Christie Clem

Frozen Shoulder

Background

Frozen shoulder is a long lasting painful condition that results in decreased range of motion at the glenohumeral (GH) joint.[1,2] The condition was first described in the late 1800's and referred to as scapulohumeral periarthritis.[3,4] In the 1930's it was called frozen shoulder by Codman and in the 1940's Neviaser referred to the condition as adhesive capsulitis.[3,5] Other names for the condition include stiff shoulder, the fifty year shoulder (in Asian countries), and contracture of the shoulder.[4] The variety of names can be contributed to the fact that frozen shoulder is still not well understood. As research attempts to explain the etiology and pathology of the condition the names for the condition have evolved. In 2011 the majority of the members of the American Shoulder and Elbow Surgeons agreed with the following standard definition for frozen shoulder: "a condition characterized by functional restriction of both active and passive shoulder motion for which radiographs of the glenohumeral joint are essentially unremarkable except for the possible presence of osteopenia or calcific tendonitis."[6p.323]

Epidemiology

Frozen shoulder has a prevalence rate of 2-5% in the general population.[2,7] Women are more affected than men. Frozen shoulder most commonly occurs between the ages of 40 and 65 years [2,7,8] with the most common age being in the mid-fifties.[2] Individuals with type I diabetes have a 40% chance of developing frozen shoulder while approximately 29% of people with type II diabetes will develop the condition.[7] Other conditions that are associated with frozen shoulder include thyroid disorders, Parkinson disease, autoimmune conditions, and heart disease,[7] Dupuytrens contractures and breast cancer treatments.[9] Having frozen shoulder in one arm increases the risk of getting frozen shoulder in the other by 5-34%.[8]

Anatomy

The glenohumeral joint is a ball-and-socket synovial joint that is surrounded by the GH capsule and ligaments. When the arm is relaxed at the side, the GH capsule is tight on the superior aspect and loose both anteriorly and inferiorly.[10] This laxity allows for full abduction and external rotation of the humerus. The GH capsule is strengthened by both the GH ligaments (superior, middle, and inferior) and the coracohumeral (CH) ligament. The three GH ligaments are thickened areas of that capsule. The superior GHL extends from the superior glenoid labrum to the neck of the humerus deep to the CH ligament.[10] The middle GHL originates on the superior anterior aspect of the labrum, inferior to the SGHL, and attaches on the proximal humerus inferior to the SGHL attachment. The inferior GHL is made up of an axillary pouch that is positioned between anterior and posterior bands.[10] The CH ligament originates on the coracoid process and joins with the anterior aspect of the GH capsule before inserting on the lesser and greater tuberosities of the humerus. Because the CH ligament attaches to both tuberosities, it crosses the bicipital groove and keeps the long head of the biceps tendon in place.[11] The triangular area between the coracoid process, the anterior aspect of the supraspinatus tendon, and the superior edge of the subscapularis tendon is known as the rotator interval.[11] The rotator interval capsule is a combination of the GH capsule, the CH ligament, and the SGHL.[11] (see Figure 1 in Appendix)

Pathology

The cause and pathology of frozen shoulder is still not well understood, but information has been gained through biopsies that have taken place during surgical interventions. One possibility is that frozen shoulder begins as an inflammatory response that is then followed by capsular fibrosis.[2,12] Because individuals have pain before they begin showing a decrease in range of motion, it is thought that the inflammatory process happens

before the fibrosis. Histological studies have found both inflammatory cells and an abundance of proliferating fibroblasts.[12] Studies have also identified the growth of new blood vessels and nerves within the GH capsule and supporting ligaments which can help explain the high levels of pain that is experienced by those with frozen shoulder.[8,12] The structures of the rotator interval including the GH capsule, CH ligament, and SGH ligament have been found to have significant fibrosis and contracture and be the primary area of change in individuals with frozen shoulder.[8,12] In addition to decreasing the laxity of the rotator interval, the overall GH capsule undergoes fibrotic changes that reduce the overall volume of the joint.[8] The structures of the rotator interval including the CH ligament resist external rotation (ER) when the arm is at the side; as the humerus is abducted, the laxity in the IGH ligament disappears and the IGH ligament prevents the humeral head from translating too far in the inferior direction.[10] As the capsule becomes fibrotic and contractures form, normal external rotation and abduction becomes limited.

