REVIEW

Shoulder Impingement Syndromes: Implications on Physical Therapy Examination and Intervention

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Abstract. A painful shoulder presents challenges in examination, diagnosis and intervention for the physical therapist because of the complexity of the structures involved. A common cause of shoulder pain is shoulder impingement syndrome. This was first described as a condition in which the soft tissues of the subacromial space were chronically entrapped and compressed between the humeral head and the subacromial arch. This definition does not account for the myriad potential causes of shoulder impingement conditions, as forms of impingement other than subacromial soft tissue compression may explain different symptomatic shoulder injuries. This paper describes shoulder impingement syndromes that have been hypothesized, identified and analyzed in the literature. Physical Therapy examination and intervention for these syndromes are also discussed.

Key words: impingement syndrome, shoulder pain, subacromial impingement, posterosuperior glenoid rim impingement, subcoracoid impingement, suprascapular nerve impingement, physical therapy examination and intervention

A painful shoulder presents challenges in examination, diagnosis and intervention for the physical therapist because of the complexity of the structures involved. The physical therapist must establish a detailed examination, which includes the history, systems review and tests and measures. The therapist evaluates the data from the examination to organize the information into clusters, syndromes and categories to determine the prognosis (including the plan of care) and the most appropriate intervention strategies. Essential to this process is a thorough understanding of the anatomic, biomechanic and pathophysiologic implications of the condition.

Shoulder impingement and rotator cuff injuries are among the most common causes of shoulder pain\(^1\). Rotator cuff problems account for one-third of physician visits for shoulder pain complaints especially for those individuals who are involved in repetitive use of the shoulder\(^2\). Shoulder impingement syndrome was described in detail by Neer in 1972 as a painful condition in which the soft tissues of the subacromial space (bursa, rotator cuff tendons, biceps tendon) were chronically entrapped and compressed between the humeral head and the subacromial arch\(^3,4\). In the 1970’s and the 1980’s, treatments were devised and refined based on this concept\(^5\). However, as clinicians and researchers studied this condition, they noted that Neer’s description did not always account for the numerous shoulder impingement conditions being seen. Consequently, additional symptomatic impingement syndromes have been hypothesized, identified and finally analyzed\(^1\).

Before discussing the actual classifications of impingement syndromes, it is helpful to briefly review the anatomy and biomechanics of the shoulder upon which the classifications are based.

Functional Anatomy and Biomechanics of the Shoulder

The shoulder joint is a remarkable joint structure that enjoys a greater range of motion than any other articulation in the human body. The joints of the shoulder complex have to rely on their associated ligaments and muscles to provide stability. As a result these soft tissues are

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susceptible to injury and degeneration. Several important anatomical structures and biomechanical concepts pertinent to the diagnosis of shoulder impingement syndromes will be presented.

The glenohumeral joint is a true ball-and-socket articulation. The articular surface of the humeral head is markedly larger than the concave articular surface of the glenoid. The glenoid fossa faces superiorly, medially and posteriorly, with a surface area only a quarter to a third that of the humeral head articular surface. This allows for large excursions of motion but contributes to the inherent instability of this joint.

The glenoid cavity of the scapula articulates with the humeral head. The greater and lesser tuberosities are two osseous prominences just distal to the anatomic neck of the humerus, and provide sites of attachment for the rotator cuff tendons. With the arm in the dependent position, the greater tuberosity is situated laterally, receiving the supraspinatus, infraspinatus and teres minor tendons, while the lesser tuberosity provides an attachment for the subscapularis. The tuberosities act as levers, placing the forces generated by these muscles at a greater distance from the axis of rotation of the glenohumeral joint to increase the resultant muscle torque.

The superior, middle and inferior glenohumeral ligaments are three capsular thickenings that act as check reins during external rotation of the humeral head. The superior glenohumeral ligament functions to support the dependent humerus by preventing the downward subluxation of the bone. It minimally contributes to anterior stability of the glenohumeral joint. According to Peidro and colleagues, the middle glenohumeral ligament provides stability during external rotation and “its stabilizing function is most crucial between 60 and 90 degrees of arm abduction.” The inferior glenohumeral ligament is the thickest of the three ligaments and provides significant dynamic stabilization of the complex by limiting external rotation with increasing abduction of the arm.

