Throwing injuries of the shoulder*

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A review of the mechanism of throwing is presented and split into the different phases. The presentation and history is often more important than the clinical signs which may only be present after repetitive throwing activity. The pathological relationship between instability and impingement is discussed. Arthroscopy has been found to be extremely helpful in extending knowledge of pathological anatomy as well as defining diagnosis and aiding treatment.

Keywords: Shoulder, throwing injuries, arthroscopy, impingement and instability

Injuries related to the throwing sports are commonly seen in the general sports injury clinic. The upper extremity accounts for 75% of these injuries with the shoulder being the most common joint involved.

The act of throwing means different things to different sportsmen and in cricket it is against the rules for the bowler to throw the ball. It could be assumed that a throw is the action we make when throwing a pebble into the sea but in fact the sequence of events and biomechanics of this are completely different from that of the straight arm throw of javelin throwing (Figure 1), the centrifugally induced velocity of hammer throwing (Figure 2), the explosive push of putting the shot (Figure 3) and the spinning pull of the discus throw (Figure 4). The majority of basic biomechanical investigation has been done on the baseball pitcher’s throwing action and the study of this mechanism can help us analyse other throwing mechanisms.

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The throwing mechanism

To understand how to prevent and treat overuse injuries resulting from a throwing action, it is important to understand the anatomical and physiological sequence of events (Figure 5).

Wind-up phase

Weight is transferred to the back foot, the trunk laterally flexed and arm extended. This can be separated into two parts, early and late cocking. In early cocking the arm is abducted at 90° and horizontally extended 30°. Late cocking involves external rotation of the shoulder and elbow flexion in the already abducted and extended arm. Infraspinatus and teres minor provide external rotation and also contribute to glenohumeral stability by drawing the humeral head towards the glenoid fossa. Subscapularis produces its peak electromyographic activity in late cocking when contracting eccentrically to decelerate shoulder external rotation and protect the anterior structures of the shoulder which are under extreme tension at this point. Supraspinatus activity peaks in late cocking as it contributes to stability by drawing the humeral head towards the glenoid preventing translation which could compromise the volume of the subacromial space. There is a significantly greater activity of the supraspinatus muscle in amateurs when tested against professionals, hence fatigue problems are much greater in the amateur. This indicates that teaching of technique has significant potential for treatment. Professional baseball pitchers are more selective, economical and proficient concerning rotator cuff and supraspinatus activities.

Acceleration phase

Activity of serratus anterior is maximal during this late cocking phase along with pectoralis major and latissimus dorsi. Pectoralis major and latissimus dorsi are the two muscles that actively impart velocity to the ball during the acceleration phase of throwing. This phase of throwing is explosive and impels the humerus into rapid internal rotation by means of a concentric contraction. Subscapularis appears to act as a steering muscle to position the head in the glenoid. The acceleration phase includes elbow extension, forearm pronation and wrist flexion.

Follow-through phase

The muscles now contract to decelerate the arm which is adducted, internally rotated and forward flexed at the end of acceleration. Posterior fibres of deltoid, supraspinatus, teres minor and infraspinatus all contract in this phase of throwing. Trapezius and the rhomboids have a high activity in decelerating scapular protraction and the biceps works in decelerating elbow extension and forearm pronation.

Presenting symptoms

Pain

The most common symptom in any sports clinic is pain and in relation to the throwing sports the history must be detailed enough to determine at what phase of the throw the pain occurs as this one fact alone can often lead to a diagnosis and treatment.

Clicking

If this is a painless click then it is probably of no significance and purely a vacuum click alone. If it is a painful click then there may well be a history of direct or indirect mechanical injury and a possible torn intra-articular structure, e.g. labrum.

‘Dead-arm’

The ‘dead-arm’ syndrome is a clearly described clinical entity in which in the acceleration phase of throwing the patient complains of the arm becoming completely useless and dropping down by the side.
sometimes associated with pins and needles but more generally a completely useless arm which can take from a few seconds only to minutes to recover. The ‘dead-arm’ syndrome is due to a momentary subluxation of the glenohumeral joint associated with compression of the brachial plexus. Treatment must hence be directed towards stabilizing the joint by whatever means.

Night pain and ache

This is frequently associated with inflammatory change in the rotator cuff and pain is referred to the deltoid insertion. The patients complain they cannot sleep on the affected side and if they were to inadvertently roll on to that shoulder the pain wakes them up. If it is an ache only preventing them from going to sleep then tendinitis must be suspected. If it is night pain that wakes them up then the possibility of a complete rotator cuff tear must be borne in mind.

