**Rotator Cuff and Subacromial Impingement Syndrome:**
**Anatomy, Etiology, Screening, and Treatment**

The glenohumeral joint is the most mobile joint in the human body, but this same characteristic also makes it the least stable joint.[1-3] The rotator cuff is a group of muscles that are important in supporting the glenohumeral joint, essential in almost every type of shoulder movement.[4] These muscles maintain dynamic joint stability which not only avoids mechanical obstruction but also increases the functional range of motion at the joint.[1,2] However, dysfunction of these stabilizers often leads to a complex pattern of degeneration, rotator cuff tear arthropathy that often involves subacromial impingement.[2,22]

Rotator cuff tear arthropathy is strikingly prevalent and is the most common cause of shoulder pain and dysfunction.[3,4] It appears to be age-dependent, affecting 9.7% of patients aged 20 years and younger and increasing to 62% of patients of 80 years and older ( $P < .001$); odds ratio, 15; 95% CI, 9.6-24; $P < .001$.[4] Etiology for rotator cuff pathology varies but rotator cuff tears and tendinopathy are most common in athletes and the elderly.[12] It can be the result of a traumatic event or activity-based deterioration such as from excessive use of arms overhead, but some argue that deterioration of these stabilizers is part of the natural aging process given the trend of increased deterioration even in individuals who do not regularly perform overhead activities.[2,4] The factors affecting the rotator cuff and subsequent treatment are wide-ranging. The major objectives of this exposition are to describe rotator cuff anatomy, biomechanics, and subacromial impingement; expound upon diagnosis and assessment; and discuss surgical and conservative interventions.

## Anatomy and Biomechanics

The supraspinatus, infraspinatus, teres minor, and subscapularis are intrinsic myotendinous structures of the shoulder that collectively form the rotator cuff. Traditional medical education describes these tissues as having discrete insertional footprints about the humeral tubercles as shown in Figure 1.[12] The supraspinatus takes on a trapezoidal or triangular shape; it originates at the supraspinous fossa of the scapula and inserts at the superior impression of the greater tubercle of the humerus.[10] The supraspinatus's primary muscle action is the initiation of abduction.[11] In the absence of a functional supraspinatus the deltoid is capable of initiating abduction, but it must generate nearly twice as much torque.[11,18] The origin of infraspinatus is the infraspinous fossa, and this muscle inserts just below supraspinatus on the middle portion of the greater tubercle, creating a trapezoid-shaped muscle.[10] Together infraspinatus and teres minor externally rotate the humerus. Teres minor tapers into a triangular shape, originating at the middle half of the lateral border of the scapula and inserts onto the lowest impression of the greater tubercle.[10] Finally, subscapularis has a large origin at the subscapular fossa and inserts onto the lesser tubercle of the humerus forming an auricular shape.[10] This muscle internally rotates and adducts the humerus. Collectively the rotator cuff muscles hold the head of the humerus in the glenoid cavity during arm elevation.[6,10-12]

While it is convenient to think of each of the rotator cuff muscles as each having well-defined insertions, in reality there is no clear insertional footprint because the whole area serves as an insertion zone for all four muscles as well as the joint capsule[10]. The tendons interdigitate and blend together, forming a common, continuous insertion onto the humerus.[5] The rotator cuff further coalesces with adjacent soft tissue structures, giving rise to the rotator cuff-capsule complex which is composed of 5 semi-distinct layers in which collagen fibers from the rotator

cuff tendons, nearby ligaments, and surrounding joint capsule intertwine.[6] As the tendons progress from the myotendinous junction to the humeral insertion they become less round and instead become more ribbon-like and increasingly interdigitated.[6]

The glenohumeral joint exhibits such a wide ROM in large part due to the disproportionate (4:1) area of the humeral head to glenoid cavity.[7] The shoulder is also unique in that most joints rely on capsular and ligamentous restriction to approximate the articular surfaces; whereas, the glenohumeral joint relies on both active (rotator cuff) and passive structures. When the arm is at rest by one's side the superior joint capsule and coracohumeral ligament are taut, but once the arm is elevated, passive tension is lost and the rotator cuff must be activated.[11] Dynamic compression or cavity compression is the primary process by which the rotator cuff stabilizes the head of the humerus against the relatively small glenoid cavity.[6,8] In a healthy shoulder, balanced moments between subscapularis and the combined forces of infraspinatus and teres minor compress the humerus into the glenoid as illustrated in Figure 2.[6,11] Normal biomechanics of arm elevation occur by a muscular force-couple mechanism: active inward and downward rotator cuff activation approximates the humeral head as the deltoid muscle force is directed upward and outward with respect to the humerus.[11]

**Fiber Type**

Force generation of skeletal muscle relies on numerous variables such as length of muscle, cross sectional area, and moment arm.[8,21] While muscle architecture may be the most important determinant in force generation, muscle fiber type composition affects speed of contraction, power fatigability, and metabolism, and is associated with muscle stiffness, rate of atrophy, and even susceptibility to injury.[21] Moreover, knowledge of fiber type is also clinically applicable for exercise prescription parameters for the rotator cuff since muscle weakness and/or fatigue has

been identified as a secondary cause of subacromial impingement.[24] For example, optimal exercise prescription parameters of resistance exercise varies depending upon fiber type predominance, and parameters of exercise can modulate fiber type distribution. Skeletal muscles and individual motor units can be classified as type I (slow twitch oxidative), type IIa (fast twitch oxidative glycolytic), and type IIx (fast twitch glycolytic).[21] Type I fibers are fatigue resistant, have slower maximum contraction velocities, and are capable of generating less force than Type II fibers.

In a cadaveric study that applied immunohistochemistry and SDS-PAGE to 6 specimens (3 male, 3 female; mean ± SD age, 65± 12 years) the fiber type percentages of the rotator cuff muscles was ascertained.[21] The investigators found that average slow myosin content (fiber type I) was 54% ±6% in supraspinatus, 41% ±8% in infraspinatus, 49% ±8% in teres minor, and 38% ±8% in subscapularis. All the muscles showed mixed fiber types, but supraspinatus had significantly greater ($p<0.05$) percentage of type I fibers compared to infraspinatus and subscapularis.

**Supraspinatus**

Because the majority of rotator cuff tears occur at or include the supraspinatus contribution to the tendinous cuff, much of the further discussion will focus on the structural and mechanical properties of this anatomic region. Human supraspinatus can be divided into anterior, central, and posterior thirds and significantly greater ultimate load, ultimate stress, and tensile modulus is found in the anterior region.[6] Studies have shown mixed results as to whether the bursal or articular aspect of supraspinatus is exposed to greater tensile stress; some indicating no difference while others have found greater tensile loading on the bursal aspect.[6,15] Abduction angle influences the biomechanical load transmission placed upon the tendon but the matter is

complex, and there are inconsistencies throughout the literature. Huang et al measured tensile loading in eight cadaveric specimens and reported that the articular aspect of supraspinatus demonstrated greater strain at 22° (7.4+/-2.6% vs. 1.3+/-0.7%, p=0.0002) and 63° (6.4+/-1.6% vs. 2.7+/-1.2%, p=0.0001) while the bursal surface exhibited greater strain at 90° (7.6+/-2.8% vs. 4.9+/-0.4%, p=0.013), finding that in all cases that strain was highest at the insertional zone rather than at mid-substance or the myotendinous junction.[16] The above reports suggest a propensity of tearing near the insertion zone at the articular surface.

