercise program of strengthening and motor control, stretching of associated scapular soft tissue stretching, and scapular mobilizations. Overall, the evidence indicates that scapular-focused programs can improve the impairments of scapular muscle performance and strength, but these changes in impairments have a limited to no effect on patient-rated pain and functional shoulder use.

Scapular-focused treatments generally are recommended to improve associated impairments, but one specific program cannot be recommended over another program. All five studies [62–66] examined the effects of scapular-focused exercise treatment, with only two of five reporting superior effectiveness. All five studies are detailed in Table 16.2. Specifically, Celik et al.

Table 16.2 Randomized clinical trials investigating the patient-rated outcomes of scapular-focused exercise treatment for shoulder pain

Study	Participants	Groups	Scapular-focused intervention	Assessment	Outcomes	Conclusions
Baskurt [62]	Subacromial impingement syndrome (n = 40)	1 = stretch, strengthen	Group 1:	– Baseline	Between groups:	No greater improvements in patient-rated measures of pain and disability (WORC) between groups. Both groups improved
		2 = stretch, strengthen, scapular stabilization ex	Stretching: anterior, posterior, inferior capsule; flexion range of motion, abduction range of motion, internal rotation stretch	– End of treatment—6 weeks	1. Pain: rest, during activity; NS	
					2. WORC (0–2100; 0 = no disability); NS	
			Strengthening: resisted internal and external elevation			
			Codman			
			Group 2:			
			Stretching: as Group 1			
			Strengthening: as Group 1			
			Codman: as Group 1			
			Scapular stabilization: scapular clock, standing weight shift, scapular depression, push-up, wall slide			
			All exercises: 3 × 10 reps			
			3/week × 6 weeks (18 visits)			
Struyf [66]	Subacromial impingement syndrome (n = 22)	1 = scapular-focused treatment	1 = stretching, scapular motor control training	– Baseline	Between groups:	Significant and clinically important improvement in those in Group 1 of the scapular-focused treatment program in SDQ-reported shoulder disability
		2 = control	2 = stretching, cross-fiction massage, eccentric rotator cuff strengthening	– 4–8 weeks (9 visits)	1. Pain, NS	
			Both groups: 9 visits over 4–8 weeks	– 12 weeks (last visit)	2. SDQ (0–100, 0 = no disability), SS > improvement in Group 1	
Moezy [64]	Subacromial impingement syndrome (n = 68)	1 = scapular-focused treatment of scapular stabilization	1 = flexibility, strengthening, scapular stabilization, and postural exercises	– Baseline	Between groups:	No differences in pain between two groups
		2 = general exercise	2 = pendulum and range of motion exercises; infrared therapy; ultrasound; electrical stimulation	– 6 weeks	1. Pain, NS	
			Both groups: 3 visits × 6 weeks; total of 18 visits		No measure of shoulder disability or functional loss	

Mulligan [65]	Subacromial impingement syndrome ($n = 40$)	1 = scapular stabilization	1 = 4 exercises; supine scapular punch, rows, scapular depression/retraction, W exercise	– Baseline	Between groups:	Trends toward greater improvement with scapular stabilization program performed first in sequence, but this improvement was not significant
		2 = rotator cuff strengthening		– 4 weeks	1. Pain, NS	
			2 = 4 resisted exercises: ER at the side, short arc military press → caption, IR at the side, horizontal abduction	– 8 weeks	2. ASES, NS	
			6 visits/4 weeks per group assignment	– 16 weeks	3. Global rating of function (SANE), NS	
			Next 6 visits/4 weeks crossover to add exercises from other group		3. Satisfaction, NS	
					4. GRoC, SS but no difference in post-hoc tests * only Group 1 improved over time	
					Both groups demonstrated improvement over time	
Celik [63]	Frozen shoulder ($n = 29$)	1 = scapular-focused exercise and glenohumeral range of motion exercises	1 = exercise aimed at strengthening/motor control of the serratus, middle and lower trapezius; upper trapezius stretching; postural exercises	– Baseline	Between groups:	Improved pain at 6 weeks in those receiving scapula-focused treatment; however, this did not translate to improved pain at 12 weeks. No effect on patient-rated function
		2 = glenohumeral range of motion exercises	2 = glenohumeral range of motion—active and passive, manual stretching	– 6 weeks	1. Pain—6 weeks, SS improvement in Group 1	
			30 visits over 6 weeks	– 12 weeks	Pain—12 weeks, NS	
					2 Constant, NS	

NS not significant, *SS* statistically significant, *ASES* American Shoulder and Elbow Surgeons Shoulder Score—patient self-report section, *SDQ* shoulder disability questionnaire, *SANE* Single Alpha Numeric Evaluation, *WORC* Western Ontario Rotator Cuff Index, *GRoC* Global rating of change

[63] reported improved pain in the short term at 6 weeks, but this superiority was not maintained at 12 weeks in patients with frozen shoulder. Struyf and colleagues [66] reported improved patient-rated outcomes in a group of patients with subacromial pain syndrome undergoing rehabilitation. The scapular-focused treatment by Struyf [66] that incorporated muscle performance training, stretching, and scapular mobilizations was superior to a program focused at the glenohumeral joint. The most recent study by Mulligan and colleagues [65] does report that sequencing the use of scapular stabilization may be important. When scapular stabilization was used in the first weeks of treatment, there was a trend of greater improvement in patient-rated outcomes. Overall, there is limited evidence for the superiority of use of scapular-focused treatment over usual care or non-scapular-focused exercise therapy in patients with shoulder pain.

Future research is needed to identify the scapular impairments that are associated with muscle and motion deficits at the scapula and glenohumeral joint and functional loss. Improvements in scapular strength have been found with scapular-focused programs, but these improvements have not translated to improved patient-rated outcomes. It is important for future research to determine the critical threshold of impairments that require treatment and the threshold of resolution for the impairments that are associated with meaningful and substantial improvements in patient-rated outcomes.

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17 Rehabilitation for Complex Scapular Dysfunction: Considerations of Pain and Altered Motor Patterns

Aaron D. Sciascia, Robin Cromwell, and Tim L. Uhl

Introduction

Scapular dysfunction has been described in detail in the previous chapters. Most discussions regarding alterations in scapular motion center on the terms "winged scapula" [1] and "snapping scapula" [2]. The "winged" scapula is a descriptive term often used to identify the patient with an asymmetrically prominent medial scapular border, either at rest or upon arm motion [1]. There is commonly a deficit in shoulder function as a result of dysfunction of the scapular muscles and/or positional imbalance of the scapula. Past literature has suggested that scapular dysfunction is primarily due to peripheral nerve lesions affecting the long thoracic, dorsal scapular, or spinal accessory nerve, and their associated musculature, or an underlying neuromuscular problem such as muscular dystrophy [3–6]. Recent literature has demonstrated scapular malpositions, or altered scapular motion can be frequently associated with shoulder pain due to alterations in the supporting bony structure, in the joints of the spine and shoulder complex, motor performance, or tissue flexibility surrounding the scapula [7–10].