The rotator cuff is composed of the supraspinatus, infraspinatus, teres minor and the subscapularis muscles. These muscles produce motion at the glenohumeral joint as well as assisting with joint stability and nutrition. These muscles arise from the scapula, with their tendons intermingling and becoming inseparable from each other and the capsule as they insert on the humerus.

The supraspinatus muscle is a fusiform-shaped muscle that originates in the supraspinatus fossa on the dorsal scapula and inserts onto the greater tuberosity. A third of its way from the origin to its insertion is the intramuscular tendon. In the sagittal plane, the tendon lies under the central portion of the acromion. One author noted that this muscle is actually composed of a ventral and a dorsal portion, and that the rotator cuff tears appear to occur anteriorly within the ventral muscle portion.

The vascularity of the rotator cuff has been noted as a possible contributing factor to rotator cuff injuries. According to Burns and Whipple, there are “consistent areas of sparse vascularity” in this region, and repeated trauma in an already vascular-deficient portion may result in inflammatory response, leading to swelling of the tendinous structures. Prescher notes that the rotator cuff is “poorly vascularized near its insertion zone”, and states that resulting degenerative changes in the area can lead to tears in the rotator cuff.

The acromioclavicular joint is formed by the acromion process of the scapula with the acromial end of the clavicle and is a relatively immobile articulation. Because scapular rotation is necessary for normal movement, disease or ossification of the acromioclavicular joint encourages the scapula and clavicle to move even more as a single unit. This pattern of use alters the path of instant center of rotation (PICR) of the scapula. Changes in the PICR of the scapula can lead to micro or macrotrauma of the subacromial structures.

The scapular spine bends at the acromial angle and becomes the acromion. This region contains several ossification sites which subsequently fuse, but in about 7–15% of cases, a disturbance in this process leads to a group of variations known as ‘os acromiale’ which can be a source of pain and impingement on the shoulder. Moreover, there are curvature variations of the acromion. Of the three acromial shapes (flat, curved or hooked), a hooked or curved undersurface combined with an increased inferior tilt is associated with a greater incidence of rotator cuff tears.

The coracohumeral ligament, which extends from the coracoid process to the more distal humeral head, divides the subacromial space into two compartments. The subacromial/subdeltoid bursa occupies the first compartment, and is in intimate contact with the anterior border of the acromion and the coracoacromial ligament. The supraspinatus outlet, which is the archway formed above the glenoid is the most common site of impingement. The second compartment is a space between the coracoid process and the lesser tuberosity.

Scapulohumeral rhythm is the term which refers to the normal and coordinated movement of arm elevation as a result of an intricate interplay of the four articulations in the shoulder complex. The scapula moves very slightly during the first 30 degrees of humeral abduction. As abduction proceeds, the scapula rotates laterally and the
sternoclavicular joint elevates, causing movement at the acromioclavicular joint. Rotation of this joint occurs around the longitudinal axis of the clavicle as the coracoclavicular ligaments pull on the clavicle’s inferior aspect. The humerus must also rotate laterally before 90 degrees to prevent the greater tuberosity from impinging on the inferior portion of the acromion process. There are several variations in the literature regarding the exact ratio value of the scapulohumeral rhythm, and it has been postulated that these differences are due to factors as arm position, gender, resisted or unresisted motion and glenohumeral capsule mobility. In general, however, most concede that the range of scapular motion does not exceed 60 degrees, while the range of glenohumeral joint does not exceed 120 degrees.

**Classifications of Shoulder Impingement Syndromes**

The four most commonly described types of shoulder impingement are anterior acromial impingement, posterosuperior glenoid rim impingement, subcoracoid impingement and suprascapular nerve (at the spinoglenoid notch) impingement. A summary table containing these syndromes, their possible causes and other hallmarks is presented on Table 1.