**Vascular supply**

Blood to the rotator cuff is supplied by up to six arteries. Three of them (suprascapular and both anterior and posterior humeral circumflex arteries) are common to most people, but the other three (thoracoacromial, suprahumeral, and subscapular arteries) are sometimes absent.[11] Supraspinatus receives its blood supply from the thoracoacromial artery which anastomoses with the anterior and posterior circumflex arteries; however, since that artery is sometimes absent, supraspinatus, and to a lesser extent infraspinatus, are often hypovascular compared to the rest of the tendinous cuff.[11] In their seminal work, Rothman and Parke found that, regardless of age, 63% and 37% of all subjects (n=72) demonstrated hypovascularity of supraspinatus and infraspinatus, respectively; whereas, only 7% exhibited hypovascular subscapularis.[13] Supraspinatus may be particularly susceptible to injury since tendon injury is often reported at sites of poor blood supply.[14] This avascular area is so significantly associated with rotator cuff tearing that it is dubbed the “critical zone.”

**Acromion**

Generally speaking, there are 3 variations of acromion shape and are classified as flat (type I), concave (type II), or hooked (type III)[20]; some sources refer to a fourth type (upturned inferior surface) but discussion will be limited to the first three.[19] In an examination of 91 MRI images of patients with complete rotator cuff tears, Hirano found that among the acromia observed 33 (36.3%) were type I, 22 (24.2%) were type II, and 36 (39.6%) were type III.[20] Examples of these acromion types are available in Figure 3. Furthermore, the size of the tears were significantly larger ($p<0.05$) in shoulders with hooked (type III) acromia compared to tears in type I and type II as sown in Table 1. This evidence suggests that acromion shape influences incidence and severity of rotator cuff tears, type III being the most common offender.

There is considerable debate as to whether congenital acromion type causes rotator cuff tearing or if acromial morphology is acquired as a result of rotator cuff tears and/or aging.[20] It certainly stands to reason that congenital acromion shape would influence subacromial impingement. On the other hand, active bone growth at the acromion is associated with greater incidence of rotator cuff tearing. In 81% of cases involving symptomatic acromioclavicular joint osteoarthritis, concurrent rotator cuff tears are observed.[19] Chambler reported acromial enthesophyte growth at the coracoacromial ligament's insertion (inferior acromion) in 15 consecutive patients undergoing modified open acromioplasty and rotator cuff repair (mean age = 62.2 ± 1.75 years).[21] It was reasoned that the stimulus for oseteoblastogensis was twofold: (1) constant static tensile loading from the coracoacromial ligament; and (2) periods of increased dynamic tensile loading when the ligament is forced upwards during impinging movements. Hence, in shoulders with an existing rotator cuff tear, impinging motions provoke further inferior acromial bone deposition which escalates a viscous cycle of impingement, giving further credence to the notion that impingement effects acromial morphology.

## Structural Malalignment

A common causes of abnormal glenohumeral mechanics includes excessive thoracic kyphosis.[6,11,22] With excessive kyphosis the scapula follows the contour of the thorax and downwardly rotates, causing the glenoid to no longer face upward which moves the freely hanging arm into relative abduction, putting the superior joint capsule on slack.[11] In order to prevent inferior subluxation of the humerus the rotator cuff muscles must contract; thereby, chronically increasing tone of the rotator cuff muscles. This chronic tonicity is one possible mechanism for adhesive capsulitis because the increased tone approximates the muscles and stimulates collagen synthesis, advancing capsular fibrosis.[11] The discussion of bony constituent alignment and subsequent adhesive capsulitis is relevant because it is thought that the condition brings about secondary shoulder impingement.[22]

## Subacromial Impingement Syndrome and Tendinopathy

Shoulder impingement is a common shoulder pathology and involves the compression, entrapment, or mechanical irritation of rotator cuff structures, subacromial bursa, and/or the long head of the biceps tendon against the coracoacromial arch in the subacromial space.[11,22] Hence, subacromial impingement syndrome is an umbrella term and may encompass a spectrum of disorders: partial thickness rotator cuff tears, rotator cuff tendinosis, calcified tendinitis and subacromial bursitis.[28] There are various theories as to why shoulder impingement occurs. Magee identifies a variety of primary causes: inflammation of the subacromial space, rotator cuff tendon degeneration, osteophytes under the acromioclavicular joint, type III hooked acromion, and glenohumeral instability.[24] Secondary causes may include: abnormal glenohumeral and/or scapulothoracic arthrokinematics, thoracic kyphosis, muscle weakness/fatigue, muscular hypomobility, posterior capsule tightness, and adhesive capsulitis.[24]

A thorough discussion of each of the aforementioned primary and secondary causes is beyond the scope of this paper, but a popular explanation for subacromial impingement syndrome the encapsulates a handful of those etiologies is the mechanical-anatomic theory described by Neer.[11,22,23] The syndrome advances in three stages: (1) a benign, self-limiting syndrome that involves acute bursitis with subacromial edema and hemorrhage; (2) repeated compression of the bursa causes it to lose its lubrication ability and the underlying rotator cuff is exposed to friction, fraying the underlying tendons, eliciting fibrosis; and (3) progression of partial to full-thickness tear and development of subacromial bony spurs.[11,24] As previously described, acromion morphology may influence the extrinsic compression explained by the mechanical-anatomic theory.

Some authors do not accept that extrinsic compression is sufficient enough reason for tendon deterioration, but rather, intrinsic etiologies such as hypovascularity, aging, and tensile forces lead to rotator cuff failure.[24] Tendinitis at the shoulder is common for both young and older persons, but in the case of the latter population the tendinitis is more likely to progress into a degenerative lesion.[4,11] With aging into the seventh decade of life collagen content of tendon decreases and ultimate strength at failure decreases, making the tissue less resilient to excessive tensile loading.[14] Moreover, as previously described the supraspinatus tendon is avascular in many specimens, but in addition aging is associated with diminution of vascularity to tendon, often with no intratendinous vascularity by age 70.[25] Without adequate vascular supply adequate repair does not occur and the metabolic demands of the tissue cannot be met.[11,25] Focal cell death triggers an inflammatory response that, without an adequate blood supply for healing, may become chronic or repetitive, producing immature scar tissue.[11,14] Scar tissue does not have the same biomechanical properties as the surrounding tendon and so this creates (1) non-functional

or suboptimal tissue and (2) creates areas of stress concentrations that beg future mechanical failure.

