Rehabilitation for rotator cuff tears

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Rotator cuff pathology is an extremely common problem in adult patients. While rehabilitation of the postsurgical rotator cuff patient is challenging, it is a problem best managed with the combined input of the patient, therapist, and treating orthopaedic surgeon. Rehabilitation protocols should respect the balance between risk of rotator cuff retear from early aggressive therapy and the development of shoulder scarring and stiffness from prolonged immobilization. It should progress at a pace that prevents shoulder stiffness, but respects the timelines of tendon healing biology. The purpose of this article is to provide an evidence-based approach, using the available basic science and clinical literature, to illustrate rehabilitation guidelines after surgical repair of the rotator cuff.

Key words: **Rotator cuff - Shoulder - Rehabilitation.**

Rotator cuff (RC) tears are one of the most common shoulder pathologies seen in adult patients. After failing a course of nonoperative therapy, RC tears are typically repaired. Multiple factors are involved in the strength of the repair, including surgical materials, and technique along with tissue quality.[1-4] A wide range of results have been reported for both open and arthroscopic RC repairs, but overall good results can be anticipated in combination with appropriate postoperative therapy. The literature demonstrates improved postoperative pain scores and increased strength in patients that have repairs of acute RC tears *versus* chronic tears and for tears that are small *versus* larger or massive tears.[5-8]

Successful rehabilitation of the RC depends on collaboration and good communication between the orthopaedic surgeon and the physical therapist. Collaboration is critical because the outcomes of RC repair are affected by the age of the patient, activity level, duration of impairment, size and location of tear, tissue quality, degree of muscle atrophy, number of cuff tendons involved, and concomitant shoulder and neck pathology. The treating orthopaedist and the physical therapist both have unique insights into each of these variables, and, therefore, mutual input is critical for success. From a joint perspective, an evaluation-based approach can successfully be employed.[9] An evaluation-based approach takes consideration of both the duration to healing and patient-specific goals. The objective of this article is to discuss rehabilitation guidelines that are consistent with the available literature and take consideration of patient-specific factors.

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Basic science

It is critical to understand the anatomy and biomechanics of the RC in order to appreciate this mechanism in a recovery or rehabilitation state. The RC is an important stabilizer of the glenohumeral joint, chiefly by depressing the humeral head and compressing it within the glenoid. With a deficient RC, proximal humeral head migration is noted with subsequent impingement on anterolateral acromion. However, many patients maintain a well stabilized humeral in the face of a complete tear of one of the four RC tendons.[10, 11] This stability appears to be due to the interlocking nature of the RC fibers. Histological analysis demonstrates what appear to be four interlocking layers of RC tissue.[12] The interlocking nature of the RC connects one tendon to the next, thus allowing the *subscapularis* to act as a humeral head depressor in the face of a complete *supraspinatus* tear. In biomechanical testing, normal humeral translation remained with simulated massive tears of the *supraspinatus* as long as the other RC tendons remained intact.[10] The *infraspinatus* and *subscapularis* are more effective humeral head depressors because of their relative force vectors.[11] When more than one tendon becomes compromised, the benefit of a redundant interlocked system becomes less efficient. In addition, *teres major* and *latissimus dorsi* also play a role in humeral head depression; so even in massive polytendinous RC tears, rehabilitative shoulder strengthening can still be effective in humeral head stabilization.[11]

The role of the long head of the biceps in glenohumeral stabilization remains more nebulous. Some studies indicate that it may have a role as a humeral head depressor or assist in anterior stabilization.[13] Whatever its biomechanical role may be, its absence after tenolysis or tenodesis does not appear to generate a clinically significant deficit. However, a degenerative long head of the biceps is well known to be a concomitant pain generator in the presence of RC disease.

Scapular dyskinesia is frequently present in face of RC pathology.[14] Abnormal scapular kinematics and even scapular winging have been noted in patients with RC disease. Scapular dyskinesia alters the inclination of the acromial arch, thereby compounding RC dysfunction.[14] Appropriate therapy for scapular dyskinesia should emphasize strengthening and normal recruitment patterns of the periscapular muscles.