Overuse/fatigue tendinitis

The rotator cuff positions the humeral head in the glenoid to provide optimum mobility and stability but is dependent on intact static stabilizers and scapular rotators for this to occur. If the static stabilizers fail to contain the head the rotator cuff must compensate for this. Similarly scapular malposition places the glenoid at a disadvantage and the rotator cuff is over-stretched to complete the throw. Elevated muscle loads lead to early fatigue and inflammatory change. Overuse injuries are most common in the supraspinatus muscle and tendon. This muscle has the greatest role in head depression during shoulder abduction and in a healthy shoulder can adequately maintain the subacromial space preventing impingement (Figure 6). However, in fatigue or overload the shoulder can no longer resist superior translation and intrinsic changes in the tendon may occur (Figure 7).

Infra spinatus tendinitis may also occur and is due to inequality of strength between the internal and external rotators. Infraspinatus and teres minor work mainly to decelerate the internal rotation of the humerus in follow through but may be damaged if they are relatively weak. It is this delicate balance of equal and opposite muscle strength that maintains the asymptomatic shoulder. If one muscle is too strong or another too weak or fatigued then symptoms result.

Impingement

Impingement (Figure 8) is the term used to describe rubbing of the bursal surface of the rotator cuff on the under surface of the coracoacromial arch (acromion, coracoacromial ligament and coracoid (Figure 9)). The mechanism of how this occurs is a matter of some dispute but undoubtedly involves mechanical constitutional and degenerative factors. The importance of these different elements varies with age. In the

Figure 6. Normal balance of deltoid and rotator cuff snugs the humeral head down into the glenoid

Figure 7. Dysfunction of the rotator cuff causes imbalance and upward subluxation of the head with impingement

Figure 8. Impingement test. Elevation causes mid-range pain worse on resistance. Reproduced by kind permission of Churchill Livingstone, from Carter Rowe, ed, The Shoulder
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younger patient (under 35) presenting with symptoms of impingement. It is commonly secondary to an underlying instability. In those over 35 the more common elements are fatigue/degenerative change. If the static restraint (capsule and ligaments) of the shoulder are ineffective the workload of the rotator cuff is increased. This overuse leads to fatigue and dysfunction. The humeral head can no longer be effectively depressed in the glenoid, superior head translation occurs and the subacromial space is reduced. Once this has occurred the cyclical failure of degenerative change within the rotator cuff occurs. During the early stages this is a completely reversible process as inflammatory and oedematous changes are the predominant feature but as this becomes a chronic feature this secondary fibrotic thickening becomes more of a mechanical problem (Figure 10). Interestingly the association of partial thickness rotator cuff tears with impingement cannot be explained on a purely mechanical base alone as these tears are frequently either intrasubstance or on the joint surface side rather than on the expected bursal surface of the cuff. Uhtoff has shown a relative avascularity of the cuff on the articular surface compared with the bursal surface.

Rotator cuff tears

This is most commonly the end result of chronic impingement. It may occur as a one-off mechanical injury. An unsuspected rotator cuff tear may often be the cause of continued symptoms following isolated dislocation of the shoulder. Prolonged overuse leads to fibrosis failure. A tear may occur through an accumulation of microtears. Propagation of a partial thickness tear may progress to a complete tear with additional trauma and tension placed upon it.

Instability

This term is used to describe excessive symptomatic displacement of the humeral head in its relationship to the glenoid fossa and is thought to be the underlying cause of many problems in the throwing shoulder (Figure 11). Stresses during the phase of throwing can cause repeated microtrauma leading to labral changes and stretching of the glenohumeral ligaments. As increases in translation occur due to the above process subluxation can occur. The increased stress placed on the rotator cuff to control the position of the humeral head leads to eccentric overuse and damage. With inadequate static stabilization and rotator cuff dysfunction the humeral head can translate superiorly causing impingement. Posterior instability occurs commonly in throwers during follow through when the posterior capsule and labrum are stretched. The posterior labrum may be abraded and the capsule restraints are stretched which permits posterior translation of the humeral head causing stretching and fatigue in the rotator cuff. This may lead to overuse tendinitis of the infraspinatus but in general posterior instability and subluxation are usually a problem that is well tolerated by the athlete and may be remedied by appropriate rehabilitation.

Impingement versus subluxation

As discussed above the history and age are important factors in delineating the two, however, specific tests may be helpful.