Degenerative changes (or acute trauma) weaken the supraspinatus until it is no longer able to center the humeral head on the glenoid.[24] Numerous investigations have noted that superior translation of the humeral head is a typical biomechanical consequence of rotator cuff injury.[6] This change of position exacerbates the condition by putting the joint at heightened risk for impingement, creating a feed forward mechanism for further degeneration.[23]

There is a tendency for some persons who experience pain from rotator cuff tendinopathy to exhibit fear avoidance behavior, avoiding tasks that require the use of the affected shoulder.[26] However, such behavior results in disuse atrophy of the rotator cuff structures which may actually increase chronicity and recurrence because, conceivably, at some point arm elevation will occur.[11] In this scenario the rotator cuff has reduced strength at failure and is at greater risk of extrinsic compression since muscle weakness promotes superior translation of the humeral head.

**Screening and Special Tests**

To effectively treat subacromial syndrome it must be correctly identified. Of course, clinical assessment of persons suspected of subacromial impingement should begin as any other assessment would, with an open mind and broad view that is funneled by objective findings and subjective interview.[27] Differential diagnosis is broad and may include acromioclavicular joint injury, bicipital tendonitis, brachial plexus injury, cervical disc injuries, cervical discogenic pain syndrome, cervical radiculopathy, cervical spine sprain/strain injuries, claviclular fractures, contusions, myofascial pain, osteoarthritis, shoulder dislocation, SLAP lesions, suprascapular

neuropathy, thoracic disc injuries, thoracic discogenic pain syndrome, and thoracic outlet syndrome.[11,23,24,28] Initial findings that may suggest subacromial impingement and rotator cuff tendinopathy include age of 40+ years but especially 65+ years (age associated degeneration), history of frequent overhead activities (swimming, baseball pitcher, etc), humeral external rotation weakness, painful abduction, and shoulder night pain and resting pain.[24,28]

There are numerous special tests that have been developed for ruling in or ruling out subacromial impingement. Michener et al examined the reliability and diagnostic accuracy of five such tests: Hawkins-Kennedy, Neer, painful arc, empty can, and external rotation resistance.[29] Conveniently, 2 of 5 of the above tests should already be part of the initial musculoskeletal examination (painful arc and resisted external rotation), and thus these special tests offer an efficient means of diagnosis. The tests have been found to have fair to substantial strength of interrater reliability as described in Table 2. The investigators found that positive findings on at least 3 of the 5 tests has good diagnostic accuracy: sensitivity = 95% CI, 0.75 (0.54–0.96); specificity = 95% CI 0.74 (0.61–0.88), +LR = 95% CI 2.93 (1.60–5.36); and -LR = 95% CI 0.34 (0.14–0.80).[29] Individual sensitivity, specificity, and likelihood ratios for each of the five tests is listed in Table 3.

Brief summaries of the tests are based upon descriptions by Magee and are as follows:[24]

- For the Hawkins-Kennedy test (Figure 4) the patient stands as the clinician flexes the arm to 90° and brings the arm to 10-20° of horizontal abduction. The arm is then passively internally rotated. A positive finding is pain provocation at the shoulder. Anecdotally, if there is no positive finding in the positioning described, then adaptations of this test include testing at 0° and then 20° of horizontal adduction, and

it is thought that pain provocation in more abducted positioning relates to greater severity of supraspinatus injury.

- For the Neer test (Figure 5) the clinician passively and forcibly elevates the patient's upper extremity in the scapular plane with the arm in medial rotation. In this position the greater tubercle of the humerus is pressed against the anteroinferior acromion. A positive test results in shoulder pain, indicating either supraspinatus or long head biceps tendon irritation.
- For painful arc test (Figure 6) the patient actively abducts his/her arm, and increased pain between 60-120° indicates a positive finding.
- The empty can test (Figure 7) entails having the patient actively abducting his/her arm to 90° in the scapular plane and then maximally internally rotating the arm (like he/she is pouring out the contents from a 12 oz can). The therapist then asks the patient to maintain the position while inferiorly directed manual force is applied. Pain or weakness indicates a positive finding.
- Finally, for the external rotation resistance test (Figure 8) while standing the patient flexes his/her neutrally aligned forearm to 90°. Next, the clinician asks the patient to maintain the position as force is applied to the patient's distal forearm, prompting the patient to apply a glenohumeral external rotation moment. Pain or weakness indicates a positive finding.

**Treatment**

Conservative treatments for subacromial impingement often includes physical therapy, NSAIDs, and corticosteroid injections.[23] Harrison and Flatow reported that only 2 of 8 studies reviewed improved pain and shoulder ROM in a clinically meaningful way and also that

NSAIDs and corticosteroids produced equivalent outcomes.[23] Hart determined that corticosteroid injections were beneficial (but similar to placebo) at producing short term pain relief of tendinopathy but that long-term deleterious consequences were likely.[31] Adverse side effects were observed in as many as 82% of corticosteroid injections. It has been noted that corticosteroid injection has a catabolic effect on tendon, increases chance of future tendon rupture, and does not actually address the mechanism of injury.[14] In order to most effectively address pathology one must consider the mechanism of injury. In this case, as previously discussed, common primary and secondary impingement is the result of poor biomechanics, muscular weakness, and degenerative changes.

Physical therapy options vary and may include therapeutic exercise, extracorporeal shock wave therapy, hot/cold therapy, taping, ultrasound, transverse friction massage, and manual therapy.[32,33] Retraining proper arthrokinematics and strengthening are hallmarks of treating subacromial impingement syndrome mechanisms; whereas, many of the aforementioned modalities are suited to controlling pain and advancing tendon healing.[23,32,33] Harrison and Flatow recommend a course of physical therapy for at least 6 weeks.[23] If at that time symptoms have not improved or they have progressed, the therapist should refer for consultation with an orthopedist. A comprehensive physical therapy strategy has been proposed by Stevenson et al.[33]

- Delivered between six to eight exercise sessions over 12 weeks
- Discharge patients who fail to attend three consecutive appointments
- Provide patients with exercise diaries to facilitate adherence
- Could also use soft tissue massage/posture correction/heat or cold
- Could use facilitation techniques such as taping or anterior soft tissue release techniques
- Could also prescribe neck exercises if judged as needed for individual patients

The authors expound upon the exercise protocols which involve 3 stages:

- Stage I: scapular stabilization exercises and active movement with no external loading, addressing scapulothoracic arthrokinematics. Scapular stabilization retraining progress from prone to seated to standing and initially involves short lever arms (flexed elbow) and later a longer lever arm (extended elbow) for additional resistance.
- Stage II: ROM exercises, isometric strengthening, and emphasis on developing pain free motion. Herein, cardinal planes of motion are directed (such as flexion, abduction, and external/internal rotation) while maintaining scapular stability and normalized scapulothoracic arthrokinematics. Progressions include resistance bands or manually directed resistance (or self-directed resistance).
- Stage III: progressive strength training with emphasis on proper mechanics.

