

Shoulder Pain

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Rotator Cuff Tendinopathy

One in three people will experience shoulder pain at some stage. For people aged over 65 years, it is the most common musculoskeletal problem. The primary cause of shoulder pain is thought to be rotator cuff “impingement”. Traditionally, this has been attributed to extrinsic pressure on the cuff. It has been proposed that impingement occurs from above, with the coraco-acromial arch exerting downward pressure on the tendons and bursa. In

support of this theory, it has been shown that people with chronic rotator cuff disease and tears are more likely to have a type II (curved) or type III (hooked) acromion. It has been assumed that this anatomical variation makes the cuff more vulnerable. The concept of extrinsic cuff disease has also been the

reason behind sub-acromial decompression surgery, particularly acromioplasty, and division of the coraco-acromial ligament.

There is growing evidence that this theory of extrinsic impingement is fundamentally flawed. Indications are that rotator cuff disease is, in the main, an intrinsic disease, starting within the tendon.

A 1997 arthroscopic investigation showed that 91% of rotator cuff tears in overhead athletes were articular (undersided) tears. A 1990 review of six separate studies found an

average of 76% of tears were articular sided. In a 1987 cadaver study, 82% of tears were either articular or intra-substance. A separate cadaver study found no correlation between tears and anatomical bony variations.

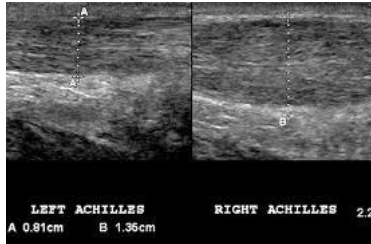
The tendinopathy model

There is good evidence that rotator cuff disease represents another type of tendinopathy, like common ones found in other parts of the body – extensor origin of the elbow, hamstring origin, patellar tendon and insertional Achilles tendon. In all of these cases it has been shown that the disease starts on the undersurface of the tendon – the surface closest to the bone. Expert opinion is that tendinopathy is not a condition of tensile overload, but of bone on tendon compression. Changes in chronic tendinopathy indicate adaptation of collagen to a fibrocartilage structure, suggesting an attempt by the tissues to adapt to compressive overload. Fibrocartilage is less capable than tendon of withstanding torsional loads. Such a structure will be vulnerable to tearing when a large or repetitive tensile load is applied. In one shoulder study, articular sided fibres were found to rupture with half the applied load compared to bursal-sided fibres.

The pathological changes found in diseased tendon in other parts of the body have been found in diseased rotator cuff. It is known that there are various stages of tendon pathology. There may be an acute reactive tendinopathy, when a sudden or



repetitive load is applied. This may occur in an overhead athlete who does an excessive amount of throwing, or in a middle-aged person who does an unaccustomed period of overhead work. Continued overload will lead to damage and eventually to chronic changes. Both acute and chronic tendon disease can lead to swelling, brought about by an increase in tendon water content. This is due to a proliferation of tendon proteins – proteoglycans, which attract water (the image on right shows a swollen Achilles tendon).



This could be the reason for the eventual impingement with superior structures. The cause in this case is the tendons pushing up, rather than the bone pushing down. There is also good evidence that a type II or III acromion may develop as a result of degeneration or tendon disease, & not be the cause. A 2002 study found no association between acromial shape & rotator cuff pathology in people over 50 years.



The researchers felt the type III acromion represented a degenerative process rather than a morphological variation. There is no evidence that people are born with these anomalies.

It is also known that bony changes can occur over a short space of time in response to stress. A good example is Osgood Schlatter lesion of the knee.

While acromioplasty is still a common procedure, a 2009 study has shown it to have equal or inferior results to bursectomy alone. Several studies have shown conservative management to be equal or better than acromioplasty at 6, 12 or 48 months. There have also been studies that have shown that acromioplasty is associated with increased antero-superior translation of the humeral head. This may lead to subsequent stress on the cuff and other pain sensitive tissues, including the bursa and coraco-acromial ligament.

Cause of pain

As with other types of tendinopathy, why rotator cuff tendons become painful is not clearly understood. Some authors have suggested that the presence of neo-vessels, often found in diseased tendons, may introduce pain nerve endings to the

normally aneural tendon. However this is not widely accepted. It is also known that the pressure associated with tendon swelling can lead to pain. What is interesting is that up to 50% of all partial & full-thickness rotator cuff tears are completely symptom free.

The bursa is a potent source of symptoms. It may become involved secondary to tendon disease. In particular, when the rotator cuff fails in its function to maintain depression of the humeral head, bursal irritation can occur between the tendon and overlying arch. Bursal swelling, the presence of neovessels, and inflammatory substances such as cytokines & substance P, have all been identified within the subacromial bursa. There is some evidence that bursal inflammation may be associated with rest and night pain, while pain due to the tendon may be more apparent on movement.

The coracoacromial ligament and presumably the bone may also be a source of pain. Stress on these structures may arise due to either increased tendon volume and thickness, or superior migration of the humeral head secondary to muscle imbalance, rotator cuff disease, or acromioplasty.



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