# Rotator cuff tears: pathology and non-surgical management

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## Abstract

Rotator cuff tears may be caused by intrinsic (biochemical) or extrinsic (physical) mechanisms, or a combination. They may also be classified as traumatic (acute) or non-traumatic (sub-acute). Due to an on-going lack of literature on natural history and disease progression, a variety of treatment approaches to manage these tears exists and consequently consensus views can fluctuate. This article will focus on the anatomy of the rotator cuff and the pathology that may affect it, before discussing the non-operative approaches commonly used. A potential algorithm for management is proposed, highlighting the timings to consider for surgical intervention.

**Keywords** conservative management; corticosteroid injection; pathology; physiotherapy; rotator cuff disease

## Introduction

Rotator cuff tears are a common condition seen in primary and secondary care. Management of this condition varies greatly between clinicians of differing specialities and even varies among surgeons. Due to incomplete scientific evidence, decisions are made based on existing knowledge and personal experience. In this review we aim to highlight some of the non-surgical options for the management of rotator cuff tears.

## Anatomy of the rotator cuff

The rotator cuff[1] is made up of four muscles whose principal functions are to stabilize the humeral head in the glenoid, as well as contributing to movement at the glenohumeral joint.

Four layers surround the glenohumeral joint. The most superficial layer consists of the deltoid and pectoralis muscles. The second layer is formed by a number of non-muscular structures: the clavipectoral fascia, which extends from the undersurface of the clavicle, surrounds pectoralis minor and attaches to the axillary fascia; the conjoined tendon of the short head of the biceps and coracobrachialis; and the coraco-acromial ligament extending from the lateral coracoid to the lateral acromion. The third layer consists of the rotator cuff, discussed in more detail below, whilst the deepest layer is the glenohumeral joint capsule and the 'ligaments' this structure forms.

The rotator cuff consists of four muscles: supraspinatus, infraspinatus, subscapularis and teres minor. Each muscle of the rotator cuff originates from the scapula and has a tendon that attaches to one of the tuberosities of the humeral head. The tendons together form an incomplete cuff around the humeral head. It is incomplete, as the tendons are required to separate around two anatomical structures: the coracoid and the long head of the biceps tendon.

The insertion and orientation of the rotator cuff tendons onto the proximal humerus is of prime importance when conceptualizing rotator cuff tears.

For this description, we will use the clock-face analogy of rotator cuff insertion points, looking from the lateral side of the left shoulder with the arm in the anatomical position (Figure 1). The area from 11 o'clock to 5 o'clock can be seen to have a confluent attachment, along the superior and posterior length of the greater tuberosity. From 7 o'clock to 10 o'clock subscapularis attaches onto the lesser tuberosity, although this attachment may be obscured by the overlying biceps tendon, which takes a 'question mark' trajectory as it enters the glenohumeral joint. This pathway is itself covered by the transverse humeral ligament. The space between 10 o'clock and 11 o'clock is present due to the fact the muscular bellies of subscapularis and supraspinatus are separated by the coracoid process. This area, the rotator interval, is occupied not only by the long head biceps, but also by the anterior shoulder capsule inferiorly and the coracohumeral ligament superiorly. The coracohumeral ligament originates from the lateral coracoid, just below the coraco-acromial ligament, coalesces with the anterior capsule and inserts as the most anterior structure on the greater tuberosity. This structure also extends posteriorly, running perpendicular to the fibres of supraspinatus and thus reinforcing the supraspinatus tendon. This is visible to the arthroscopist as a curved cable running anterior to posterior along the joint side of the supraspinatus.

The infraspinatus and teres minor muscles can be observed to join each other near their insertion and are therefore often identified together and called the posterior rotator cuff.[1]

## Pathology

Rotator cuff disorders are the commonest cause of pain and loss of function around the shoulder. Causes may be split into either traumatic or non-traumatic, and intrinsic or extrinsic.

Traumatic tears are most often secondary to either shoulder dislocations or a fall. As a consequence, any patient over 45 years old, with loss of shoulder function after a shoulder dislocation or who has had more than 4 weeks of shoulder dysfunction after a shoulder injury should undergo MRI or ultrasound imaging to investigate for a possible acute rotator cuff tear.[2]