8-10 total exercises are recommended and initial set and rep ranges will vary depending upon patient tolerance, but 3 sets of 10 per exercise is the prescribed target.[33] Lovering has suggested that since rotator cuff muscular fatigue/weakness is often implicated in pathological glenohumeral arthrokinematics, higher rep ranges may be preferred.[21] Training these muscles for endurance through resistance exercise will facilitate transition of type II muscle fiber into type I, enhancing resilience to fatigue.[21] Much of the protocol espoused by Stevenson et al agrees with intervention selection supported elsewhere in the literature:[22]

- Scapulothoracic stabilization interventions
- Serratus anterior strengthening or retraining
- Upper trapezius activation reduction
- Posterior shoulder stretching

- Pectoralis minor stretching
- Thoracic extension posture and exercise

It has been reported that transverse friction massage should be a staple in rotator cuff tendinopathy treatment.[11] While the exact mechanism is not understood, transverse friction massage mobilizes scar tissue, promoting healing and fibrotic tissue remodeling.[11] Kim et al have found that eccentric loading of supraspinatus in abduction produces similar strength gains as concentric loading, but that the additional tensile strain imposed by greater eccentric loads may be more beneficial for maintaining fiber bundle length, remodeling, and healing.[34]

**Surgical Intervention and Considerations for Physical Therapy**

For patients not responding to conservative treatment or for considerable partial tear (>50% of supraspinatus tendon) and full thickness rotator cuff tears, surgical intervention is considered.[30,35] Prior to surgery MRI imaging and/or arthroscopy are common procedures used to inform the orthopedist and patient.[23] The exact nature of the repair depends on the precise location of the rupture, but generally speaking the goal of surgical management is to reestablish a strong tendon-bone integration and restore normal biomechanics to the joint.[35]

Skilled physical therapy is necessary after surgery, especially considering failure rates as high as 39.8% of patients recovering from surgical intervention for full thickness tears.[36] Post-surgical protocols vary and may be limited by the operating physician's orders, but a common restriction is limiting shoulder flexion to <90°.[38] There is no consensus on best practice following surgery, and the evidence is mixed. For example, Chang et al reported that early ROM of motion activity accelerated recovery and reduced post-operative stiffness;[39] whereas, Iannotti found that delayed

physical therapy protocols that limited passive range of motion were similar to a protocols with early passive range of motion after rotator cuff repair.[37]

Lee et al compared the outcomes between aggressive early passive rehabilitation and limited early passive rehabilitation.[38] The protocols are listed in Table 4. In the aggressive therapy group, treatment initiated same day as surgery, entailing twice daily passive ROM into tolerable shoulder flexion and 30° of external rotation. In addition, patients were instructed to self-administer passive range of motion to tolerance 3 times per day. Otherwise, an abduction brace was worn for 6 weeks and no return to recreational activity that involved the shoulder was permitted for 6 months. In the limited therapy group patients only participated in shoulder flexion up to 90° by continuous passive motion machine twice per day. At three weeks the limited group ramped intervention protocols to match the aggressive group. At 6 and 12 months follow up there was no statistical difference between the two groups for ROM or strength.

**Conclusion**

The rotator cuff is a complex structure that is vitally important to the proper functioning of the shoulder. A myriad of factors such as intrinsic glenohumeral instability, age, vascularity, bony alignment and morphology, muscular weakness, and repetitive overhead activities predispose the rotator cuff to subacromial impingement syndrome and associated tendinopathy. There are wide ranging treatments used to treat these pathologies.

**Appendix**

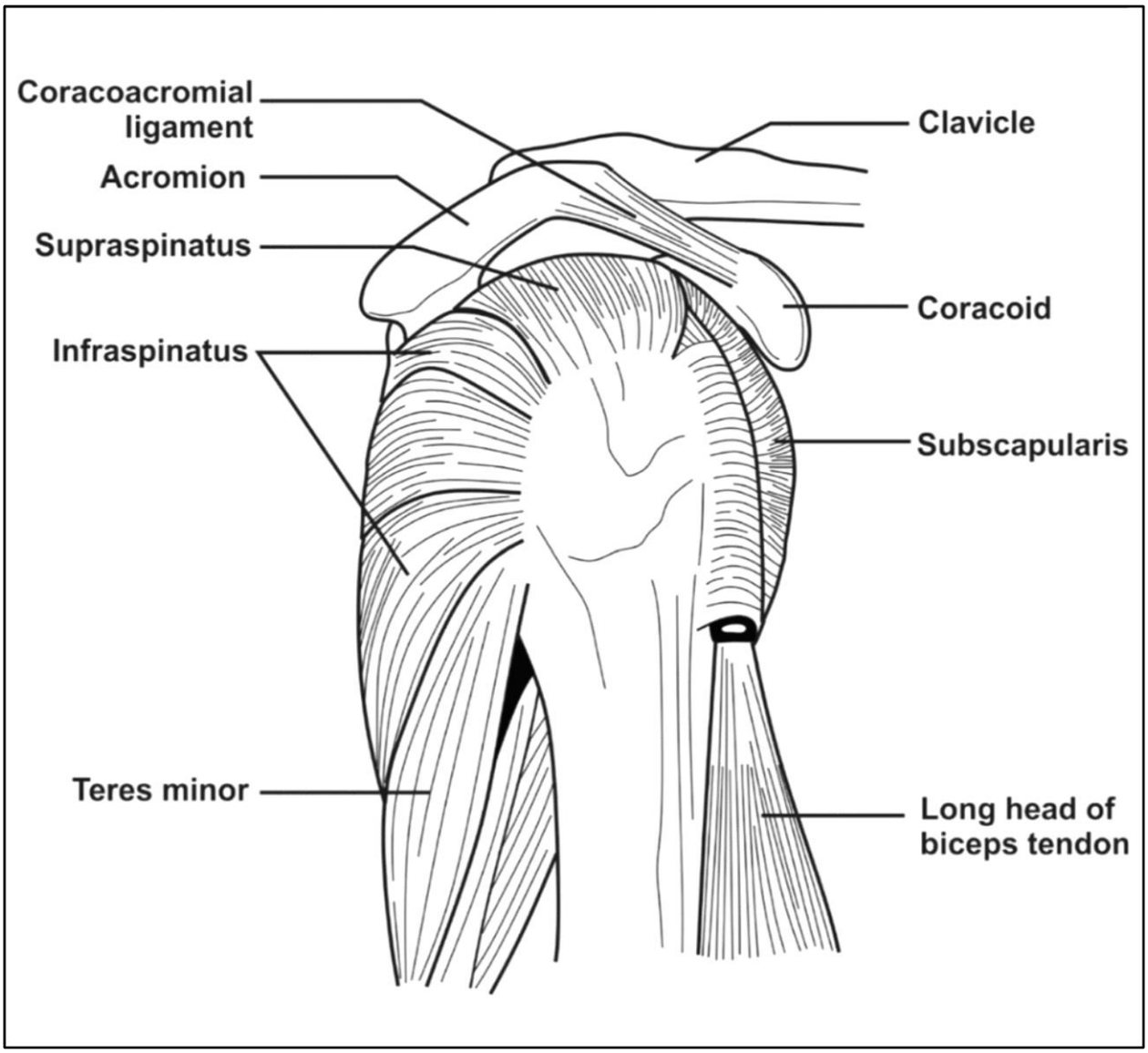


Figure 1. Showing separate insertions of the rotator cuff tendons.[12]