The subacromial bursa has a significant role in impairment with RC disease.[15-17] This is largely because of the rich innervation of the bursa including free nerve endings, Ruffini corpuscles, and Pacinian corpuscles.[15] In patients with known chronic RC degeneration, the bursa itself has been found to secrete bone morphogenic proteins (BMPs) which are thought to induce degeneration of the RC by activating osteogenesis.[16] Thus, a biologically active and inflamed bursa may be detrimental to the cuff itself.[16] When the bursa develops adhesions, a mechanical impingement can develop between the RC and the acromium.[17]

Pathomechanics and pathophysiology

Multiple etiologies have been proposed as to why the RC fails.[18-20] Neer proposed the extrinsic theory in which external factors such as the acromium lead to impingement and subsequent RC tears.[18] Nirschl disputed this idea by suggesting that failure is due to the intrinsic nature of a weakening RC rather than external forces. He found no true inflammation in the degenerative RC, but rather angiofibroblastic dysplasia, suggesting that the cause was not extrinsic mechanical disruption but rather a histologic change in the RC tissue.[19] Some presented evidence that RC failure is due to repetitive tensile microtrauma. Others suggest that it is due to progressive hypovascularity of the RC. The classic teaching was that the critical zone of the hypovascular cuff was at the deep surface, anterior edge of the *supraspinatus*, 1 cm proximal to its insertion. Recent studies have discounted that idea by using laser Doppler to show that substantial blood flow exists in the critical zone.[20]

Whatever the mechanism of failure, it is clear that due to their viscoelastic nature, the

RC tendons tolerate exceptional tensile loads – up to 100 N/mm. Conversely, the RC is comparatively weak in resisting compressive and shear forces.[21] Biomechanical work demonstrated the RC experienced greater strain in positions of increasing abduction and with the existence of partial RC tears. This study suggests the position and origin of how large tears may be initiated. Furthermore, stiffness of the cuff was significantly decreased on the articular side of the tendon where tears typically originate. The *anterior supraspinatus* has an increased muscle-to-tendon ratio, a fact, which may explain why tears progress from anterior to posterior.[22]

As supported by Nirschl's work, RC degeneration or tendinopathy seems to also play a role in the development of full-thickness RC tears.[19] As the RC weakens, extrinsic and eccentric overload likely lead to further microtrauma and eventual tear.[23] The tendinopathy does not appear to be an evolving inflammatory process but a transformation of the RC tissue to angiofibroblastic material.[19] This transformation results in a loss of the biomechanical integrity of the cuff and eventual complete tendon failure.

Healing potential

Full-thickness RC tears do not heal without surgical augmentation. No evidence exists of spontaneous healing. This should be kept in mind by a patient's therapist if a trial of non-operative therapy is to be prescribed for a full-thickness tear. Therapy should focus on periscapular musculature strengthening and strengthening of the remaining RC muscles. After the tendon is repaired, most healing appears to be from the bone, but some authors argue that the bursa itself has a role in repair.

Timeline of tendon healing

As is also true with the flexor tendons, the RC tendons heal in three phases: 1) inflammatory, 2) proliferative, and 3) maturation phase. The first phase, which occurs within the first week of injury or repair, is one characterized by inflammation. Platelets induce clot formation, and fibrin and fibronectin cross-link with collagen fibrils. This creates a weak lattice that helps to control further bleeding. Polymorphonuclear leukocytes and other granulocytes are attracted by chemokines to assist in the inflammatory process, releasing histamine and bradykinin to increase vascular permeability. This, in turn, brings more inflammatory factors to the recently repaired tissue.

The second phase, the proliferative, replaces the inflammatory phase at 2-3 weeks after repair. Fibroblasts, myofibroblasts, and endothelial cells replace the leukocytes and inflammatory mediators seen in the inflammatory phase. Capillary buds begin infiltrating the tissue and granulation tissue appears. The granulation tissue begins to replace the immature lattice created during the inflammatory phase. Type III collagen and glycosaminoglycans are dispersed in a random pattern by fibroblasts.