Complex scapular dysfunction, characterized by moderate to severe pain with accompanying overt scapular dyskinesis and limited use of the arm, can be due to multiple factors. The most typical factors are neurological damage (long thoracic or spinal accessory nerve palsies), traumatic injury (detachment of one or more scapular muscles), or chronic adaptations from unresolved injury, impairment, or soft tissue dysfunction. In cases of scapular dysfunction with neurologically rooted causes, rehabilitation can be performed to restore some level of arm function; however if the conservative measures fail, surgical options, such as muscle transfers, may need to be considered [11]. Due to the disrupted anatomy, patients with scapular muscle detachments will most often find a reduction in pain and an improvement in function with surgical reattachment rather than nonoperative rehabilitation [9]. The patients with long-standing scapular dysfunction without neurological or traumatic causes pose the most challenges for clinicians. There is not a standardized method of treatment, and not every patient will find a resolution. Pain and decompensation from

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long-standing dysfunction create a challenge for clinicians beyond that of typical musculoskeletal impairment. It is possible that patients may experience not only biomechanical and anatomical alterations but also alterations in pain processing. These patients tend to require individualized care where the clinician must apply both the science and art of rehabilitation.

Applying individualized care begins with a thorough assessment of the causes of pain and dysfunction where specific limitations from the patient's perspective need to be identified and prioritized. It is possible that poor outcomes may persist if the causes of pain or each patient's needs are not completely identified. This chapter will introduce the concept of altered pain processing and the impact it can have on scapular function, provide details of the kinetic chain approach for rehabilitation, and offer modifications of the kinetic chain approach for complex cases of scapular dysfunction.

Non-exercise-Based Considerations

Recent work has begun to identify factors related to pain perception and psychological constructs that could contribute to a person's physical dysfunction. Chronic injury could result in the prolonged release of neuropeptides which could lead to changes in supraspinal pain processing resulting in hypersensitivity [12, 13]. This hypersensitivity, termed central sensitization, is characterized by an amplified pain response where a patient is more susceptible to perceiving high levels of pain with low-level nociceptive stimuli as well as pain over a larger area of the body than what is truly affected [13]. The pain can also persist long after the painful stimulus has been removed or tissue injury has healed. This phenomenon as well as other variations in pain processing (peripheral sensitization or absent pain sensitization) has been shown in both experimentally and clinically present pain [14, 15]. Additionally, injuries can result in a mixed pain state where the nociceptive response is not the only contributor to a person's pain.

Recently, psychological factors have been identified that could influence the patient's perception of pain felt in the presence of shoulder injury [14–18]. One such consideration would be a patient's tendency to be a pain catastrophizer. Pain catastrophization is characterized by an exaggerated negative mental state about actual or anticipated painful stimuli [19]. Patients are typically in a constant state of awareness of painful sensations, a feeling of helplessness that the pain will not go away, and fear of movement that will worsen pain. In order to not confuse the different constructs, a simplified description of central sensitization and catastrophization would be that central sensitization is how the body responds due to brain neuroplasticity [13] regarding pain perception while catastrophization is how the person copes with pain based on previous experiences.

Patients with chronic complex scapular dysfunction could experience these alterations in pain processing. However, the challenge that exists for clinicians to appreciate is that traditional rehabilitation efforts such as the application of therapeutic modalities or exercise may not be successful. Patients with altered pain processing can be treated one of two ways [20, 21]: (1) apply treatments that could address pain and dysfunction due to movement-elicited pain or (2) apply treatments based on pain at rest. With movement-elicited pain, patients will, as the term suggests, perceive pain only with active or in some cases passive movement. Therefore, it is possible that there is compromised anatomy and physiology driving the sensations. In this situation, traditional rehabilitation efforts such as pain control with anti-inflammatory medications and mechanical modalities may be initially attempted as well as a prescription of therapeutic exercise to help alleviate the painful stimuli and dysfunction. However, if these efforts fail to reduce pain, other

measures may need to be considered such as prescription nonnarcotic medications designed to reduce neurologically mediated pain through the blockage of ion channels known to carry the nerve impulses to the brain. The concept is that the pain "volume" is turned up, and the system needs to be quieted down for the therapeutic exercise to take effect [22].

Conversely, patients who suffer from pain at rest may have characteristics of central sensitization or pain catastrophization. These patients may benefit from neuroscience pain education [23]. Pain education teaches patients about pain perception and the physical responses that can occur. This approach appears to have more success when the education is based on the neurophysiology of pain (how each person's brain perceives pain) rather than traditional anatomical and biomechanical focuses (i.e., torn tissue as the cause) [24, 25]. These programs may have benefit for patients with chronic complex scapular dysfunction as these patients can present with characteristics similar to patients with chronic shoulder pain [14, 16, 17]. However, it should be noted that some authors have found better success when neuroscience pain education is combined with other interventions such as manual therapy or aerobic exercise rather than utilizing education as the sole intervention [26, 27].

Treatment of complex scapular dysfunction may need to follow assessment and treatment of pain perception to have a positive effect on the patient. Questionnaires and quantitative sensory testing can be used to evaluate the level of pain perception in the initial patient evaluation. Questionnaires such as the painDETECT questionnaire [28], the Brief Pain Inventory [29], and Pain Catastrophizing Scale [19] can be used to obtain a patient perception of painful experiences. Quantitative sensory testing could be performed bilaterally with mechanical devices designed to evoke a painful response via different temperature and force ranges [14, 16, 17] or via tactile sensory devices such as varying gauged monofilaments, brushes, vibration, and sharp/dull pin devices [30].

If pain at rest is present, pain education and pharmacological interventions should be attempted initially. Conversely, if a patient has pain with movement, initial efforts of traditional therapeutic exercise programs should be developed. However, the complexity of the scapular dysfunction often disallows typical rehabilitation maneuvers (characterized by long lever arm movements) to be effective. Clinicians are suggested to follow the kinetic chain approach as patients will likely benefit from a comprehensive "retraining" of the body as a unit. However, the presence of pain and complex dysfunction may limit the effect of the kinetic chain approach. In these instances, supplementing the rehabilitation program with neuroscience pain education may be attempted. It is often appropriate to modify the kinetic chain-based exercises where fewer degrees of freedom are allowed or utilize more closed chain exercises early to decrease the stress on the highly irritated tissues.