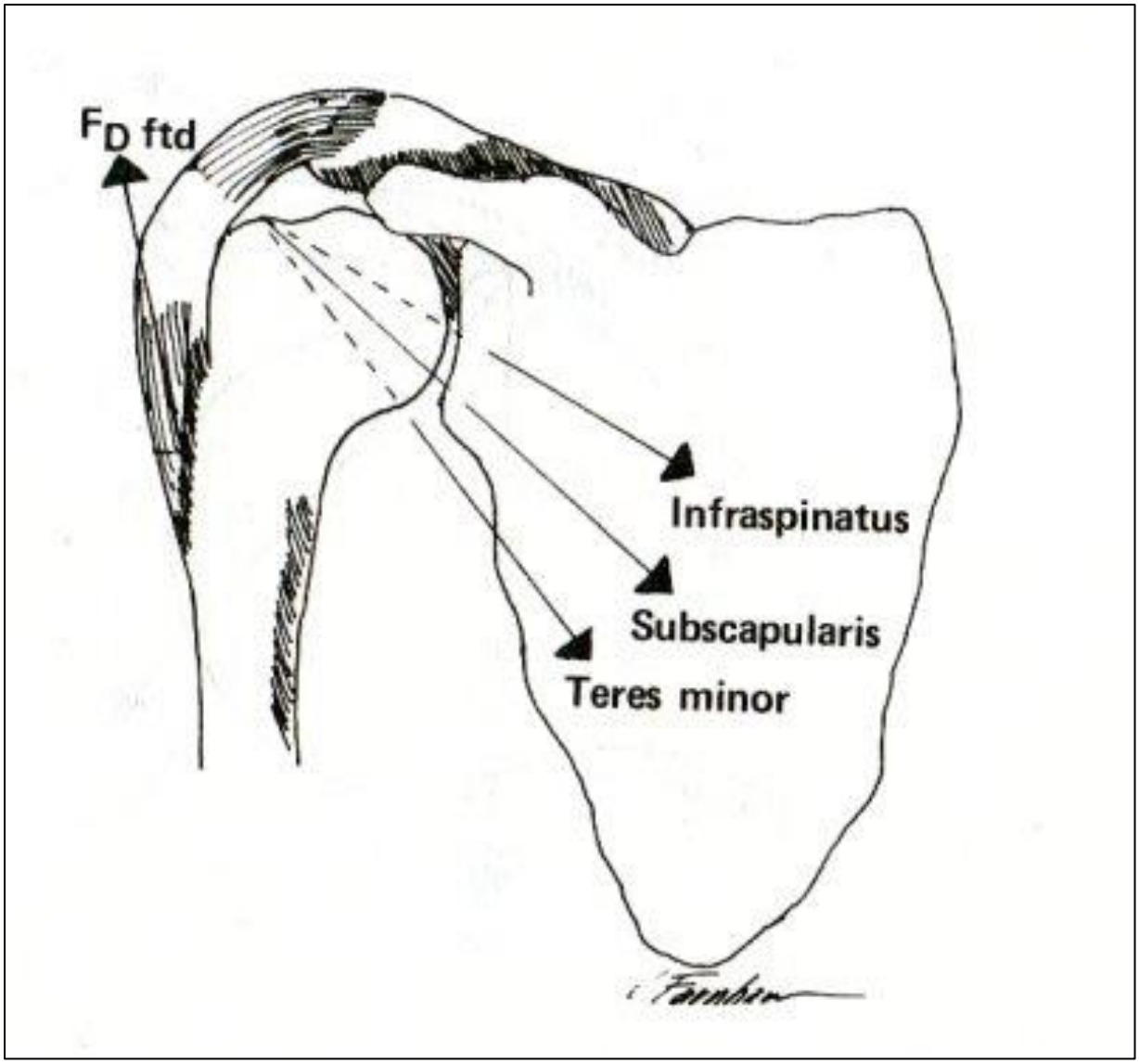


Figure 2. Approximation of the humeral head to the glenoid by balanced force coupling moments between subscapularis and both infraspinatus and teres minor during early abduction.

*Image courtesy of http://3.bp.blogspot.com/-LNBjYjWllwo/VmmP8SGuAZI/AAAAAAAACZ4/_4vfmnJcXVI/s1600/shoulder%2Bcentration.jpg*

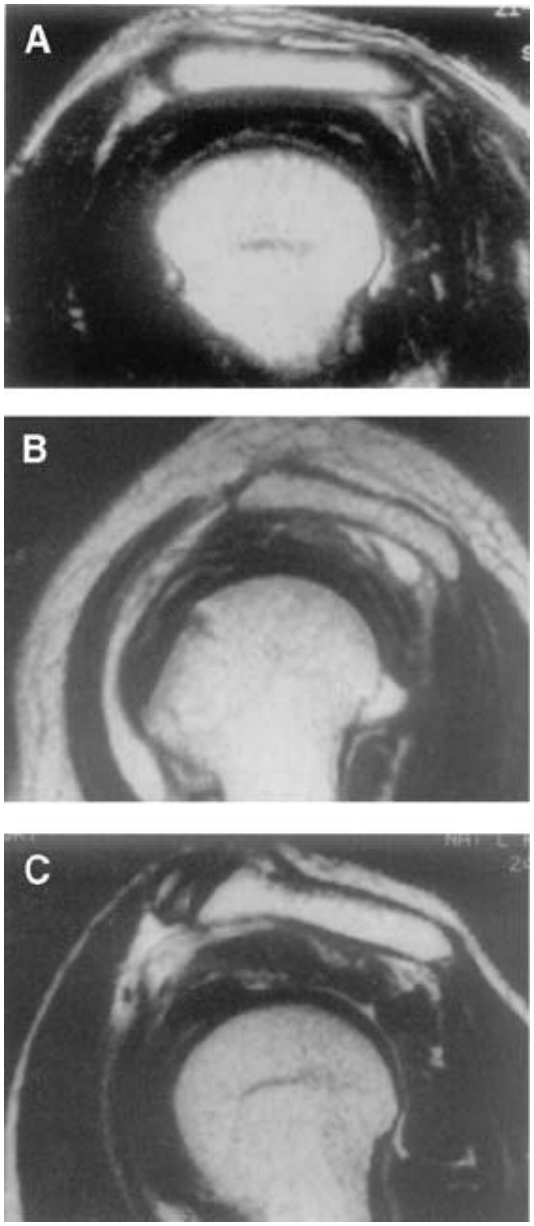


Figure 3. Acromial shapes determined by the sagittal oblique plane on MRI: type I (flat) (**A**), type II (curved) (**B**), and type III (hooked) (**C**). The longest dimension of the acromion was used to assess type[20]