Classification

A clearly defined classification system for frozen shoulder has not been consistently used throughout the literature. In 2011 the following classification system was proposed by Zuckerman in an effort to make clinical diagnoses more consistent and increase the ability to compare research studies.[6] Individuals who have a limitation in both active and passive range of motion of the GH joint when x-rays show nothing other than possibly osteopenia or calcific tendonitis, will be diagnosed with either primary or secondary frozen shoulder. Primary frozen shoulder is also referred to as idiopathic and there is no known cause or related condition. If the etiology or an associated condition can be identified, the person is classified with secondary frozen shoulder.[6] Secondary is divided further into three groups: systemic, extrinsic, and intrinsic.[6,8] Secondary systemic includes individuals with diabetes,

thyroid conditions, autoimmune conditions or any other systemic condition that has been associated with frozen shoulder. Secondary extrinsic includes pathologies that are not directly related to the shoulder such as but not limited to stroke, heart disease, distal arm fracture, and breast cancer treatment. Secondary intrinsic frozen shoulder is caused by a pathology within the shoulder itself (ex: rotator cuff injury, upper arm fracture, damaged labrum, acromioclavicular injury, biceps tendinopathy).[6,8]

Stages

Frozen shoulder is typically thought to progress through three stages (freezing, frozen, and thawing),[2,5] but others describe it as having four phases (Table 1 in Appendix).[8,9] There is no clear beginning and end to each phase, but instead they represent the clinical and histological progression that has been seen. Here it will be described as four stages. Stage 1 begins with a gradual onset of pain at the end ranges of GH motion, aching pain during rest, and more intense pain with movement.[8,9] People are often unable to sleep on the affected side and night pain is common. During stage 1 motion is restricted by pain, but when under anesthesia full range of motion is possible. Contractures are not present at this time, but there is an inflammatory synovial reaction.[9] Stage 2 is also called the "painful" or "freezing" stage; the intensity of pain often increases and motion becomes more limited in all directions.[9] Full range of motion is not possible under anesthesia due to a clear reduction in the axillary pouch.[9] Stage 3 is commonly called the "frozen" stage. The pain is often less common and less severe, but stiffness or lack of motion dominates.[9] During this time there is a decrease in the synovial inflammation and the number of blood vessels in the area, but a continued increase in the capsular fibrosis. A full loss of the axillary pouch has been found which goes along with the findings that there is no improvement in the range of motion when the individual is under anesthesia.[8,9] Stage 4 is referred to as the "thawing" or chronic

stage. There is little, if any, pain, but large range of motion losses are still present.[8,9] Gradually motion will improve although there is not specific consensus on how much motion will return or how long it will take. Frozen shoulder has been said to resolve in 12 to 18 months, but long-term studies show it may take several years and that full resolution is not a given.[8] A survey of over 200 patients found that 59% had a "near normal shoulder" 52 months after symptoms began according to the Oxford Shoulder Scale (OSS), 35% had mild/moderate symptoms and 6% had severe symptoms, but at three years 56% reported mild/moderate symptoms.[13] Another long-term study found 45% of patients had pain or range of motion restrictions when evaluated 40-48 months after onset.[14] Less than half of the 40% that had a limited range of motion, realized their motion was restricted.[14] This supports that the majority of individuals that experience frozen shoulder will have normal functional mobility three to four years after onset. Although the range of motion in most directions was significantly less than that of controls matched for age and sex.[14]

Diagnostics

Clinical diagnosis of frozen shoulder is based on the patient's history and physical exam.[2,7,8] The individual will present with a gradual onset of pain in a unilateral shoulder that often occurs at night and is severe enough to prevent lying on the involved side or wakes them during sleep. The end ranges of motion are also painful.[8] A definition that has been used in many studies is "ROM loss greater than 25% in at least two planes and passive external rotation loss that is greater than 50% of the uninvolved shoulder or less than 30 degrees of external rotation."[8] Reduction in external rotation, abduction, forward flexion, and internal rotation are the most common.[7] No specific capsular pattern has been identified. External rotation is more limited when the GH joint is at 0 degrees of abduction as compared to 90 degrees of abduction while internal rotation is the opposite (more

restricted at 90 degrees abduction).[15] Range of motion restrictions should have been present for at least one month,[8] but several studies have used a minimum three month duration as part of the diagnostic criteria.[15] Functional mobility will be limited due to the pain and decreased range of motion; placing a hand in a back pocket and reaching overhead or behind the neck can be difficult or impossible to perform.[2,8]