After 3 weeks, the maturation or remodeling phase takes over. Biosynthesis slows and tissue reorganization begins to form a mature scar. Type I collagen replaces the type III collagen seen in the proliferative phase. The new collagen fibrils align to resist tensile forces rather than organizing in the haphazard fashion seen in the previous phase. Remodeling continues until mature scar tissue replaces the weaker proliferative tissue. Different animal studies show a wide variability in the time it takes for the maturation phase to reach a mature tendinous scar capable of resisting the forces experienced by the RC.[24, 25] Some studies show a mature repair at the 12-16 week period while others report the tendon is not at a mature state until the 26th week.[24, 25] The wide variation suggests that that active strengthening of the RC should proceed slowly and not until the biomechanical integrity of the RC has been restored.

Immobilization

Following RC repair, most patients are initially immobilized in some type of sling

because inadvertent or excessive early motion could jeopardize the repair.[26-28] More surgeons are now using an abduction immobilizer rather than a traditional sling. The idea behind the abduction immobilizer is that it places the shoulder in a more optimal position for RC healing and minimizes tension on the repaired tissue. There is evidence from animal models that this device may be superior to the traditional sling. When the arm is in the traditional adducted position while in a sling, the blood flow to the humeral head is more impaired than in the abducted position.[26] Furthermore, there is evidence in the sheep model that immobilization reduces tension on the repaired tissue.[27] Hatakeyama *et al.* reported the RC repair is in its safest position at 30° of elevation in the scapular plane and with external rotation (ER) limited from 0 to 60°.[28] In the rodent model, immobilized repaired *supraspinatus* tendons were compared to those allowed free range of motion (ROM). The immobilized tissue demonstrated higher collagen orientation, increased viscoelastic properties, and more genes expressing extracellular matrix proteins.[2]

The above evidence suggests that immobilization for 4 to 6 weeks with an abduction-type brace is warranted.[27, 28] We recommend that the arm be placed in the device at 45 to 60° of abduction to minimize tension and improve blood flow to the repaired tissue. Surgeons and therapists should be cognizant of their patients' comfort in these devices, as some patients find them to be unwieldy while others find them to be protective and supportive.

Continuous passive motion

The evidence to support the use of continuous passive motion (CPM) devices for the shoulder is scarce. CPM has been shown to improve cartilage metabolism in joints other than the shoulder, but its role in shoulder rehabilitation remains unclear. In a prospective, randomized, blinded, controlled study, 26 patients who underwent RC repair and subacromial decompression were compared with CPM and standard rehabilitation *versus* standard rehabilitation alone. At 3 months, there was no difference in the shoulder scores between the 2 groups, however, the CPM cohorts had a greater ROM. The paper noted that there was no added detriment to the RC repair by the early use of CPM.[29]

A second prospective randomized study included 31 patients who underwent RC repair and compared CPM for the first 4 weeks following surgery *versus* supervised passive ROM. At a mean follow-up of 22 months, there was no difference in standardized outcome measures, pain scores, ROM, or isometric strength. The supervised passive ROM was noted to be more cost-effective.[30]

From the existing evidence, there does not appear to be enough data to support the use of CPM. Furthermore, in light of the expense of these machines and the fact that many patients find them awkward and even painful, their use should be limited to specific circumstances where the development of post-surgical adhesions is likely and postoperative motion is a premium.

Physical therapy modalities

Various physical therapy modalities exist for the treatment of the RC after repair. These include transcutaneous electrical stimulation, iontophoresis, nerve stimulation, and ultrasound. As the data from controlled studies involving RC rehabilitation is scarce, their implementation remains controversial for this purpose. Their effect on tendon healing is also unknown. Extrapolating from other data in the physical therapy literature, these devices may have a role in pain reduction and improving motion and, therefore, the therapist and treating orthopaedic surgeon should discuss each modality's use for patient-specific circumstances.

Cryotherapy

Cryotherapy is a commonly applied modality among therapists and trainers in the pres-

ence of pain and trauma.[31-33] Its benefits include pain reduction, reduction of inflammation and decreased cell metabolism. Pain reduction has been reported to occur at temperatures between 10-15º Celsius.[32, 33] In a randomized, controlled trial investigating the effects of continuous cryotherapy on postoperative arthroscopic and open RC repairs, Singh *et al.* found that using temperatures between 7.2 and 13º Celsius helped to decrease pain, improve restful sleep and comfort during a 21-day regimen of use.[31] Speer *et al.* documented similar results during the first 24 h of use, while additionally noting decreased need for pain medication and less pain during motion 10 days postoperatively.[32] Osbahr *et al.* demonstrated that continuous use of cryotherapy after open RC repairs significantly reduced subacromial and glenohumeral joint temperatures compared to controls. Increases in joint temperature can stimulate the production of proteolytic enzymes, which have been shown to have adverse effects on joint cartilage.[33]

Transverse friction massage

Cyriax had advocated the use of transverse friction massage for tendonitis/tendinosis,[34] however, currently no literature exists to support its use for RC rehabilitation after surgical repair. Like other physical therapy modalities, its use should be patient-specific rather than a universal implementation.