Figure 4. Hawkins-Kennedy Test

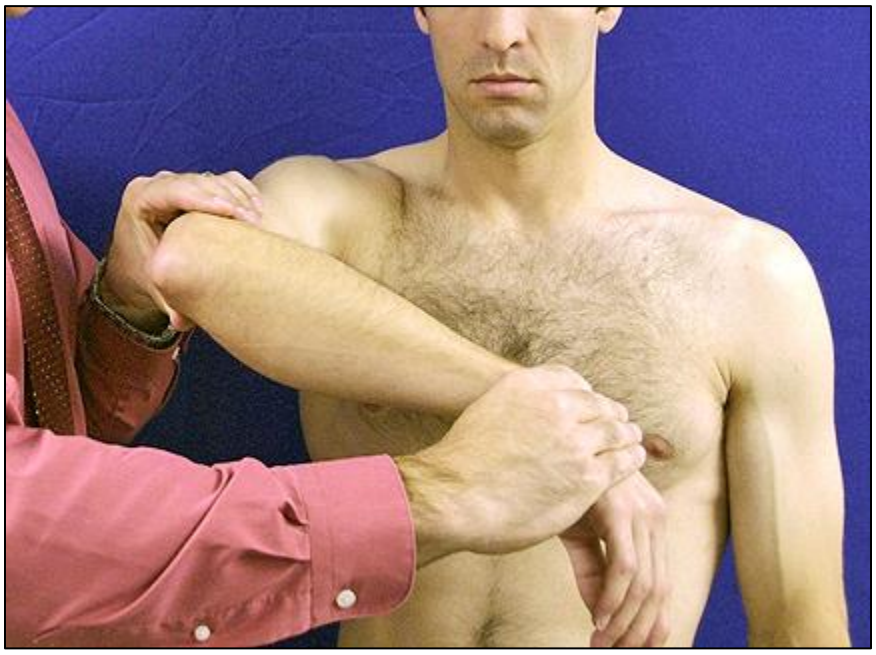

*Image courtesy of http://s0www.utdlab.com/contents/images/EM/60425/Hawkins_Kennedy_test.jpg?title=Hawkins+Kennedy+test+for+shoulder+impingement*

Figure 5. Neer Test.

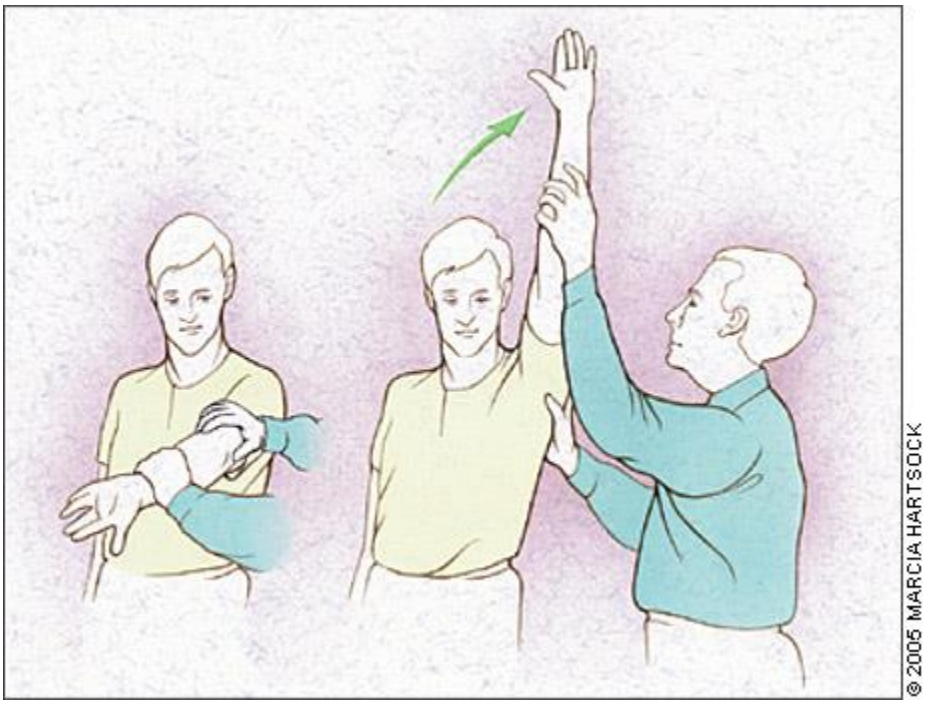


*Image coutesy of https://i.pinimg.com/originals/6c/2b/a6/6c2ba65e7e7363d94c3aa28e80c34428.jpg*

Figure 6. Painful Arc Test

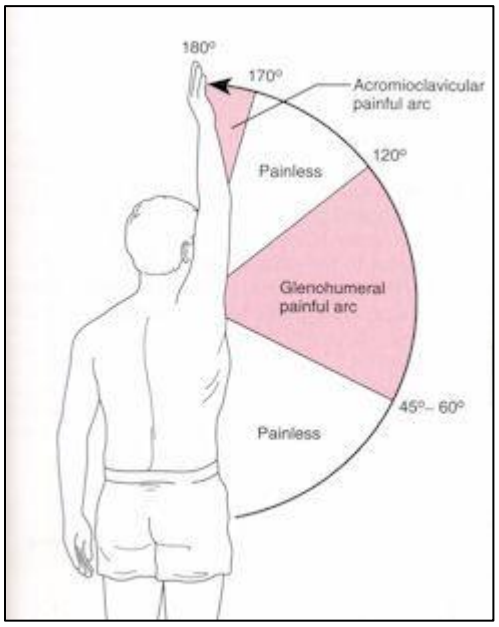


*Image courtesy of https://i.pinimg.com/originals/79/97/eb/7997eb866bbf8a8fee02055e3a4715da.jpg*

Figure 7. Empty Can Test[24]