Understanding the Kinetic Chain Approach

The kinetic chain rehabilitation approach is not unlike other treatment philosophies where the acute stage primary goal is to protect healing tissue and reduce pain [31]. The kinetic chain approach is characterized by its focus on treating the body as a unit rather than specifically targeting localized symptoms at the injured joint [31, 32]. This model is routinely used as a framework to describe the manner in which the individual body segments interact with each other to perform a dynamic task. By definition then, the kinetic chain is a coordinated sequencing of activation, mobilization, and stabilization of body segments to produce a dynamic activity [33].

A kinetic chain rehabilitation framework for shoulder injury describes an alternative shoulder

rehabilitation approach that focuses on three critical components [32]. First, patients are upright during exercise performance rather than be positioned supine or prone when possible to simulate functional demands. Second, the lever arm on the shoulder and trunk is shortened to reduce the load on the injured arm. Finally, arm motions should be initiated using the legs and trunk to facilitate activation of the scapula and shoulder muscles, which is a typical neurological pattern of motion [34, 35]. This framework was later expanded to include a set of progressive goals [31]: (1) establish proper postural alignment, (2) establish proper motion at all involved segments, (3) employ facilitation of scapular motion via exaggeration of lower extremity/trunk movement, (4) apply exaggeration of scapular retraction in controlling excessive protraction, (5) utilize the closed chain exercise early, and (6) work in multiple planes.

First, clinicians are encouraged to establish proper postural alignment. Proper posture can be achieved with a logical and progressive treatment plan to restore skeletal segmental stability and mobility through muscle reeducation, soft tissue mobility, and spinal/rib mobilization. Since the core drives kinetic chain function, it is imperative that optimal stabilization and force generation can occur. Muscle reeducation and strengthening of the core muscles should begin early in rehabilitation, targeting both local and global muscles [36]. In this first stage of the kinetic chain approach, soft tissue deficits, i.e., inflexibilities of both upper and lower extremities should also be addressed. These deficiencies can impede progressions if left unattended and delay treatment process. Segmental mobility of the thoracic spine and rib cage mobility is necessary for the scapula to track appropriately during arm motion. The lack of skeletal alignment, adequate tissue mobility, and core stability can impede scapular muscular ability to activate properly.

The next logical progression is to direct treatment toward the scapula. Primary stabilization and motion of the scapula on the thorax involve the coupling of the upper and lower fibers of the trapezius muscle with the serratus anterior and rhomboid muscles. The lower trapezius has the role as a scapular stabilizer to counter the actions of the serratus anterior. Anatomical dissection has revealed that the lower trapezius fibers minimally change length during arm elevation and therefore function to prevent the serratus anterior from pulling the scapula laterally and anteriorly around the thorax as it upwardly rotates the scapula during arm elevation [37, 38]. The serratus anterior contributes to all components of three-dimensional motion of the scapula during arm elevation contributing to produce scapular upward rotation, posterior tilt, and external rotation while stabilizing the medial border and inferior angle preventing scapular winging [39].

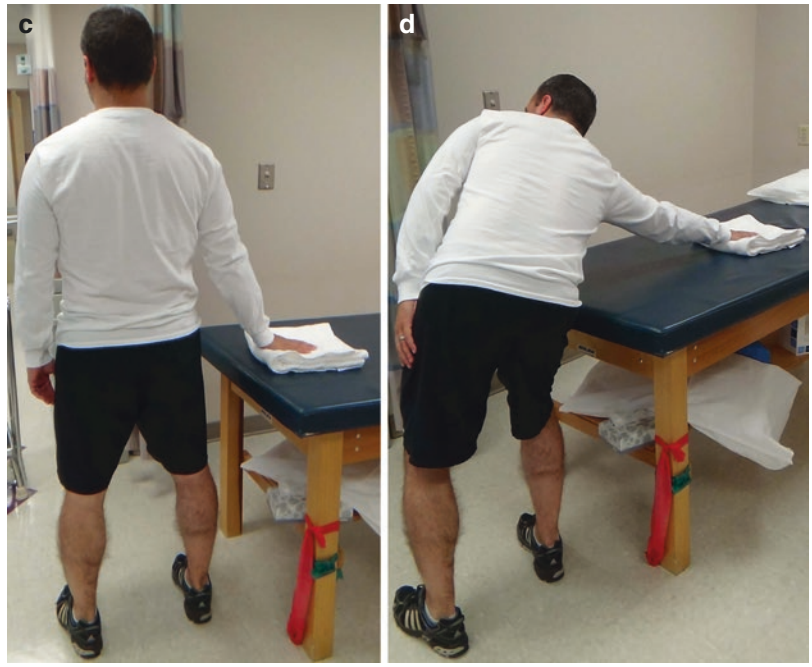
Arm function overhead requires that the scapula obtains a position of posterior tilt and external rotation which allows optimal muscle shoulder activation that is synergistic with trunk and hip musculature. This kinetic chain pattern of activation then facilitates maximal muscle activation of the muscles attached to the scapula [32]. This integrated sequencing allows the retracted scapula to serve as a stable base for the origin of all the rotator cuff muscles, allowing optimal concavity compression to occur [40, 41]. Therefore, implementing scapular stabilization exercises which incorporate lower extremity stability and muscle activation would be appropriate.

During the functional phase in the latter stages of the rehabilitation process, general glenohumeral strengthening would be introduced. Open chain exercises attempt to isolate the rotator cuff muscles through long lever arms performed in single plane ranges of motion which could potentially create shear across the joint creating muscular irritation. These exercises are often performed in less functional positions of lying down or in side lying which discourage proper kinetic chain activation [42–44]. Only after the kinetic chain links have been optimized should traditional strengthening measures such as these be introduced. However, the measures should also be tailored to involve the kinetic chain links as an integrated unit rather than in isolation to simulate normal function. A sample program of kinetic chain-based exercises is contained in the Appendix (Figs. 17.1, 17.2, 17.3, 17.4, 17.5, 17.6, and 17.7).