**Anterior subacromial impingement syndrome**

The anterior subacromial impingement syndrome was first described by Neer in the 1970’s, and has been the basis for several studies on this topic. In his study of the acromion, Neer suggested that impingement occurs when the soft tissues filling the subacromial space (subacromial bursa, supraspinatus tendon, biceps tendon) are chronically entrapped between the humeral head and the coracoacromial arch. This condition is associated with progressive degenerative changes of the supraspinatus tendon in its hypovascular region and of the biceps tendon. The predisposing factors to this condition include the shape and orientation of the acromion, presence of osteophytes at the acromioclavicular joint, ossification of the coracoacromial ligament and the presence of os acromiale. Neer postulated that 90 to 95% of rotator cuff tears result from subacromial impingement that progress in a predictable manner. Neer described a three stage progression from impingement to cuff tear. Stage 1 consists of tendon swelling and hemorrhage and widening of the subacromial-deltoid bursa. This stage is common in young athletes (swimming, throwing sports) and must be differentiated from acromioclavicular arthropathy and shoulder instability. Stage 2 disorder consists of fibrosis and thickening of the rotator cuff tendons including microscopic and partial tears. Stage 3 represents partial or complete rotator cuff or biceps tendon tear, with associated bony changes such as greater tuberosity sclerosis and acromion traction osteophytes. Pain and disability are severe in Stage 3.

Neer developed the anterior acromioplasty to eliminate the source of the impingement; a technique that has been widely used with favorable results. In this technique, the undersurface of the acromion is rendered flat, without overhang, while preserving the entire length of the acromion. In this configuration, part of the acromion still extends beyond the anterior border of the clavicle. Rockwood and Lyons noted a large number of patients who either had continuing or recurring symptoms of impingement after anterior acromioplasty as proposed by Neer. Despite the removal of the anterior acromion, the remaining portion of the clavicle may impinge the subacromial bursa and rotator cuff during flexion of the shoulder joint.

**Table 1. Summary of shoulder impingement syndromes**

<table>
<thead>
<tr>
<th>Type of shoulder impingement syndrome</th>
<th>Possible causes</th>
<th>Other hallmarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior subacromial impingement</td>
<td>soft tissues filling the subacromial space (subacromial bursa, supraspinatus tendon, biceps tendon) are chronically entrapped between the humeral head and the coracoacromial arch</td>
<td>Neer described a three-stage progression from impingement to cuff tear; several variations anterior acromioplasty has been shown to have good results</td>
</tr>
<tr>
<td>Posterosuperior glenoid rim impingement</td>
<td>Compression of the inner fibers of the rotator cuff and the fibers of the posterior superior labrum between the greater tuberosity and the posterior superior glenoid</td>
<td>Association between repetitive motion and impingement syndrome; has been seen in patients involved in throwing sports or top level water polo players; forklift drivers also seen with the condition</td>
</tr>
<tr>
<td>Subcoracoid impingement</td>
<td>May result from posterior elevation of the humerus in a plane of 30 degrees from the frame combined with internal rotation</td>
<td>Continuous contact between the coracoid tip and most prominent part of the lesser tuberosity; surgical intervention possible</td>
</tr>
<tr>
<td>Suprascapular nerve impingement</td>
<td>Encroachment of the suprascapular nerve</td>
<td>Calcified or hypertrophic spinoglenoid ligament and excessive nerve angulation when it curves the infraspinus fossa are important pathogenic factors</td>
</tr>
</tbody>
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arm. This observation then led to a modified Neer acromioplasty, which involves removing the anteroinferior portion of the acromion after removing the anterior prominence of the acromion. The authors reported consistently good or excellent results from this procedure\(^5\).

Another surgical option for patients with less severe forms of anterior subacromial impingement is division of the coracoacromial ligament without acromioplasty. In the study by Burns and Whipple\(^13\) it was suggested that the main source of impingement in younger patients is the coracoacromial ligament and that in these patients, anterior acromioplasty is unnecessary unless there are osteophytes in the acromion.