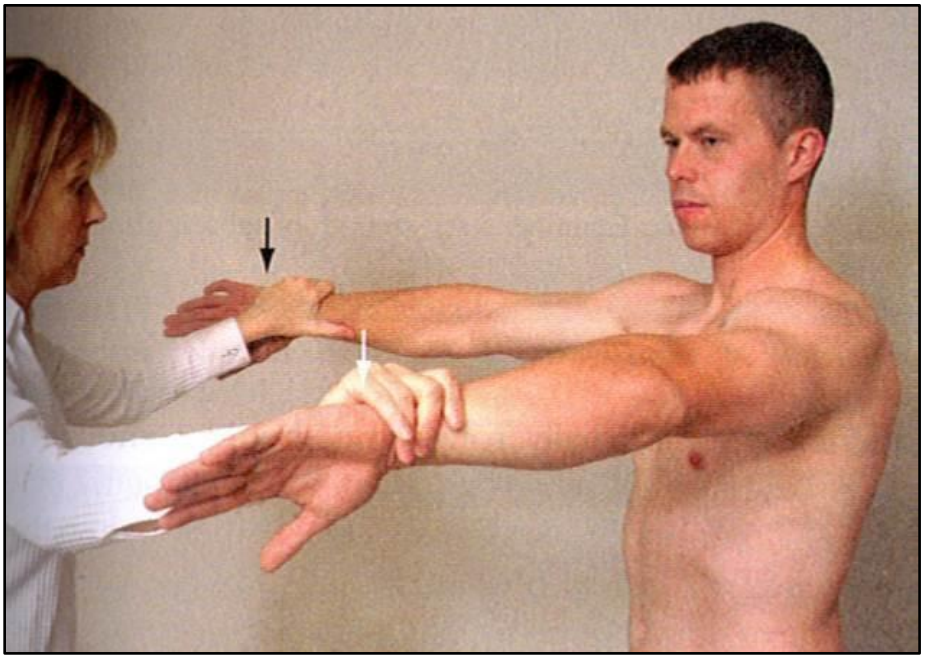

Figure 8. External Rotation Resistance Test

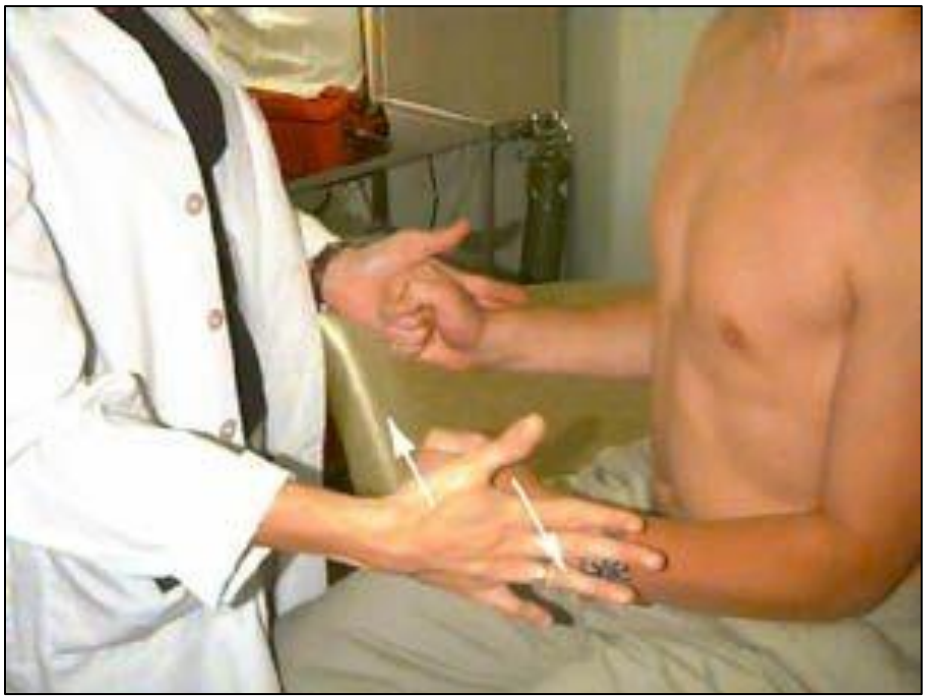

**Image courtesy of http://www.nismat.org/data/images/sexam1352199481017.jpg*

Table 1. Size of rotator cuff tear by acromion shape on $T_2$-weighted MRI.[20]

| Acromial shape | Diameter in coronal oblique plane (cm) | Diameter in sagittal oblique plane (cm) |
|---|---|---|
| Type I (n = 33, 36.3%) | 1.9 ± 1.4 | 1.8 ± 1.2 |
| Type II (n = 22, 24.2%) | 2.0 ± 1.4 | 1.7 ± 1.2 |
| Type III (n = 36, 39.5%) | 2.7 ± 1.4* | 2.4 ± 1.3* |

*$p<0.05$ type III vs type I or type II.

Table 2. Interrater Kappa Reliability Coefficients and Agreements of 5 special tests for subacromial impingement syndrome.[29]

| Test | Kappa Coefficient (95% CI) | Percentage Agreement |
|---|---|---|
| Hawkins-Kennedy | .39 (.12–.65) | 69 |
| Neer | .40 (.13–.67) | 71 |
| Painful arc | .45 (.18–.72) | 73 |
| Empty can (Jobe) | .47 (.22–.72) | 76 |
| External rotation resistance | .67 (.40–.94) | 87 |

Table 3. Diagnostic Accuracy of Subacromial Impingement Shoulder Tests[29]

| Test | Sensitivity (95% CI) | Specificity (95% CI) | +LR (95% CI) | −LR (95% CI) |
|---|---|---|---|---|
| Hawkins-Kennedy | .63 (.39–.86) | .62 (.46–.77) | 1.63 (.94–2.81) | .61 (.31–1.20) |
| Neer | .81 (.62–1.0) | .54 (.38–.69) | 1.76 (1.17–2.66) | .35 (.12–.97) |
| Painful arc | .75 (.54–.96) | .67 (.52–.81) | 2.25 (1.33–3.81) | .38 (.16–.90) |
| Empty can (Jobe) | .50 (.26–.75) | .87 (.77–.98) | 3.90 (1.50–10.12) | .57 (.35–.95) |
| External rotation resistance | .56 (.32–.81) | .87 (.77–.98) | 4.39 (1.74–11.07) | .50 (.28–.89) |

Table 4. The 2 rehabilitation protocols post-surgical intervention.[38]

<table>
<tr><th>Period</th><th>Aggressive Early Passive Rehabilitation (Group A)</th><th>Limited Early Passive Rehabilitation (Group B)</th></tr>
<tr><td>Hospitalization Period</td><td>Forward flexion up to tolerable angle (usually >90 degree)<br>External rotation up to 30 degree<br><br>By therapist (2 times*)<br>By patient (3 times)</td><td>Forward flexion (<90 degree)<br><br>By CPM (2 times)</td></tr>
<tr><td>After discharge to 3 weeks</td><td rowspan="2">Forward flexion up to tolerable angle (usually > 90 degree)<br>External rotation up to 30 degree<br><br>By patient (3 times)</td><td>Forward flexion (<90 degree)<br><br>By patient (2 times)</td></tr>
<tr><td>3 weeks to 6 weeks</td><td>Forward flexion up to tolerable angle (usually >90 degree)<br>External rotation up to 30 degree<br><br>By patient (2 times)</td></tr>
<tr><td>After 6 weeks</td><td colspan="2">Start active assisted exercise<br>Permit all passive exercises including internal rotation and abduction</td></tr>
</table>

**References**


1. Pratt NE. Anatomy and biomechanics of the shoulder. *J Hand Ther.* 1994 Apr-Jun;7(2):65-76.
2. van Kampen DA, van den Berg T, van der Woude HJ, Castelein RM, Scholtes VA, Terwee CB, Willems WJ. The diagnostic value of the combination of patient characteristics, history, and clinical shoulder tests for the diagnosis of rotator cuff tear. *J Orthop Surg Res.* 2014 Aug 7;9:70.
3. Akpinar S, Ozkoç G, Cesur N. Anatomy, biomechanics, and physiopathology of the rotator cuff. *Acta Orthop Traumatol Turc. 2003*;37 Suppl 1:4-12.
4. Teunis T, Lubberts B, Reilly BT, Ring D. A systematic review and pooled analysis of the prevalence of rotator cuff disease with increasing age. *J Shoulder Elbow Surg*. 2014 Dec;23(12):1913-1921.
5. Clark JM, Harryman DT 2nd. Tendons, ligaments, and capsule of the rotator cuff. Gross and microscopic anatomy. *J Bone Joint Surg Am.* 1992 Jun;74(5):713-25.
6. Karas V, Wang VM, Dhawan A, Cole BJ. Biomechanical factors in rotator cuff pathology. *Sports Med Arthrosc.* 2011 Sep;19(3):202-6.
7. Prescher A. Anatomical basics, variations, and degenerative changes of the shoulder joint and shoulder girdle. *Eur J Radiol.* 2000 Aug;35(2):88-102.
8. Bassett RW, Browne AO, Morrey BF, An KN. Glenohumeral muscle force and moment mechanics in a position of shoulder instability. *J Biomech.* 1990;23(5):405-15.
9. Thompson WO, Debski RE, Boardman ND 3rd, Taskiran E, Warner JJ, Fu FH, Woo SL. A biomechanical analysis of rotator cuff deficiency in a cadaveric model. Am J Sports Med. 1996 May-Jun;24(3):286-92.