Fig. 17.1 The sternal lift begins with the trunk and knees slightly flexed (**a**). While keeping the arms at the side of the body, the patient is instructed to stand up tall retracting the scapulae (**b**)

Fig. 17.2 Table slides are performed standing to assist with flexion (**a**, **b**) and abduction (**c**, **d**). For either direction, the patient is instructed to rest the hand on a towel and allow the trunk to drive the arm

Fig. 17.2 (continued)



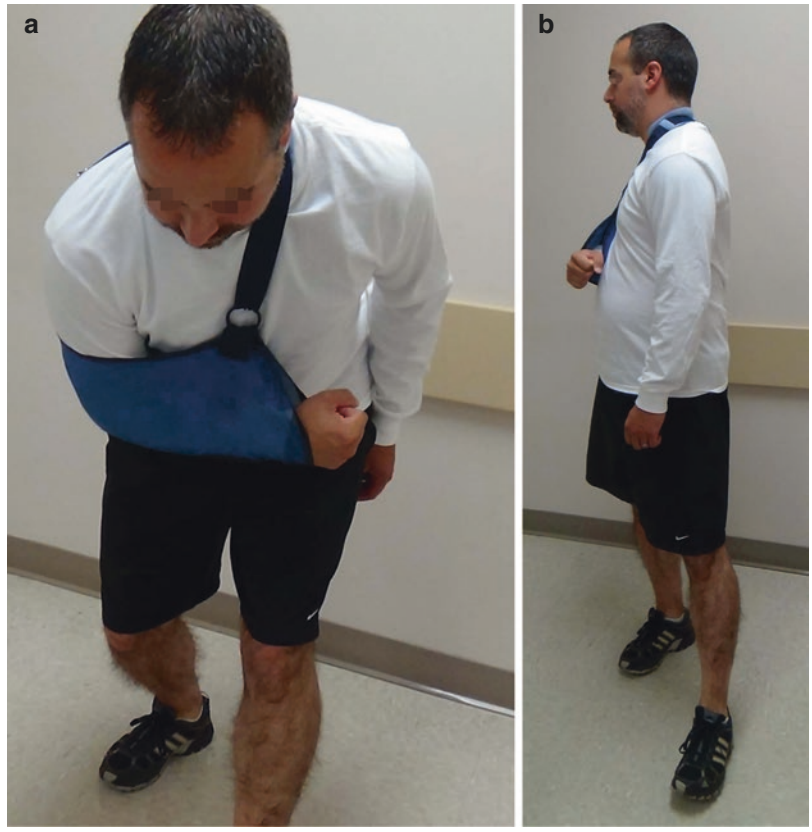


Fig. 17.3 The lawn mower exercise can be performed early in the recovery process when immobilized as part of early protection. The patient is instructed to have a staggered stance with the opposite leg forward and the trunk flexed (**a**). The patient then shifts his or her weight back with slight trunk rotation, facilitating scapular retraction (**b**)

Fig. 17.4 The lawn mower may be performed without immobilization. The starting position remains the same; however, the arm is allowed to be slightly flexed and pointing toward the opposite knee (**a**). The patient then shifts his or her weight back with slight trunk rotation, facilitating scapular retraction (**b**)

Fig. 17.5 The low row is performed standing with the hand of the involved arm placed against a firm surface (such as the side of a table or other object) and the hips/knees slightly flexed (**a**). The patient is instructed to extend the hips and arm allowing scapular retraction to occur (**b**)

Fig. 17.6 The low row may be progressed by replacing the table with 2–3 pound-free weights (**a**). The patient is instructed to extend the hips and arms but also to externally rotate the arms to gain scapular retraction (**b**)

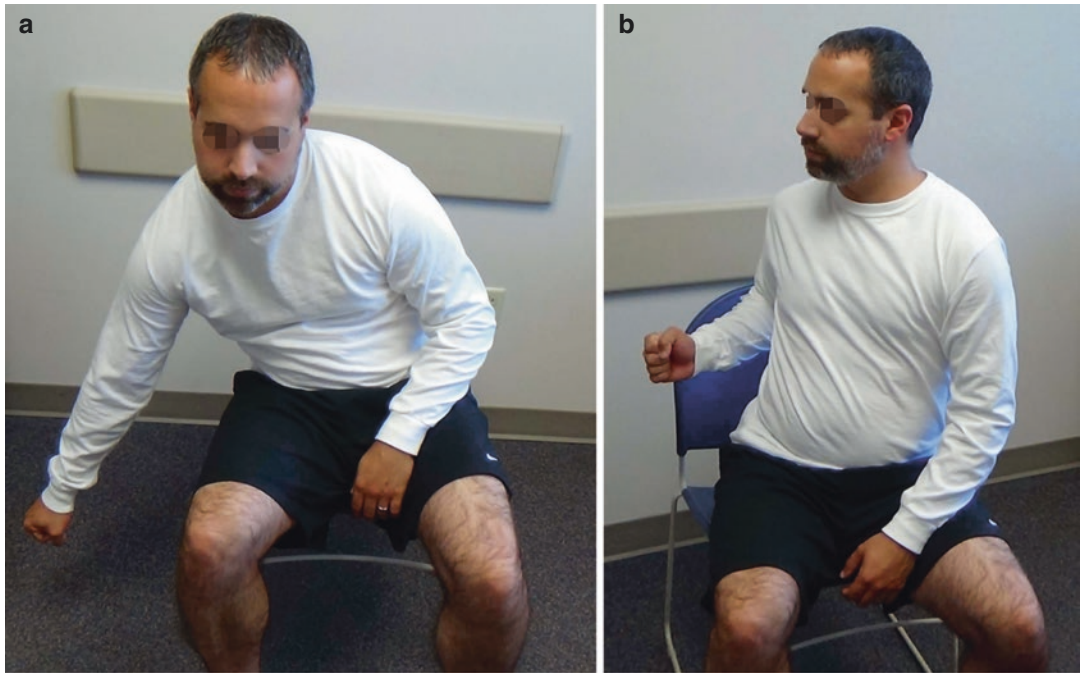


Fig. 17.7 The fencing maneuver is performed initially while sitting with the arm slightly abducted (**a**). The patient is instructed to rotate the trunk and adduct the arm to gain scapular retraction and depression (**b**). This exercise may be progressed to where elastic tubing is used while standing (**c**). The patient is instructed to step laterally while rotating the trunk and adducting the arm to gain scapular retraction and depression (**d**)