Rotator cuff impingement and partial thickness rotator cuff tears

Subacromial impingement and partial RC tears less than 50% of the cuff thickness are typically treated with nonoperative therapy and other nonsurgical modalities. More than 70% of patients with subacromial impingement will have good-to-excellent results with therapy alone. The natural history of partial thickness tears is less clear. Nonetheless, therapy for these conditions should focus on regaining full ROM, synchronizing the activation of the RC muscles and periscapular musculature, and normalizing glenohumeral and scapulothoracic kinematics.[35] Overhead strengthening programs should emphatically be avoided as they place the RC in a position of impingement with the acromial arch. Another goal of therapy should be to improve internal rotation (IR) lost from posterior capsular contraction as this type of contracture causes increased antero-superior translation of the humeral head and thus worsens impingement symptoms.[35, 36] With strengthening of the RC muscles, superior head translation is also reduced and subacromial impingement symptoms should thereby be reduced. Joint mobilization used with a supervised strengthening program is more effective than exercise alone in respect to outcomes of strength improvement, pain reduction, and function.[37, 38]

Full-thickness rotator cuff tear

While there is no evidence that a full-thickness RC tear will heal without surgical treatment, therapy can be successful in patients with these lesions. However, the problem remains that we have no pretreatment measures to predict who of these patients will have good results with nonoperative management *versus* those patients who will continue to have pain and functional limitations. Electromyographic (EMG) studies demonstrate that the *subscapularis* muscle remains active in patients who have maintained good shoulder function in the face of full-thickness RC tears. Therefore, therapy which strengthens this important humeral head depressor seems prudent.[39] The objective of strengthening a *supraspinatus*-deficient shoulder is to maintain a balanced force-couple between the *subscapularis* and *teres minor* and *infraspinatus*.

It is important to keep in mind prior to strengthening the RC, ROM should be restored and pain minimized. Strengthening in the face of pain and inflammation can be counterproductive. Once ROM, pain control, and residual periscapular musculature strength is restored, a gradual improvement in overall shoulder function can be antici-

pated. If, however, the patient fails nonoperative therapy, a shoulder with good preoperative motion is much more likely to have a positive result with surgical repair than a stiff shoulder.

Perioperative and postoperative planning

Once a patient elects to undergo surgical repair of the RC, the patient, the orthopaedic surgeon, and the physical therapist should all communicate on patient-specific factors that can be anticipated during the perioperative and postoperative period. Comorbidi-ties, prior shoulder surgery, and tobacco abuse should be part of this discussion, specifically, how these factors might impact the patient's healing and rehabilitation. An immediate smoking cessation program should be strongly encouraged (if not enforced) as tobacco abuse can adversely affect tendon healing. The patient, the surgeon, and the therapist should also be aware of the status of the RC musculature going into surgery as this has a direct impact on surgical success. If fatty atrophy of the RC is present, surgical results are less optimal with some literature reporting a retear incidence as high as 85%.[40]

Good communication among the orthopaedist, the patient, and the anesthesiologist is also important to determine the best anesthesia and pain control plan for the patient. For RC repair, an interscalene block has many advantages over general anesthesia. Pain during the first 24 to 36 h can be better controlled. Furthermore, an interscalene block has the advantage of muscle paralysis, which can prevent inadvertent muscle activation that could potentially jeopardize a newly repaired RC.