10. Vosloo M, Keough N, De Beer MA. The clinical anatomy of the insertion of the rotator cuff tendons. *Eur J Orthop Surg Traumatol.* 2017 Apr;27(3):359-366.
11. Hertling D, Kessler RM. Chapter 11: Shoulder and Shoulder Girdle. *Management of Common Musculoskeletal Disorders: Physical Therapy Principles and Methods 4th Edition*. Lippincott Willians & Wilkins, Philadelphia, Pennsylvania: 2006.
12. Yamamoto A, Takagishi K, Osawa T, Yanagawa T, Nakajima D, Shitara H, Kobayashi T. Prevalence and risk factors of a rotator cuff tear in the general population. *J Shoulder Elbow Surg.* 2010 Jan;19(1):1 16-20.
13. Rothman RH, Parke WW. The vascular anatomy of the rotator cuff. *Clin Orthop Relat Res.* 1965 Jul-Aug;41:176-86.
14. Gross MT. Chronic tendinitis: pathomechanics of injury, factors affecting the healing response, and treatment. *J Orthop Sports Phys Ther.* 1992;16(6):248-61.
15. Nakajima T, Rokuuma N, Hamada K, et al. Histologic and biomechanical characteristics of the supraspinatus tendon: reference to rotator cuff tearing. *J Shoulder Elbow Surg*. 1994;3:79–87.
16. Huang CY, Wang VM, Pawluk RJ, et al. Inhomogeneous mechanical behavior of the human supraspinatus tendon under uniaxial loading. J Orthop Res. 2005;23:924–930.
17. Bey MJ, Kline SK, Zauel R, et al. Measuring dynamic in-vivo glenohumeral joint kinematics: technique and preliminary results. *J Biomech*. 2008;41:711–714.
18. Wuelker N, Plitz W, Roetman B, Wirth CJ. Function of the supraspinatus muscle. Abduction of the humerus studied in cadavers. *Acta Orthop Scand.* 1994 Aug;65(4):442-6.

19. Ha AS, Petscavage-Thomas JM, Tagoylo GH. Acromioclavicular joint: the other joint in the shoulder. *AJR Am J Roentgenol.* 2014 Feb;202(2):375-85.
20. Hirano M, Ide J, Takagi K. Acromial shapes and extension of rotator cuff tears: magnetic resonance imaging evaluation. *J Shoulder Elbow Surg.* 2002 Nov-Dec;11(6):576-8.
21. Lovering RM, Russ DW. Fiber type composition of cadaveric human rotator cuff muscles. *J Orthop Sports Phys Ther.* 2008 Nov;38(11):674-80.
22. Ludewig PM, Reynolds JF. The association of scapular kinematics and glenohumeral joint pathologies. *J Orthop Sports Phys Ther.* 2009 Feb;39(2):90-104.
23. Harrison AK, Flatow EL. Subacromial impingement syndrome. *J Am Acad Orthop Surg.* 2011 Nov;19(11):701-8.
24. Magee DJ. Chapter 5: Shoulder. *Orthopedic Physical Assessment 6tht Ed.* Elsevier Saunders; St. Louis, Missouri: 2014.
25. Brewer BJ. Aging of the rotator cuff. *Am J Sports Med.* 1979 Mar-Apr;7(2):102-10.
26. Woollard JD, Bost J2, Piva SR, Kelley Fitzgerald G, Rodosky MW, Irrgang JJ. The ability of preoperative factors to predict patient-reported disability following surgery for rotator cuff pathology. *Disabil Rehabil.* 2017 Oct;39(20):2087-2096.
27. Nitz AJ, Bellew JW Jr, Hazle CR. Evaluation of Musculoskeletal Disorders. *Orthopedic and sports physical therapy.* 3rd ed. St. Louis: Mosby-Year Book; 1997.
28. Silva L, Andréu JL, Muñoz P, Pastrana M, Millán I, Sanz J, Barbadillo C, Fernández-Castro M. Accuracy of physical examination in subacromial impingement syndrome. *Rheumatology (Oxford).* 2008 May;47(5):679-83.

29. Michener LA, Walsworth MK, Doukas WC, Murphy KP. Reliability and diagnostic accuracy of 5 physical examination tests and combination of tests for subacromial impingement. *Arch Phys Med Rehabil*. 2009 Nov;90(11):1898-903.
30. Magee T, Williams D. 3.0-T MRI of the supraspinatus tendon. *AJR Am J Roentgenol*. 2006 Oct;187(4):881-6.
31. Hart L. Corticosteroid and other injections in the management of tendinopathies: a review. *Clin J Sport Med*. 2011 Nov;21(6):540-1.
32. Masood Umer, Irfan Qadir, and Mohsin Azam. Subacromial impingement syndrome. *Orthop Rev*. 2012 May 9; 4(2): e18.
33. Stevenson K, Jackson S, Shufflebotham J, Roddy E, Foster NE. Development and delivery of a physiotherapist-led exercise intervention in a randomised controlled trial for subacromial impingement syndrome (the SUPPORT trial). *Physiotherapy.* 2017 Dec;103(4):379-386.
34. Kim SY, Ko JB, Farthing JP, Butcher SJ. Investigation of supraspinatus muscle architecture following concentric and eccentric training. J Sci Med Sport. 2015 Jul;18(4):378-82.
35. Lorbach O, Baums MH, Kostuj T, Pauly S, Scheibel M, Carr A, Zargar N, Saccomanno MF, Milano G. Advances in biology and mechanics of rotator cuff repair. *Knee Surg Sports Traumatol Arthrosc*. 2015 Feb;23(2):530-41.
36. Chung SW, Kim JY, Kim MH, Kim SH, Oh JH. Arthroscopic repair of massive rotator cuff tears: outcome and analysis of factors associated with healing failure or poor postoperative function. *Am J Sports Med.* 2013 Jul;41(7):1674-83.

37. Iannotti JP. A delayed physical therapy protocol that limited passive range of motion was similar to a protocol with early passive range of motion after rotator cuff repair. *J Bone Joint Surg Am.* 2012 Nov 21;94(22):2094.
38. Lee BG, Cho NS, Rhee YG. Effect of two rehabilitation protocols on range of motion and healing rates after arthroscopic rotator cuff repair: aggressive versus limited early passive exercises. *Arthroscopy.* 2012 Jan;28(1):34-42.
39. Chang KV, Hung CY, Han DS, Chen WS, Wang TG, Chien KL Early Versus Delayed Passive Range of Motion Exercise for Arthroscopic Rotator Cuff Repair: A Meta-analysis of Randomized Controlled Trials. *Am J Sports Med.* 2015 May;43(5):1265-73.