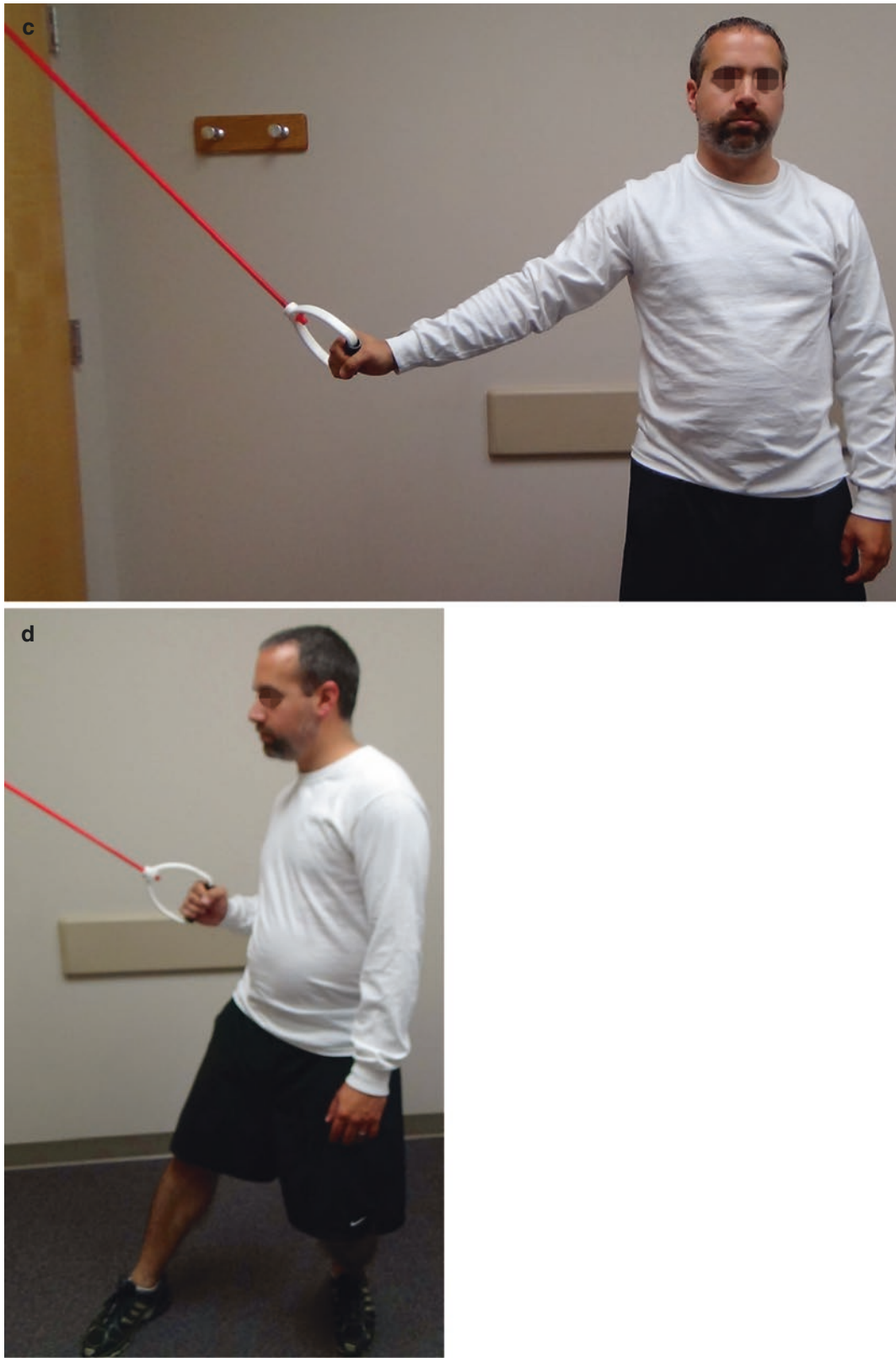


Fig. 17.7 (continued)

Modifying the Kinetic Chain Approach

While the framework and goals described above for treating scapular dysfunction assist clinicians with targeting the body as a unit, there are some cases when this framework needs to be adjusted to help patients overcome their functional deficits. In particular, patients with complex scapular dysfunction with no specific cause may not find immediate success with the kinetic chain rehabilitation program. Patients with chronic complex scapular dysfunction often present with the following symptoms and clinical findings:

- Hypertrophic or overactive upper trapezius, often seen as one of the few muscles to activate early and in some cases continuously, i.e., shrugging with arm elevation thus limiting fluid and full range of motion
- Limited activation from the lower trapezius or serratus anterior or both during arm elevation creating medial border and inferior angle winging (excessive anterior tilting and internal rotation of the scapula)
- Pectoralis minor and latissimus dorsi tightness
- Atrophy of the rotator cuff musculature
- Pain with arm movement which may be accompanied by audible popping or grinding beneath the scapula not caused by osteophyte formation (snapping scapula) (repositioning for pain relief)
- Hypersensitivity to pain such as complex regional pain syndrome or other pain centralization characteristics
- Scapular motion during arm movement that may present as an observable uncontrollable spastic motion.

In an attempt at explaining these clinical manifestations of scapular dysfunction, we offer the following explanation.

When a patient with chronic complex scapular dysfunction attempts to move his or her arm, the humeral head cannot center within the glenoid fossa. The inability to center the humeral head in the glenoid suggests that concavity compression is likely compromised. If concavity compression is not functioning routinely, rotator cuff atrophy could occur. The glenohumeral joint functions best when concavity compression is achieved, and the body will still attempt to achieve the desired tasks by substitution patterns incorporating the scapula. It is suggested from the literature that upper trapezius activation increases to compensate for dysfunction of the rotator cuff or diminished concavity compression during tasks requiring arm elevation. Additionally, the pectoralis major, minor, and latissimus dorsi can be inappropriately used as stabilizers in the presence of long-standing dysfunction. The repetitive use of these muscles may contribute to the addition of muscular pain from myofascial trigger points [45]. Tight pectoralis major and minor muscles can alter scapular position toward a more anteriorly tilted and internally rotated position [7]. A tight latissimus dorsi can limit humeral elevation and external rotation [46]. Over time, as these tissue inflexibilities are maintained, abnormal scapular motor pattern eventually becomes the default pattern with arm movements resulting in altered motor control. Since these movement patterns of the scapula and arm are not common, deleterious effects such as subscapular bursitis (snapping scapula), chronic pain, spasm, and abnormal motor timing may arise. Therefore, the integrated design of the kinetic chain approach may be difficult for patients with complex scapular dysfunction to perform and would thus require modification to limit the number of segments being utilized during maneuver performance. Clinician must consider all factors that can be causing these alterations so common deficiencies and examples of possible kinetic chain exercise modification are detailed below [31, 32].

Address Proximal Deficits

Kinetic chain-based upper extremity rehabilitation requires enhancements to be made to a

deficient core. The enhancements should serve as the foundation for what is known as integrated rehabilitation [31, 32]. Integrated rehabilitation utilizes core function where the legs, hips, and trunk drive the arm throughout movements. Ideally, rehabilitation should first be focused on developing core strength and stability to optimize anatomy. The next step would be to integrate core function with correctly coordinated shoulder tasks. Finally, patients should be progressed from individual to complex tasks which adequately directs and educates the motor system to perform optimally thereby reducing redundancy in the system [33].