Other pathologies must be ruled out, especially during stage 1 of the frozen shoulder when there is not yet a passive range of motion restriction. The isometric strength of the rotator cuff is typically normal,[8,16] although pain related weakness may be present when near the end ranges of motion. Special tests that require the patient to actively or passively move to end ranges can produce false positive results. A main sign of frozen shoulder will be the firm or "tethered" end feel of the passive range of motion.[16]

Imaging studies can be used to rule out underlying pathologies such as osteoarthritis and rotator cuff tendinopathies, but are not the reference standard when diagnosing frozen shoulder. Radiographs should be normal except for possible osteopenia or calcific tendonitis.[6,8] MRI's are not necessary, but can help with differential diagnosis. MRI findings consistent with frozen shoulder include CH ligament and rotator interval thickening,[2,3,8] reduced axillary pouch,[8] and synovitis-like abnormalities around the long head of the biceps tendon.[3]

Interventions

Frozen shoulder is considered a self-limiting condition with a variety of interventions that can be utilized. Research has not found a definitive treatment approach, but multiple treatments have shown benefits. Because individuals recover at different rates and some have symptoms that are still present long-term,[8,13,14] patient education is vital. Information regarding the natural progression of the condition,[8,17] the importance of doing a daily home

exercise program,[8,17] and the need to match the intensity of exercises to the irritability level[17] should be provided. Common conservative interventions include corticosteroid injections, intra-articular dilation (distension) and physical therapy treatments such as modalities, joint mobilizations, and stretching, and a home exercise program. More aggressive approaches include manipulation of the GH joint under anesthesia and arthroscopic surgical release.

Oral medications, acetaminophen and non-steroidal anti-inflammatories, are typically used early for pain relief, but there is little research to support their use. A systematic review for NSAIDs related to multiple types of shoulder conditions found a statistically significant improvement as compared to placebo, but the studies used had small sample sizes and poor quality.[18]

A systematic review (SR) evaluating the effectiveness of GH corticosteroid injections found the injections provide short term relief for frozen shoulder.[7] These benefits are seen during stage 1 and stage 2 (freezing).[19] Statistically and clinically meaningful improvements for pain and function were observed at 6-weeks, but not longer. Corticosteroid injections outcomes at 12 weeks were similar to other interventions and placebo.[7] Injections resulted in a greater improvement in outcome measures as compared to physical therapy alone at a short-term 6-week followup.[7] Intra-articular injections resulted in greater improvements in pain and ROM as compared to extra-articular injections.[7] Corticosteroid injections work by decreasing the inflammatory response and decreasing pain,[8] so accurate placement of the medication could be important for the greatest benefits to be realized. Physicians are more accurate with injection placement when using image-guided injection techniques as compared to anatomical landmark injection techniques (92-100% accuracy vs. 26-79% accuracy).[7] The SR did not find a significant difference between the injection techniques, but

image-guided placement may result in better outcomes according to some individual studies.[2]

Intra-articular dilation or distension involves injecting the GH joint capsule with saline or a saline/corticosteroid combination. The saline is injected into the capsule under pressure, in an effort to expand the capsule since the overall volume is typically reduced with frozen shoulder.[8] A systematic review found short-term improvements in pain, range of motion, and overall shoulder function when distension was done with a combination of saline and corticosteroid, but distention with corticosteroids has not been shown to be better than only a corticosteroid injection.[2]

Modalities can improve symptoms of frozen shoulder, but the use of modalities with other interventions and the low quality evidence makes it difficult to state if a single modality is effective when used alone or better than another.[8,20] A systematic review by Page et al. was only able to identify that low-level laser therapy (LLLT) may be better than placebo after six days of treatment and LLLT plus exercise may be better than exercise alone at four weeks for pain control.[20] A systematic review by Jain found strong evidence that LLLT improves pain, moderate evidence that it improves function, and no evidence that it affects ROM.[19] The addition of deep heat, short wave diathermy, to stretching made significant gains in ROM as compared to superficial heat/stretching or stretching alone. The deep heat also provided benefits in pain and function.[19] No benefits in pain, ROM, or function were found with ultrasound in the systematic review,[19] but ultrasound was found to increase only ROM immediately post treatment and at a 3-month follow-up in study by Dogru et al.[8] There is not strong evidence for the use of other modalities such as ice or electrical stimulation, but trying these interventions may be appropriate for individuals with high levels of pain.