Postoperative rehabilitation

Timelines for RC rehabilitation should respect the delicate balance between the appropriate duration of tendon healing and the risk for the development of a stiff shoulder. Recent evidence from a prospective randomized control study demonstrated better results from RC patients who were rehabilitated more gradually on a decelerated program than those who had a standard rehabilitation to full activities. This information should be kept in mind when rehabilitating the arthroscopically repaired patients, whose postoperative pain may be minimal and thus feel that they can begin immediate strengthening on a cuff tendon that biologically is not ready for aggressive early motion.[41]

Prior to establishing timelines for therapy, the RC lesion should be categorized as a large/massive tear *versus* a small tear. RC atrophy should also be taken into account during the rehabilitation period. Patients with large tears or with fatty atrophy should have a substantially decelerated rehabilitation program compared to those patients with smaller tears. Poor quality of tissue repaired and osteoporotic bone should also prompt a more guarded rehabilitation program. The age of the patient is also important variable in regards to outcome. Patients over 70 years old who undergo RC repair old should be on more gradual rehabilitation program. The treating orthopaedic surgeon should communicate with the therapist about which of the four RC tendons were repaired as the involved tendons obviously affect therapy protocols. Other concomitant procedures such as *biceps tenodesis* or tendon transfers should also be communicated.

Rehabilitation for RC repairs is typically divided into four phases. Phase 1 (0-6 weeks) consists of passive ROM of the affected shoulder with the goal of motion and minimal stress to the repaired tendon. Phase 2 (6-12 weeks) encourages active ROM with gradually increasing loads to the repaired tissue. In Phase 3 (10-16 weeks), strengthening of the RC musculature is initiated. Phase 4 (16-22 weeks) combines strengthening of the RC musculature with muscle conditioning and transitions the patient into full activity. With each of these phases, the patient experiences a gradual progression of loading to the RC tendons. This idea of progressive loading is supported by EMG studies, which

demonstrate the lowest EMG activity in phase 1 and the highest EMG activity in the latter phases.[42]

Phase 1: passive range of motion (0-6 weeks)

This is the phase where the balance between tendon healing and the prevention of postoperative adhesions is the most delicate. At this stage, the RC can tolerate very little load before failure. Initially (0-2 weeks), the cuff repair integrity is relying on a weak fibrin lattice and suture from the repair. At 2-3 weeks, type III collagen begins to replace the fibrin clot, but the fibers of this collagen are not stress oriented. This haphazard collagen framework is unable to bear any substantial force.

The clinical data seem to correlate with the basic science as well. In a randomized, controlled trial, patients who employed a more decelerated rehabilitation program (where the shoulder experiences very little stress) tended to have better postoperative results than those who underwent a standard therapy program.[41] Early aggressive motion in the latter group may have lead to the disruption of this weak biologic construct of fibrin and type III collagen, and the failure of their RC lead to poor outcomes.[41]

Despite the above data, we would not recommend complete shoulder immobilization because the benefits of early passive motion for RC repair have been demonstrated in numerous clinical studies. A patient should be immobilized in an abduction brace at 30 to 45°, but the patient should be allowed to come out the device for passive ROM exercises. Especially for larger tears, we recommend pendulums and mid-range supine ER and IR in the plane of the scapula with a stick for the first 4 weeks, subsequently followed by low-load passive ROM during the next 2 weeks. Dockery demonstrated in healthy shoulders with surface EMG that the use of pulleys or self-assisted flexion elicited greater percentages of maximal voluntary effort (17.6% and 8.7%) *versus* physical therapy (PT) assisted passive ROM and pendulums (5% each) and ER/IR with a stick (less than 5%).[35, 43] During weeks 0-6, excessive passive ROM adduction and IR should be avoided. For smaller tears, low-load passive ROM can begin earlier (week 2). Regardless of tear size, no active glenohumeral motion should be performed during phase 1.

The abduction immobilizer should be worn any time during the first 6 weeks when the patient is not performing ROM exercises. The advantages of the abduction component of the immobilizer are that it potentially increases blood flow to the tendon and decreases the tendon excursion length compared to a shoulder in a traditional sling. At rest, the patient should use cryotherapy in conjunction with abduction immobilizer.