Patients with chronic complex scapular dysfunction will often not be able to immediately begin performing routine dynamic tasks even with core stability developed. For example, asking a patient to carry out an exercise that requires arm elevation in isolation could exacerbate their current symptoms due to the long lever arm requirements especially if the lower extremity components of the kinetic chain are not engaged. These patients seem to have an absence of a proprioceptive-kinesthetic sense to move the scapula properly during this task. The first modification to overcome this issue would be to employ conscious correction of the scapula as a foundational exercise (Fig. 17.8). If this technique is not successful, incorporation of a mirror to provide visual feedback for scapular positioning would utilize the principles of motor control and motor reeducation [47, 48]. The visual knowledge of results would allow the patient to have real-time feedback regarding scapular positioning.

Most postural concerns can be addressed by improving the flexibility of the musculature and/or the mobility of the structural components. The flexibility of both the upper and lower extremity can be increased via standard static, dynamic, and/or ballistic stretching. Based on previous findings regarding flexibility deficits in upper extremity dominant athletes, the hamstring, hip flexor, hip adductors, hip rotator, and gastrocnemius/soleus muscle groups should be targeted for the lower extremity. Improving lower extremity muscle flexibility has been linked to improving lower body movement patterns and improving overall athletic performance [49–52]. The pectoralis minor, latissimus dorsi, and posterior shoulder muscles should be the point of focus for the upper extremity [53–56]. However, clinicians treating soft tissue inflexibilities at or around the shoulder in patients with chronic complex scapular dysfunction should consider time and load applied during stretching [57]. The scapular and shoulder muscles in these patients are often hypersensitive when stretching to natural limits especially when the amount of pressure applied to the limb being stretched as well as the time the stretch is held is not accounted for. In these instances, using the principles of prolonged persistent stretching and accounting for total end range time may be useful [57, 58].

Fig. 17.8 Before performing any arm movement, patients are instructed to consciously retract the scapulae

Facilitate Scapular Motion

Peri-scapular muscles such as the serratus anterior and lower trapezius are a focus point in early rehabilitation. The previously described kinetic chain approach advocated early training which incorporated the trunk and hip in order to facilitate proximal to distal sequencing of muscle activation. This method may need to be adjusted in the most complex cases of scapular dysfunction. First, scapular rotation occurs during arm elevation; however, in the reeducation of scapular muscle function, we have found focusing on isolated translation is more beneficial. Many patients have poor postural and proprioceptive awareness of the scapula. Therefore, focusing on simple translation movements first appears to enhance awareness while activating scapular musculature. Performing these exercises in a closed chain fashion may facilitate proprioception, reduce degree of freedom, and lessen muscular demand on irritated scapular muscle tissues [59].

Closed chain exercises in the upper extremity have the ability to stabilize structures by facilitating joint congruity and co-activating surrounding musculature [60, 61]. This involves placing the distal segment (either the hand or elbow) on a fixed surface such as a table or a movable surface like a ball to facilitate compression of the humerus toward the scapula. Closed chain exercises are an appropriate rehabilitation method in restoring and improving proprioceptive-kinesthetic awareness. An example of incorporating closed chain exercise would be to instead of asking a patient to perform scapular retraction and humeral external rotation (at the side) while standing, the patient would perform scapular retraction while sitting and the arm supported by a table (Fig. 17.9). Sitting removes the movement that would otherwise come from the lower extremity segments yet still allows the trunk and core to be utilized in the performance of the exercises. This rationale follows the theoretical principles established by Bernstein where the fewer degrees of freedom would allow the motor system to optimize basic movements prior to transitioning to larger more functional movements that require greater degree of freedom [62, 63].

Fig. 17.9 An example of arm supported external rotation. The arm remains rigid on the table while the trunk is rotated laterally

Once the patient has shown improvement with the simple exercises, progression to complex kinetic chain movements such as supported arm elevation while sitting (Fig. 17.10) and then eventually arm movement while standing would be encouraged. Utilizing the trunk and/or lower extremity in order to promote coordinated scapular motion is ideal in that it mimics kinetic chain sequencing. Minimal stress is placed on the glenohumeral joint during trunk extension which can facilitate scapular retraction (Fig. 17.11).

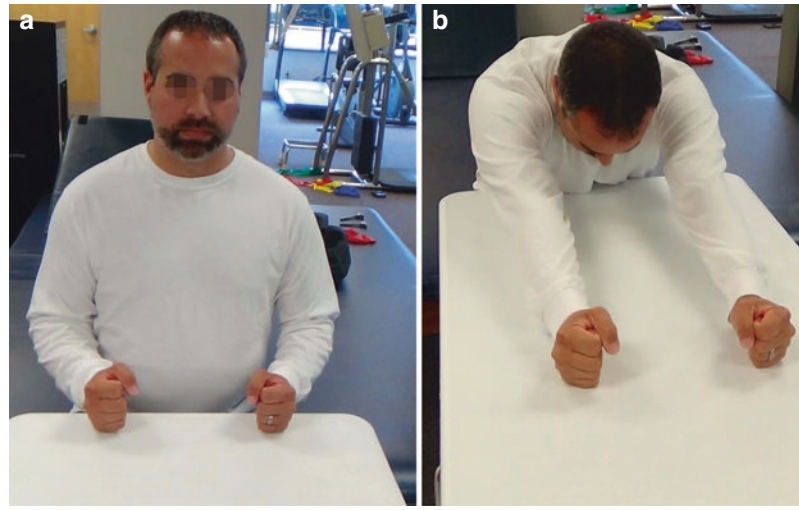


Fig. 17.10 Closed chain arm elevation is performed sitting (**a**) with the patient instructed to allow the trunk to drive the arms into elevation (**b**)

Fig. 17.11 To facilitate scapular retraction and posterior tilting, the patient is positioned sitting with the arms at the side internally rotated (**a**). The patient is instructed to sit up tall and externally rotate arms (**b**)

Fig. 17.12 To facilitate scapular retraction and depression, the patient is positioned sitting with the arm across the trunk (similar to a sling position) (**a**). The patient is instructed to rotate the trunk and look over the shoulder (**b**)

Additionally, when a patient is having difficulty with arm movement due to scapular dysfunction, it is imperative to utilize other segments. This is the time to expand the degrees of freedom during the motions. Specifically, using trunk movement to achieve arm elevation allows the dysfunction at the scapula and shoulder to be overcome.