Physical therapy exercise and mobilizations are highly recommended during stage 3 (frozen) and stage 4 (thawing) to assist with pain reduction, regaining ROM, and improving function.[19] The physical therapy intervention should be based on the irritability level of the patient; as the irritability/pain decreases the intensity of the intervention can increase to focus on regaining ROM (Table 2 in Appendix).[17] Patients with high irritability will perform easy, short-duration ROM exercises for pain reduction and receptor input; ROM exercises should be done 2-3 times a day for a few seconds in almost a pain-free range.[17] Pushing into the pain at this point has not been found to be beneficial.[8,17] The intensity and hold time of the stretch should increase as the pain decreases as well as the frequency of times performed during the day. A systematic review on manual therapy and exercise by Page et al. determined that manual therapy and exercise may not do as well as corticosteroid injections for short-term results.[21] A systematic review by Favejee et al. identified the following: high grade mobilizations improved GH mobility and decreased disability more than low grade mobilizations, posterior mobilizations increased external rotation more than mobilizations in the anterior direction, end-range mobilizations and mobilizations with movement were more beneficial than mid-range mobilizations, and mobilizations with movement were better than end-range mobilizations for scapulohumeral rhythm.[22] Overall, physical therapy exercise and mobilizations can improve range of motion. All patients should perform a HEP that consists of passive ROM exercises at the GH joint: flexion, extension, internal rotation, external rotation, horizontal adduction, and abduction (Figure 2 in Appendix).

If improvements do not occur with conservative interventions after six months[17] or after one year[2] referral to a physician for consideration of a more invasive procedure could be the next step. Manipulation under anesthesia (MUA) adequately increases the patient's

ROM, but does not allow the physician the ability to isolate specific tissues.[2,17] MUA involves stabilizing the scapula while the humerus is forcefully moved past end-range to "tear" the capsule. Multiple studies have had positive outcomes in 75-100% of the subjects undergoing MUA,[17] but there is the risk of fracture, nerve injury, muscle injuries, and labral tears.[23] Arthroscopic capsular release allows the physician to select specific parts of the capsule to release. Arthroscopic release has resulted in improved outcomes: less pain, improved ROM, and better function. A systematic review by Grant et al. found very little differences between the results of MUA and arthroscopic capsular release.[23] This SR found capsular release resulted in more abduction and external rotation, 6 degrees and 8 degrees respectively, as well as a slightly higher, but non-significant difference in a disability outcome measure. Similar complication rates were also found between capsular release and MUA, 0.5% and 0.4% respectively.[23] Manipulation is often done immediately following the arthroscopic capsular release to regain full motion. Arthroscopic capsular release combine with MUA demonstrated ROM improvements of ~70 degrees in flexion, ~30 degrees in external rotation, and internal rotation gains measured by reaching to L1 instead of L5 in a study of 50 patients that had failed over six months of conservative treatment.[24] Patients that have diabetes have outcomes that are significantly better following capsular release, but their improvements are significantly lower than those without diabetes and often have long-lasting limitations.[25]

Outcome Measures

The Disability of the Arm, Shoulder, and Hand Scale (DASH) and the Shoulder Pain and Disability Index (SPADI) are two of the most commonly studied shoulder outcome measures.[8,26] Because neither have been found to have floor or ceiling effects and both have good responsiveness, they are recommended in the outpatient setting.[26] The SPADI is more

responsive than the DASH for patients with frozen shoulder.[8] The DASH is a self-report questionnaire with 30 items that are scored on a one to five scale. The raw score is converted to a 0-100 scale with 0 meaning no disability and 100 fully disabled.[27] The minimal detectable change (MCD) is between 6.6 – 12.2 points (average 10) with 10.2 points as the change needed to be clinically meaningful.[8] The QuickDASH is a shortened 11-item questionnaire that also measures physical function in patients with an upper extremity condition. The QuickDASH has been found to be valid, reliable, and responsive, but the original DASH provides a more comprehensive picture.[28] The SPADI is a self-report questionnaire that assesses pain and disability. Five questions are about pain and 8 questions address disability for a total of 13 total items.[8] The MDC for the SPADI is 18 points while a change between 8 and 13 is clinically meaningful.[29,30] Since pain and limited ROM are the two primary complaints of frozen shoulder, both of these should be objectively measured, in addition to, using an outcome measure for disability. The visual analog scale can be used for overall pain, but can also be used in connection with specific activities that are difficult such as pain during sleep, reaching behind the back, or when getting dressed. Both active and passive ROM can be measured with a goniometer in the following planes of motion: flexion, abduction, internal and external rotation at 90 degrees of abduction (45 degrees if 90 is not possible), and external rotation at 0 degrees abduction.[8]