At 2-6 weeks, we recommend the use of aquatherapy. This allows for stretching and passive ROM of the involved shoulder. Two to three sessions per week for 15 min per visit are appropriate. At 6-8 weeks, the patient can progress to active ROM of motion in the pool. At 10-12 weeks, underwater strengthening exercises can be performed. The advantage of aquatherapy is that it allows for active-assisted motion in a gravity-reduced setting. In the pool, the tension on the RC is lessened, and it has been noted that active shoulder elevation in the abilitation has dramatically lower musde activation than in typical rehabilitation situation.[38] This allows for earlier active motion by keeping loads reduced on the repaired RC tissue.[38]

Scapulthoracic joint mobility and strength should be included in the early rehab of the shoulder. In a study by Smith *et al.* surface and fine wire EMG showed scapulothoracic strengthening exercises can be performed safely with minimal activity occurring in the *supraspinatus*, *infraspinatus*, and *teres minor* while the glenohumeral joint was immobilized.[44] Scapulothoracic exercises included scapular retraction, depression, protraction, and scapular clocks performed in sitting. Percentage of max voluntary contraction was noted greatest in the *trapezii*, *serratus anterior*, and *subscapularis*. Subsequently, the *subscapularis* rehabilitation program should avoid early scapular strengthening.[44] During late phase 1 (week 5) progression to early

active assisted ROM and sling weaning may be appropriate.

Phase 2: active range of motion (6-12 weeks)

The tendon healing model suggests maturation and remodeling phase are still occurring during this time.[24, 25] At this point, low level loading will allow for appropriate tensioning forces that will strengthen the repaired tendon.[24, 25] Progression of active assisted range-of-motion (AAROM) to full active ROM in gravity assisted to gravity resisted exercise should be done with normal scapulohumeral and scapulothoracic kinematics. With normal scapulohumeral rhythm and scapulothoracic kinematics, the glenohumeral muscles are placed in a position to generate tension more efficiently as well as to avoid a position of impingement of the RC between the acromial arch and humerus.[45, 46] While pulleys may now be safely initiated at this time in regards to tendon loading, their use should be closely supervised to assure proper kinematics so that compressive forces are not applied to the repaired tendon. RC isometrics should be progressed to submaximal (50% effort) to protect the healing tendon. Scapulothoracic joint strengthening should include prone rows, prone extension with ER, and prone horizontal abduction with ER. The latter should be initiated first with the elbow bent to 90° to decrease the lever, then progressed as tolerated.[47] Early proprioceptive training can now be performed.[47] An example Ellenbecker uses is the supine balance position (90-100° of flexion with scapular protraction) with rhythmic stabilization techniques. This is accomplished as the patient holds the position while the therapists applies appropriately graded alternating external/internal, and flexion/extension forces.[47] Because the triceps and biceps cross the shoulder joint, initial AAROM against gravity should now begin. Scar mobility should be addressed to avoid adherence to deeper tissue levels, which may prevent full ROM or cause pain. End range stretching at progressively higher intensities and joint mobilization techniques (grades III and IV) may be initiated to address any muscular or capsular limitations. Again, care should be taken if larger repair is involved. The patient should be weaned from the sling at this point, and pain should be well controlled.

Phase 3: initial strengthening phase (10-16 weeks)

When sufficient glenohumeral and scapulothoracic kinematics have been demonstrated in phase 2, strengthening may be initiated at 10 weeks postoperatively. Tendon healing at this point is strong enough to allow for resistive exercises. Attempting to strengthen a shoulder that does not demonstrate sufficient ROM, however, may cause pain, subacromial impingement, and stress on the repair.

Initial strengthening should be started below shoulder level. Insufficient strength of the RC muscles decreases their ability to function as depressors of the humeral head. This may lead to increased subacromial contact and impingement of the repair.

Initial exercises in this phase of strengthening include ER (*infraspinatus*, *teres minor*), IR (*subscapularis*), forward flexion (anterior deltoid, *supraspinatus*) and rows (posterior deltoid and periscapular muscles) with elastic cord resistance. Resistance should allow for high repetitions to RC build muscle endurance and to decrease the risk of excessive stress on the repair early in the strengthening phase. With sufficient ROM and appropriate cuff strength, advanced exercises may be added as tolerated.

The efficacy for the most appropriate exercises to strengthen the RC muscles has been argued in various studies.[48] Variability usually lies within the method of determining muscle activation.[48] EMG fine wire analysis provides immediate data on muscle activation, but it has been shown to have poor reproducibility and a small detection area where electrode migration is possible during exercise. T2 magnetic resonance imaging (MRI) measures muscle water content

during the relaxation time after muscle activation. These studies have been shown to be reproducible and reliable at detecting muscle activation.