As the shoulder heals and is ready for motion and loading, the movement patterns of activation using both ipsilateral and contralateral leg motion could be introduced.

As the final modification of the rehabilitation program, exploitation of the transverse plane should be attempted as this will help accentuate both scapular retraction and protraction (Fig. 17.12). By forcing proximal stability, the hip and trunk muscle activations, which have been demonstrated to precede arm motion, will be more efficient during a specified task [64]. In addition to generating and transferring energy to the distal segments, this component of rehabilitation allows the utilization of the stable base for arm motion [65]. Rehabilitation programs should attempt to encourage stimulation of proper proprioceptive feedback as well, so the patient can return to their desired level of function [31, 32, 60].

Case Examples

Two case studies have been shared to illustrate the design of a rehabilitation program for a patient with complex scapular dysfunction.

Case #1: A 15-year-old female multi-sport athlete (basketball and soccer) presented to her physician's office complaining of right shoulder and scapular pain that had been present for approximately 12 months. She reported no known mechanism of injury. She noted that her shoulder symptoms began with non-painful "snapping" which after a few months progressed to a more pronounced "catch" with pain during arm movement. She cannot use the arm for balance during activities because it is too painful to move into flexion and abduction. Her physician ordered various imaging with MRI revealing possible labral injury with "tumors" on her scapula. Electromyographic (EMG) testing was unremarkable suggesting no neurological involvement. The patient and her parents elected for her to undergo surgery for removal of the subscapular bursa as it was believed to be contributing to the catching sensation but chose not to address the possible labral pathology. Following surgery, the patient had reduced catching and snapping; however, scapular movement became dysfunctional. She returned to participating in soccer; however, the pain increased and became more severe with basketball activities so that she had to stop, choosing to seek further evaluation from a different physician.

On her initial examination with the second physician, the following physical findings were documented:

- Severe scapular dyskinesis with multiple different movement patterns
- Postural abnormalities including anteriorly rotated and downward sloping right shoulder girdle, an anteriorly tilted and superiorly elevated right scapula, forward head positioning, posteriorly rotated pelvis with lumbar lordosis, and flattening of the cervical and thoracic spine
- Inability to raise the right arm anteriorly and laterally beyond 75° without severe scapular dystonia
- Marked lower trapezius weakness 3/5 manual muscle testing grade
- Palpable pain and tightness over the latissimus dorsi, pectoralis minor, and upper trapezius
- Dynamic hip weakness per a single leg squat maneuver
- Negative labral and rotator cuff findings

The diagnosis by the second physician was determined to be scapular dystonia with lower trapezius deficiency.

Initial treatment included commonly utilized kinetic chain-based exercises such as the maneuvers contained within the Appendix. The exercises were demonstrated to the patient who returned to her home state to perform the regimen. The patient returned for follow-up 6 weeks later demonstrating improved core strength and stability, active range of motion, and reduced scapular dystonia. However, scapular control remained poor, and pain continued. After a consultation with the treating physician and two physical therapists, the concern was that the motor control aspects related to this patient were not improving, and the motor patterns may be deeply ingrained. Surface EMG was utilized to attempt to verify muscle activation patterns and intensity.

Application of the surface EMG revealed excessive latissimus dorsi activation during arm lowering with simultaneous lower trapezius activation. In addition to these demonstrable muscle activation alterations, the patient's scapula quivered during both arm elevation and lowering highlighting the scapular dysfunction. Additionally, the patient developed infraspinatus atrophy. These findings lead the treatment team to modify the kinetic chain-based program. With the intent of restoring the neural pathways for the neuromuscular activity to a more typical pattern, the exercises were changed by unloading the arm via the combination of closed chain arm support while asking the patient to sit during exercise

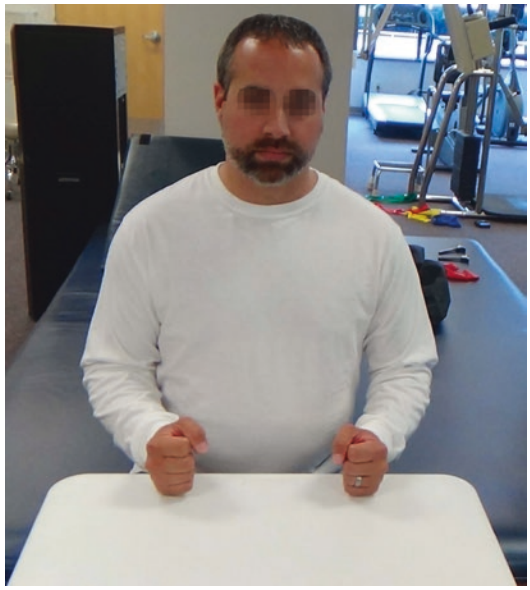


Fig. 17.13 The patient is positioned sitting with both arms resting on a table. The patient is instructed to minimize shoulder movement while moving one hand in circular directions three to five times for 10–20 s each

Fig. 17.14 As a progression to the closed chain arm elevation, patients may be advanced to supported arm elevation in the plane of the scapula

performance. For example, the patient was asked to sit upright while contracting the core muscles in order to stabilize the central segments of the body. She was instructed to consciously position the scapula in retraction and depression. The arm was supported on a platform in front of the patient while she was instructed to perform small, clockwise movements with the arm (Fig. 17.13). This exercise was used to improve the firing timing sequence of the serratus anterior, rhomboids, lower trapezius, and rotator cuff musculature while attempting to inhibit the firing of the upper trapezius and latissimus dorsi. Next, the patient was positioned sitting with the support platform to her side. Using a similar trunk position as the first exercise, she was next asked to place her arm across her trunk (similar to wearing a sling) and to maintain the arm position. The patient was instructed to rotate the trunk laterally while keeping the arm position (Fig. 17.9). This exercise was designed to facilitate the lower trapezius by gravity assisting scapular retraction and depression with trunk rotation. Additional modifications were made as illustrated in Figs. 17.14, 17.15, and 17.16.

In this case example, the large movements typically employed with kinetic chain-based programs were not able to be performed by a patient with complex scapular dysfunction. The modifications to the exercises were intended to continue treating the body as a unit which was evident through the complementary movements of the trunk during exercise performance. The patient responded well to the unloading of the arm and facilitating the scapular motion with gravity and trunk mobility in conjunction with the restricted range arm movements. She not only found relief of pain but a decrease in her scapular dystonia when smaller shoulder and scapular movements were employed.