Conclusion

Frozen shoulder is considered a self-limiting condition that causes disability through high levels of pain and decreased ROM, but the underlying cause is not well understood.[2,8,9,12] Some researchers belief frozen shoulder begins as an inflammatory process which then transitions to a fibrotic condition resulting in a tightened GH joint capsule with reduced ROM.[2,12,17] The CH ligament, rotator interval, and axillary pouch are the structures most

commonly affected[3,8,11] resulting in losses of external rotation, abduction, and flexion. The condition progresses through four stages: 1) early pain with full ROM, 2) "freezing" or "painful", high levels pain, limited motion, 3) "frozen", less pain, increased ROM restrictions, 4) "thawing", little to no pain, large ROM deficits, ROM begins to improve.[8,9] Resolution often occurs within one to four years, but complete resolution may never occur.[13,14] Conservative treatment should be attempted for six months to 1 year before surgical interventions are explored. Patient education about the progression of the condition and the importance of a daily HEP should be provided. There is not evidence to support a specific conservative intervention as the best,[2,7,17,19,20,22] but the pain irritability level of the patient and the stage of the progression should be considered when deciding a treatment approach.[2,17] Intra-articular corticosteroid injections are effective at reducing short-term pain and should be used during the early stages.[2,7,8] Physical therapy (exercise, ROM, mobilizations, modalities) is important for improving ROM especially during the "frozen" and "thawing" stages.[9,17,19,22] More invasive treatment options such as MUA and arthroscopic capsular release are available for patients that have persistent disability when conservative methods fail. [2,8,17,23]

Resources

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Appendix

Figure 1 – Rotator Interval Anatomy[11] – Petchprapa et al. 2010

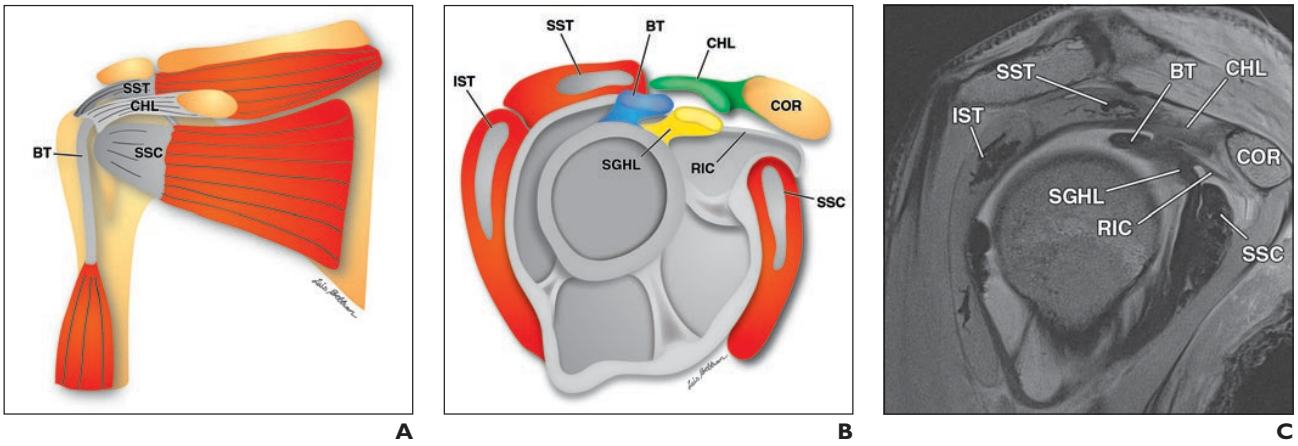

Fig. 1—Rotator interval.
A–C, Illustrations in coronal (**A**) and sagittal (**B**) planes and corresponding sagittal MR proton density–weighted arthrogram (**C**) show boundaries of rotator interval, which are defined by coracoid process (COR) at its base, superiorly by anterior margin of supraspinatus tendon (SST) and inferiorly by superior margin of subscapularis tendon (SSC). Contents of rotator interval include long head of biceps tendon (BT), coracohumeral ligament (CHL), superior glenohumeral ligament (SGHL), and rotator interval capsule. Rotator interval capsule (RIC) is anterosuperior aspect of glenohumeral joint capsule, which merges with CHL and SGHL insertions medial and lateral to bicipital groove. CHL arises from base of coracoid process, traverses through subcoracoid fat, and inserts on anterior humerus. IST = infraspinatus tendon.