Takeda *et al.* determined that the full can and empty can exercises were most effective at strengthening the *supraspinatus* muscle.[49] T2 relaxation time imaging was used to detect muscle activation after subjects performed three different exercises. When compared to horizontal abduction of the shoulder in the prone position, the full can and empty can exercise shows higher T2 relaxation times.[49] In a similar study, Horrigan determined that resisted side lying abduction was the most effective at strengthening the *supraspinatus* muscle.[50] MRI studies have shown that the combination of IR with forward elevation in the scapular place significantly reduces the subscromial space thus leaving the shoulder susceptible to impingement.[51] Because of these findings, we do no advocate this motion when rehabilitating the RC.

Exercises that increase pain, stiffness and/or swelling should be altered or avoided temporarily. Restoring normal scapulothoracic kinesis should be primary goal in restoring normal shoulder function as well as trunk and core strength.

Phase 3 should thus include RC exercises that do not put the repair at risk of impingement or excessive loads that may be damaging. Continued PROM, AAROM, stretching and manual therapy is indicated with the presence of ROM and joint limitations. Proprioceptive and neuromuscular exercises may be advanced as tolerated and should be included in the initial strengthening phase.

Phase 4: advanced strengthening (16-22 weeks)

When sufficient RC strength is demonstrated after phase 3, patients may be advanced to phase 4. Phase 4 is a continuation of strengthening that includes strengthening of the larger prime mover muscles of the shoulder, such as the *pectoralis major, latissimus dorsi* and deltoid muscles. Overhead activity is advanced as tolerated in the absence of any impinging symptoms. Exercises may be advanced to more sport specific type exercises, such as golf and tennis swings, or exercises that promote work or activities of daily living (ADL) specific activity. Return to sports is allowed when patients exhibit optimal ROM, RC strength, proprioceptive and neuromuscular function, scapulothoracic kinesis and remain absent of pain, swelling and symptoms of impingement.

Conclusions

Rehabilitation of the RC presents a unique problem to the therapist, orthopaedic surgeon, and patient. The timeline of tendon healing must be respected; however, motion must also be preserved, keeping in mind, most patients would prefer a weaker shoulder to a stiff one.[49] To achieve optimal results with therapy, communication among the therapist, patient, and orthopaedic surgeon is essential – before and after surgery. The objective of therapy should be to restore passive and active ROM, minimize pain, and maximize strength and function.

Treatment protocols and timelines must be adjusted to account for the size of the tear, tissue and bone quality, age of patient, degree of tissue atrophy, and tendons involved. All protocols should initially provide for shoulder motion with minimal stress to the tendon. The rehabilitation should progress in four phases: phase 1 (0-6 weeks) passive ROM only; phase 2 (6-12 weeks) active ROM; Phase 3 (12-16 weeks) strengthening; phase 4 (16-22 weeks) strengthening, conditioning, and transition to full activity. When good surgical technique of the RC is combined with appropriate rehabilitation, excellent results can be anticipated.

Riassunto

Riabilitazione delle rotture della cuffia dei rotatori

La patologia della cuffia dei rotatori è un problema estremamente comune nei pazienti adulti. Poiché il recupero postoperatorio del paziente affetto da rot-

tura della cuffia è impegnativo, il problema è affrontato meglio con una gestione combinata tra paziente, fisioterapista, e ortopedico curante. I protocolli di riabilitazione dovrebbero rispettare l'equilibrio tra la rirottura dovuta a una terapia troppo aggressiva in fase iniziale, e lo sviluppo di aderenze e rigidità da immobilizzazione prolungata. La riabilitazione dovrebbe essere condotta a un ritmo che prevenga la rigidità di spalla, ma rispetti i tempi della biologia della guarigione dei tendini. L'obiettivo di questo articolo è fornire un approccio basato sull'evidenza, sulla scorta della scienza di base e della letteratura clinica, allo sviluppo di linee guida della riabilitazione dopo la riparazione chirurgica delle rotture di cuffia.

Parole chiave: Cuffia dei rotatori - Spalla - Riabilitazione.

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