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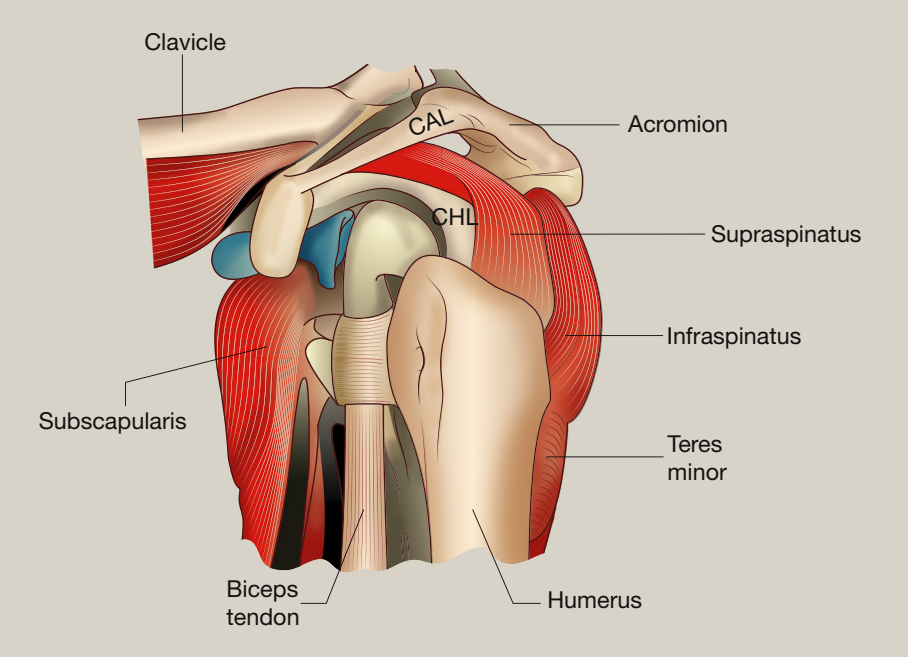


**Figure 1** Insertion of the rotator cuff muscles.[1]

Intrinsic causes relate to those affecting the integrity of the tendons themselves, including age, diabetes, inflammatory conditions and more rarely connective tissue disorders such as Marfan's syndrome.

Extrinsic causes relate to damage to the rotator cuff tendons secondary to factors external to the tendon. The effect of this is impingement of the tendons. Impingement may be caused by anything that reduces the relative space available for the tendons to move under the coraco-acromial space. This may be due to repetitive friction of the tendons leading to an inflammatory reaction, osteophytes on the acromio-clavicular joint or calcification of the coroco-acromial ligament forming subacromial spurs, as well as inflammatory conditions. The morphology of the acromion has also been shown to have an effect, individuals with type III (hooked) acromions having a higher risk of impingement.

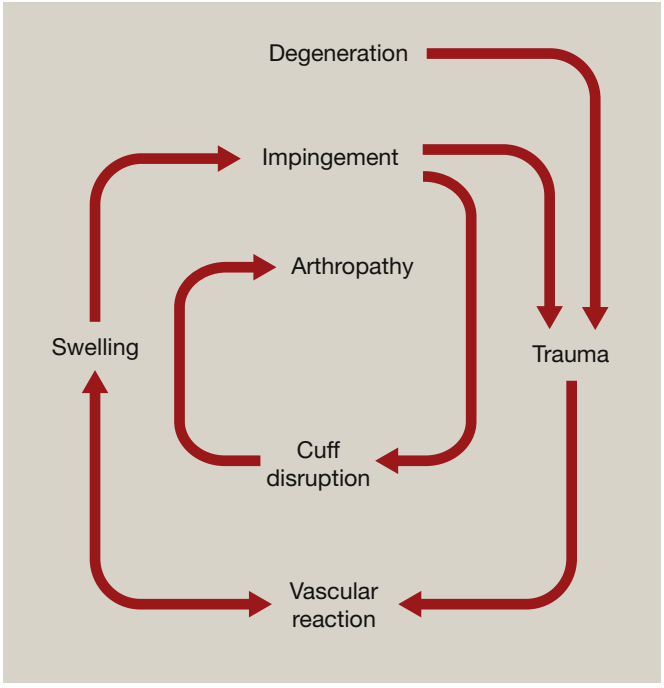


**Figure 2** The spiral of rotator cuff lesions. Reproduced from ref. [4] with permission from Taylor and Francis.

Impingement often leads to pain and subsequent avoidance of any precipitating movements, which would ordinarily help promote tendon repair. With repetitive insults or impaired healing however, partial tears can develop in the rotator cuff (supraspinatus), which may progress to full thickness tears and in time larger tears, and in some patients subsequent arthritis (rotator cuff arthropathy) (Figure 2). These non-traumatic tears usually start in the supraspinatus before extending into infraspinatus and teres minor muscles. Population studies have demonstrated that more than a quarter of rotator cuff tears are asymptomatic,[3] it is unknown how many of these become symptomatic and which shoulders will progress to rotator cuff arthropathy.

Rotator cuff arthropathy is the end stage problem for patients with chronic rotator cuff tears that get larger over time, where rotator cuff insufficiency leads to superior migration of the humeral head, altered biomechanics of the glenohumeral joint, and subsequent arthropathy.

## Principles of treatment

Generally, symptomatic acute tears are urgent problems that are treated with early surgery, especially in the young patient.[1] There is variation in surgical decision-making for patients with chronic rotator cuff tears, especially around when to intervene with surgery. However most surgeons would advocate at least some non-operative management first. There is no agreed algorithm amongst the orthopaedic community for the management of rotator cuff tears, and whilst each patient case must be considered individually, we have suggested a potential algorithm to


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