**Posterosuperior glenoid rim impingement**

In the 1990s, with the advent of more advanced imaging procedure such as anatomic, kinesiologic and magnetic resonance imaging and arthroscopic data, another type of rotator cuff impingement was discovered resulting from compression of the inner fibers of the rotator cuff and the fibers of the posterior superior labrum between the greater tuberosity and the posterior superior glenoid\(^5\). An association between repetitive motion (in patients who perform repetitive activities at work or sports) in the shoulder and impingement syndrome and rotator cuff tears has been postulated by several authors\(^5\). This condition has been reported as a cause of shoulder pain during the late cocking phase in patients involved in throwing sports\(^9\) and in top level water polo players\(^20\). This type of injury can also occur in non-throwers. Forklift drivers may develop an internal impingement while driving in reverse and looking over their opposite shoulder as their steering arm is placed in a position of abduction and external rotation\(^5\).

**Subcoracoid impingement syndrome**

A decrease in the capacity of the coracohumeral compartment can be due either to a decrease in space or to an increase in contents\(^15\). Subcoracoid compression of the supraspinatus tendon may result from posterior elevation of the humerus in a plane of 30 degrees from the frame combined with internal rotation\(^13\). Anterior pain over the coracoid results from repeated arm flexion and internal rotation. The continuous contact between the coracoid tip and the most prominent part of the lesser tuberosity causes progressive bone degeneration, inflammation of the subscapularis bursa and substance damage of the subscapularis tendon\(^4\) or ossification\(^13\) that may develop and result in isolated partial or complete tear\(^4\). Impingement of the tendinous cuff on the coracoid process has also been noted in patients following an unsuccessful rotator cuff surgery\(^21\).

The most important favoring factors are inherent abnormalities in orientation and length of the coracoid process\(^22\) and acquired bone changes in the coracohumeral space components resulting from lesser tuberosity or coracoid process displaced fractures especially if healed in pseudoarthrosis. The coracohumeral space can be crowded by an isolated traumatic tear of the subscapularis, usually associated with a dislocation of the long head of the biceps\(^15\). Neoplasms on the base of the coracoid process can also impinge the supraspinatus tendon\(^23\).

Surgical intervention of subcoracoid impingement syndromes include reattachment of the subscapularis, open reduction of the long head of the biceps followed by repair of the transverse ligament, removal of calcific deposits and fixation of chip fractures. Coracoplasty is also a treatment option although this entails the resection of the coracoacromial ligament\(^15\).

**Suprascapular nerve impingement syndrome**

The suprascapular nerve may lie no more than 1 cm from the glenoid rim\(^24\) and may be susceptible to impingement. A calcified or hypertropic spinoglenoid ligament and excessive nerve angulation, when it curves entering the infraspinatus fossa, are considered important pathogenetic factors for this syndrome. Nerve entrapment from ganglion cysts at the spinoglenoid notch\(^23\) or consequent to local trauma has been reported. This syndrome has been reported in volleyball players of both genders. Infraspinatus muscle atrophy, decreased strength in external rotation and pain in the posterior and lateral dominant arm are found in approximately 20% of professional players. Degeneration of the nerve secondary to friction and stretching of the terminal branches against the base of the notch have been reported as one of the causes of the syndrome\(^4\).

### Implications on Physical Therapy Intervention: Incorporating the Guide to Physical Therapist Practice in Examination, Differential Diagnosis and Intervention of Shoulder Impingement Syndromes

It is evident that there are several other forms of shoulder impingement aside from the traditionally understood anterior subacromial impingement. Clinicians need to identify and select tests and measures that will allow them to differentially diagnose the condition and then to determine the appropriate prognosis (including plan of care) and intervention. Information contained in The Guide to Physical Therapist Practice (The Guide)\(^25\) will be utilized to analyze differences between the shoulder impingement syndromes, including considerations regarding intervention strategies.