Table 1 – Four Stages Adhesive Capsulitis[9] – Neviaser 2010 p. 2347

TABLE 1
Stages of Adhesive Capsulitis

	Symptoms	Signs	Arthroscopic Appearance	Biopsy
Stage 1	Pain referred to deltoid insertion Pain at night	Capsular pain on deep palpation Empty end feel at extremes of motion Full motion under anesthesia	Fibrinous synovial inflammatory reaction No adhesions or capsular contracture	Rare inflammatory cell infiltrate Hypervascular, hypertrophic synovitis Normal capsular tissue
Stage 2	Severe night pain Stiffness	Motion restricted in forward flexion, abduction, internal and external rotation Some motion loss under anesthesia	Christmas tree synovitis Some loss of axillary fold	Hypertrophic, hypervascular synovitis Perivascular, subsynovial capsular scar
Stage 3	Profound stiffness Pain only at the end range of motion	Significant loss of motion Tethering at ends of motion No improvement under anesthesia	Complete loss of axillary fold Minimal synovitis	Hypercellular, collagenous tissue with a thin synovial layer Similar features to other fibrosing conditions
Stage 4	Profound stiffness Pain minimal	Significant motion loss Gradual improvement in motion	Fully mature adhesions Identification of intra-articular structures difficult	Not reported

Table 2 – Treatment Strategies Based on Irritability Level[17] – Kelley et al. p. 140

TABLE 4 Treatment Strategies Based on Irritability Level

	High Irritability	Moderate Irritability	Low Irritability
Modalities	Heat/ice/electrical stimulation	Heat/ice/electrical stimulation	...
Activity modification	Yes	Yes	...
ROM/stretch	Short-duration (1-5 s), pain-free, passive AAROM	Short-duration (5-15 s), passive, AAROM to AROM	End range/overpressure, increased-duration, cyclic loading
Manual techniques	Low-grade mobilization	Low- to high-grade mobilization	High-grade mobilization/ sustained hold
Strengthen	...	...	Low- to high-resistance end ranges
Functional activities	...	Basic	High demand
Patient education	+	+	+
Other	Intra-articular steroid injection	...	...

Abbreviations: AAROM, active assisted range of motion; AROM, active range of motion.

Figure 2 – Passive ROM Exercises for HEP – Kelley et al. p.140

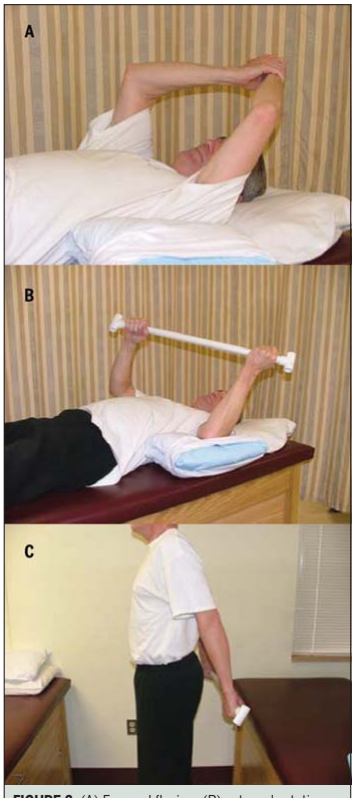

FIGURE 2. (A) Forward flexion, (B) external rotation, (C) extension.

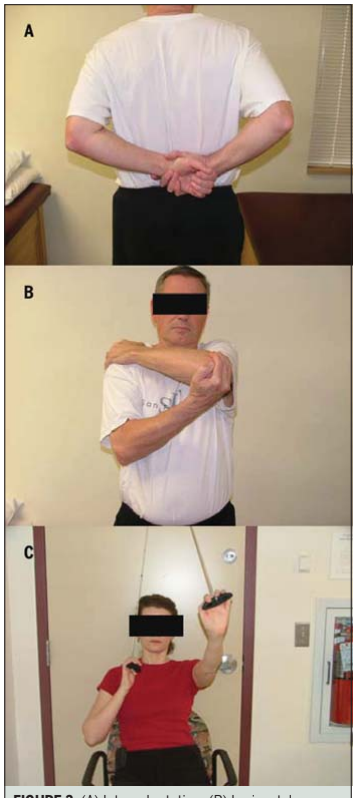

FIGURE 3. (A) Internal rotation, (B) horizontal adduction, (C) pulley for elevation.