Case #2: A 46-year-old male patient presented with severe right scapular pain and inability to use his right arm in forward flexion or during overhead activities. His initial injury occurred when he was attempting to cut steel bolts as part of his regular work duties. In the middle of a cut, the tool slipped causing the patient's arm to be forcefully distracted away from his body. He immediately felt a pop and burning pain over the medial border of his right scapula. He underwent numerous physician

Fig. 17.15 The seated sternal lift begins with the trunk slightly flexed and the arms internally rotated resting on the table (**a**). While keeping the arms at the side of the body, the patient is instructed to stand up tall and externally rotate the hands which assist in retracting the scapulae (**b**)

evaluations, receiving diagnoses of impingement, rotator cuff tendonitis, and cervical radiculopathy. After multiple months of rehabilitation and little to no relief in pain, he was referred to our office where he was diagnosed with a scapular muscle detachment [9]. Surgical treatment was selected which revealed that both his lower trapezius and rhomboids were detached from the scapula. The muscles were reattached, and the patient performed approximately 8 months of postoperative physical therapy [9].

The surgical intervention and subsequent postoperative rehabilitation reduced the pain; however, the patient began to develop kinesophobic traits where he was afraid to move his arm forward due to the fear of the presurgical pain returning. On a follow-up visit 18 months after the surgery, the patient stated that "I can move my arm only if I think about. When I move my arm without thinking, the pain in the shoulder blade and under my armpit takes my breath away." He also reported hypersensitivity with low-level nociception.

Based on his statements, it was decided that the next treatment approach would be rooted in the principles of motor control, mainly neuroimaging. The thought was that there might be a disconnect between body perception and pain processing similar to an amputee experiencing phantom limb pain [66]. During the same follow-up visit, the patient was positioned standing with two full-length mirrors in front of him and one mirror perpendicular to his body. The perpendicular mirror impeded the patient's view of his involved arm while only being able to see the non-involved arm. The patient was instructed to focus on the reflection of the non-involved limb in the mirrors, so it appeared as

Fig. 17.16 The asymmetrical push-up is performed with hand position staggered on a wall (**a**). The patient is instructed to move the body forward but only until the forearms are touching the wall (**b**)

though he was looking at a full image of his body. When the patient signaled that the image looked complete, he was instructed to elevate both arms forward up to 90° of elevation. After performing 12 repetitions, he was then instructed to go as far past shoulder level as he felt comfortable with.

With the use of the mirrors, the patient could raise both arms equally to approximately 110° of forward elevation with little reported pain. When the mirrors were taken away, the patient began to become inhibited again only being able to perform five repetitions of elevation before pain prohibited him from continuing. The patient was instructed to carry out a similar regimen for 20 min a day, 5 days a week until his next follow-up which was 3 months later. On the next follow-up, the patient had full arm elevation (160°) with little to no pain and without the use of the mirrors.

Pain sensation and their response are regulated by brain maps, the areas of the brain that process information which then send the information onto the appropriate structures to execute the response, which is activated by external and internal stimuli [66, 67]. In the case above, the mirror technique eliminated or decreased pain by altering the patient perception of body image. Pain and body image are described as being closely related, with the brain maps processing sensory input and also producing the image for the person [66]. Since the brain maps are designed to conduct both tasks, it is reasonable to consider that one task can influence the other. Historically, pain has been viewed as unidirectional with the pain traveling from the area of injury to the brain. However, in the case above, it appears as though the opposite occurred in that the brain projected pain onto the body which confounds the unidirectional pathway idea. The body perception concept is typically not considered as an intervention to control pain and function as most clinicians who treat musculoskeletal conditions instruct patients to perform unilaterally, ipsilateral actions while focusing on muscle contraction sensations and global movements with little involvement of the non-involved side. The body image concept would suggest that bilateral movements may be beneficial as part of neuromuscular education [67, 68]. Theoretically, the ability to see both limbs during a dynamic task would help a patient "retrain"

the brain to perceive an appropriate, balanced body image thus improving function.

Summary

Rehabilitation of complex scapular dysfunction should follow a kinetic chain-based regimen that addresses specific deficits within individual links which can aid in restoring the natural proximal to distal muscle activation sequencing. The deficiencies can be addressed through a logical progression of therapeutic interventions focusing on muscle facilitation, flexibility, strength, proprioception, and, finally, endurance training with integrated kinetic chain components. Specifically, functional exercises designed to simulate loading stresses in the scapulohumeral complex should be implemented throughout the rehabilitation program in a logical stepwise manner. Preventative or prospective exercises to minimize future loading stresses should be included at the end of recovery as part of the return to function.

Appendix

Rehabilitation Progression Functional shoulder rehabilitation is an approach that challenges the shoulder to work at progressively higher levels of function ending ultimately with return to maximum activity. These challenges are applied clinically within familiar neuromuscular systems and mechanically sound kinetic chains. There is an emphasis on spinal posture and hip muscle activity *facilitating* the scapular and glenohumeral motion. Strength is built within functional movement patterns.

Foundational Kinetic Chain-Based Sample Program Tips: Increase the degrees of freedom by using multiple segments fluidly, i.e., exaggerate trunk flexion to attain extension so scapula can easily depress using gravity, rotate trunk to facilitate scapular retraction, side-bend trunk to facilitate improved rotation, and use quick stretch reflex to promote improved muscle contractibility.

Note: Cues may need to alter based on patient for same exercise and same problem.

- Sternal lifts (Fig. 17.1)
- Standing table slides (Fig. 17.2)
- Stagger stance for lawn mower in sling, shift weight back for trunk rotation (Fig. 17.3)
- Standing lawn mower (Fig. 17.4)
- Low row (Fig. 17.5)
- Progress low row to external rotation with arms at side which helps with spinal mobility via trunk extension, keep arms close to body (Fig. 17.6)
- Advance to fencing (Fig. 17.7)

Modification Sample Program Tips: Reduce degrees of freedom by limiting number of active segments, i.e., sitting position versus standing and reduce range of motion while allowing muscular activation of the main muscles while unloading the weight of the arn via closed chain maneuvers.

- Conscious correction of scapular position (Fig. 17.8)
- Trunk-driven external rotation using table for support (Fig. 17.9)
- Closed chain arm elevation (Fig. 17.10)
- Scapular retraction facilitated by trunk extension (Fig. 17.11)
- Scapular retraction facilitated by trunk rotation (Fig. 17.12)
- Stirring the pot (Fig. 17.13)
- Closed chain arm elevation in the plane of the scapula (Fig. 17.14)
- Seated sternal lifts (Fig. 17.15)
- Asymmetrical hand push-ups (Fig. 17.16)

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