According to The Guide, examination is a comprehensive screening and specific testing process that leads to a diagnostic classification or, when appropriate, to a referral to another professional\(^25\). Through the examination,
the physical therapist may identify impairments, functional limitations, disabilities, changes in physical function or overall examination findings to establish the diagnosis and the prognosis (including the plan of care). The three components of examination include patient/client history, systems review and tests and measures. The patient client history may provide important clues as to the possible causes of impingement. For example, Stages 1 and 2 of Neer’s anterior subacromial impingement classification usually occurs in younger individuals26). Neer’s Stage 3 is common in patients over 40, as degenerative bony changes (e.g. roughening, erosion, osteophyte formation) usually occur as the condition progresses. Repetitive strain on the shoulder has been cited as a contributing factor to impingement5,26). Previous history of shoulder subluxation, dislocation, or instability may predispose an individual to superior glenoid impingement. Jobe coined the term “silent subluxators” to describe the pathophysiology of shoulder pain in the overhead athlete27). Information regarding prior surgery and medical tests such as diagnostic imaging or exploratory arthroscopy will aid in identifying the specific structures involved. The systems review may reveal other predisposing factors to impingement, including the identification of systems interactions that may potentially hasten or delay the rehabilitation process, or may indicate the need for referral to another professional.

Considering tests and measures, the location of pain may give the clinician additional information on the nature of the disorder. For example, throwers with superior glenoid impingement typically complain of pain along the posterior joint line when the arm is externally rotated to 90° of abduction and then horizontally extended5, while individuals with anterior subacromial impingement often complain of pain in the anterior aspect of the shoulder especially with arm elevation19). In terms of joint integrity and mobility, the therapist may identify areas of tenderness in the rotator cuff, subcoracoid and subacromial regions, coracoacromial ligament or joint capsule. Identification of joint play movement allows the therapist to identify any hypermobility or hypomobility in the shoulder. Posterior capsular tightness results in anterosuperior migration of the humeral head causing encroachment of the humerus on the coracoacromial ligament and/or acromion27). In contrast, if the capsule is lax and/or the dynamic stabilizers are inadequate, the humeral head will displace excessively, which may cause secondary impingement. In terms of special tests, the patient with a rotator cuff lesion due to impingement may demonstrate positive Neer impingement sign27), Hawkins-Kennedy27), Reverse Impingement Sign27), and Neer and Welch’s tests27). Since mild shoulder instability may be a predisposing factor for anterior acromial, posterosuperoglenoid rim and subcoracoid impingements, the load and shift test, the apprehension test, the relocation test5, and the Impingement test27) may be positive in these conditions.

Range of motion (including muscle length) will provide the therapist information regarding limitation of both active and passive range of motion in the shoulder joint as well as possible reasons for these limitations. ROM testing combined with selective tissue tension testing (SLTT) will point out whether the lesion is in the contractile or inert structures. However, because the impingement syndromes may affect both the contractile and inert structures, SLTT will not be sensitive enough to allow for differential diagnosis. Considering that scapular asymmetry may correlate with impingement, the physical therapist may use the modified lateral scapular slide test to compare scapular movement between sides during abduction28). The test is performed by measuring the distance between the T7 spinous process and the inferior angle of the scapula when the arm is in five different positions. According to Kibler28), a difference of greater than 1 cm between sides when the arm is in approximately 45 or 90-degree position correlates with glenohumeral impingement.

Examination of the patient’s sensory integrity and reflex integrity will determine any central or peripheral nervous system involvement as well as differentially diagnose other possible system and subsystem interactions that may account for the presenting signs and symptoms. A patient with a suprascapular nerve impingement may demonstrate decreased sensation as well as muscle atrophy10 of the infraspinatus and supraspinatus muscle. Lastly, muscle performance must be examined to identify patterns of weakness in the muscles of the shoulder girdle.