If the patient lies supine with the arm abducted to 90° and externally rotated (Figure 12), this does not usually cause pain with the primary impingement.
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However, if the impingement is secondary to an instability, abduction and external rotation may induce pain and more importantly can be relieved by applying pressure anterior to the joint to attempt to sublux the humeral head within the glenoid posteriorly. The mechanism of this is thought to be an over-lax anterior capsule with a relatively tight posterior capsule which allows the forward subluxation in abduction/external rotation, e.g. the young tennis player after repetitive serving. If there is any doubt as to the origin of the site of the pain local anaesthetic may be injected into the subacromial bursa which should relieve pain from both primary and secondary impingement.

**Treatment**

As has been discussed, the two main problems in the throwing athlete appear to be a combination of impingement and instability. In the older athlete true impingement may be present but in the younger patient impingement secondary to instability must be suspected. From the history and examination these two components must be delineated. If impingement is thought to be the primary cause of the problem then this must be staged according to the usual Neer classification. In the early stages, rest and anti-inflammatory treatment alone may suffice. In longstanding cases steroid injection and non-steroidal anti-inflammatory agents may be helpful. The usual recommended regimen for this is never more than three injections at 3-weekly intervals and rarely to be used in the young. The steroid injection is helpful diagnostically and also prognostically. The effect of the steroid is to reduce inflammation and hence thickening due to oedema. If the steroid is mixed with local anaesthetic and injected into the subacromial bursa (Figure 13) then three reactions may occur.

1. Complete resolution of symptoms but return of symptoms after a few hours, i.e. the time taken for the local anaesthetic to wear off. This confirms that the diagnosis of the site of pain is correct but that probably the secondary inflammatory changes are

![Figure 11. Stability of the humeral head in the glenoid can be likened to a seal balancing a ball. Reproduced by kind permission of Churchill Livingstone, from Carter Rowe, ed, The Shoulder](image1)

![Figure 12. Relocation test. Pain on forced external rotation in abduction — relieved by posteriorly directed force suggests impingement secondary to instability. Reproduced by kind permission of Smith and Nephew Dyonics, Cambridge, UK](image2)

![Figure 13. Painful arc can be relieved by local anaesthetic injection. Reproduced by kind permission of Churchill Livingstone, from Carter Rowe, ed, The Shoulder](image3)
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only a minimal part of the symptoms and that the secondary fibrotic changes are probably predominant. It is therefore probably useless to give any further injections.

2. Resolution of symptoms which lasts for a variable period of time, e.g. a few weeks, but then the symptoms return. One can conclude from this that inflammatory change is at least part of the problem and hence is reversible. A further injection would certainly be worthwhile with hopefully further prolonged symptomatic relief. If, after the second injection, the symptoms return after a similar time period then it is probably pointless going on to a third injection. However, if the second injection brings prolongation of relief then a third injection may well be indicated and may induce permanent relief of symptoms. However, if injection therapy has only a temporary benefit then if circumstances dictate this may be the group that would benefit from surgical decompression. This may now be done arthroscopically with minimal perioperative morbidity and equal success to open procedures (Figure 14a,b).

3. If injection induces complete resolution of symptoms then obviously no further treatment is necessary.

If instability is thought to be the prime cause of symptoms the exact degree and direction of instability must be elicited by examining the phase of throwing and stress testing. The weak and fatiguing muscles must be strengthened, the posterior capsule should be stretched and that particular athlete’s throwing technique carefully monitored with appropriate coaching. It may take a year to reverse bad habits in capsular stretching.

However, if all these measures fail then surgical stabilization may well be considered. Capsular shift procedures correctly indicated are reliable and need not limit the range of motion. Arthroscopy enables subtle changes to be seen which confirm the clinical

Figure 14. a Three portal arthroscopic subacromial decompression - shaver, scope, drain; b Arthroscopic subacromial decompression - the anterior and inferior one-third of acromion is removed with a shaver. Reproduced by kind permission of Smith and Nephew Dyonics, Cambridge, UK

Figure 15. SLAP lesions. Superior anterior/posterior labral tears – worse on follow through

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diagnosis of instability. Lamination of the antero-
inferior capsule indicate overstretching and in con-
junction with chondromalacia seen on the poster-
superior humeral head is diagnostic of anteroinferior
subluxation. Arthroscopy has been enormously help-
ful in recognizing previously unrecognized problems,
e.g. superior labral anterior and posterior tears (slap
lesions described by Snyder et al.\(^6\)). These occur in
the follow-through phase of throwing when the
biceps tendon is avulsed from the superior glenoid
taking with it either part of the anterior or posterior
labrum. The mechanism of this has been confirmed
by Andrews who has electrically stimulated the
biceps in cadavers and reproduced this lesion\(^6\).

Arthroscopic methods of shoulder stabilization are
not yet as reliable as open methods but hold promise
for the future.

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