**Physical Therapy Intervention**

Physical therapy intervention for the patient with shoulder impingement syndrome will primarily depend on the presenting impairments and disabilities of the patient. Conservative management should be considered before to surgical intervention3,5,29–32). Also, as mentioned earlier, the key to successful management of this condition is the correct identification of the causes of the pathology to prevent recurrence of the condition and facilitate the patient’s achievement of highest functional level.

Several treatment interventions have been suggested for successful management of shoulder impingement syndromes. Decreasing pain, reducing joint and tissue swelling, inflammation and restriction must be achieved especially during the initial stages of the condition. Initial therapy should assist rather than retard soft tissue healing3). Application of therapeutic modalities, gentle active and passive range of motion, stretching and mobilization exercises17) are important in achieving this goal.

All mobility impairments should also be addressed. Interventions to decrease hypermobility, improve hypomobility and improve stability of the shoulder
girdle\(^{(17,33)}\) should be initiated to assist in the correct positioning of the humeral head, thereby avoiding impingement in the subacromial or subcoracoid areas and preventing the humerus from impinging on the posterosuperior glenoid rim.

Another goal is to improve force or torque production and endurance and alter length tension relationships\(^{(33)}\) in an effort to restore balance of the posterior and anterior structures of the shoulder. The therapist should also recognize the role of the muscles of the scapula\(^{(34)}\) in the normal functioning of the upper quadrant. Stretching and strengthening exercises, joint mobilizations and other manual therapy techniques\(^{(35)}\) must be provided with the goal of achieving precise active mobility, with consideration of the normal timing and sequence of movements in the clavicle, scapula and shoulder. Posture and movement reeducation must also be performed to incorporate these gains into function. This includes functional training for activities of daily living, instrumental activities of daily living and sport-specific tasks\(^{(33)}\). Ergonomic modifications must be initiated as well as other work-related modifications to prevent recurrence of the condition. The importance of patient education in the management of shoulder impingement syndromes must also be emphasized. This patient education must focus on home program compliance and empowerment of the patient\(^{(33)}\). The patient must be an active participant in the achievement of positive outcomes in rehabilitation of this condition.

In terms of specific physical therapy clinical protocols that are proven to improve outcomes of patients with a medical diagnosis of shoulder impingement syndromes, there are very few published studies that attempted to answer this question. A study by Conroy and Hayes\(^{(36)}\) investigated the effect of joint mobilization as a component of a comprehensive treatment for shoulder impingement syndrome. The study showed that joint mobilizations decreased the 24-hour pain rating and pain with subacromial compression test, but no differences in range of motion or function were observed. In the study by Bang and Deyle\(^{(32)}\) it was noted that subjects who received manual therapy techniques provided by a physical therapist showed significantly more improvements in pain and function compared to those who only received a supervised flexibility and strengthening program. This is the only study that has been found that assessed the validity of specific physical therapy interventions in improving a patient’s functional outcome measurements. A summary of the intervention highlights is presented in Table 2.

### Conclusion

The current available literature on the interventions for shoulder impingement syndromes is largely based on the premise that effective management of this diagnostic entity is predicated on the presenting alterations in the anatomic, physiologic and biomechanic properties secondary to the continuum of the condition, as well as the resulting impairments and disabilities experienced by the patient. Although most intervention strategies are grounded on solid scientific principles, the challenge to the physical therapy community is to objectively validate these approaches in an effort to generate the best practice parameters that will provide optimal results in the most expeditious amount of time and the least amount of resources. This becomes a more daunting task considering the significant variability in the presentation of the condition on each patient or client. The profession has to provide the society with scientific proof that the interventions provided are truly effective and efficient.

### References


### Table 2. Physical therapy intervention highlights for shoulder impingement syndromes

- Reduce pain and inflammation of the joint and tissues
- Address mobility impairments
  - decrease hypermobility, increase hypomobility and improve stability
  - improve force or torque production and endurance and alter length tension relationships
- Posture and movement reeducation
  - functional training for activities of daily living
  - ergonomic modifications
- Patient education
  - home program compliance
  - empowerment of patient