THE ROTATOR CUFF

Bibhas Roy, Yogesh Joshi

INTRODUCTION:

The annual prevalence and incidence of people consulting for a shoulder condition is about 2.4% in the UK [1]. Shoulder pain in the population is also a longterm disabling symptom with about 50% complaining of symptoms 3 years after onset[2]. Common conditions that can result in chronic shoulder pain include rotator cuff disorders, frozen shoulder, shoulder instability, and shoulder arthritis. Between 30% and 70% [3]of such shoulder pain is said to be due to disorders of the rotator cuff. Mixed disorders are however, common,[3] and this needs to be remembered during a dignostic exercise.

No discussion of the rotator cuff should take place without mentioning E. Amory Codman. In 1934, at his publisher's expense, Codman published his famous book on the shoulder [4]. The foreword to the second reprint of this book states 'detailed anatomic description of the shoulder and the many clinical entities which affect it has undoubtedly been the greatest gift Codman has made to the surgeons of this world. He stressed chiefly the importance of making an early diagnosis of rupture of the supraspinatus tendon. He pointed out in his own way that only 100 neglected cases of this lesion might cost more than the gross income of the average doctor in a lifetime.' We have little evidence of the burden this particular problem has on society, but there is evidence that problems of the rotator cuff are expensive healthcare problems with a mean cost of $14000 per case in the US[5]. In spite of the problem being very common, as yet we do not fully understand the pathogenesis and natural history of tears of the rotator cuff, and do not always know when to operate and when we do operate, which operation to perform[6].

ANATOMY:

Rotator cuff is a dynamic stabiliser of the shoulder joint and contributes in all shoulder movements. The muscles of the rotator cuff are the supraspinatus, infraspinatus, teres minor and the subscapularis. The tendons of these muscles coalesce together to form the rotator cuff. The tendons at the level of the shoulder joint are inseparable except for the subscapularis, which is attached to the rest through the rotator interval.

They form force couples in coronal as well as transverse plane and leads to a concavity compression of the humeral head in the shoulder joints. During abduction, the joint reaction force is directed towards the concavity of the glenoid, which improves the stability of the joint as the rotator cuff compresses the humeral head to oppose the superior pull of the deltoid[7]. The joint retains its stability as long as force couples in between the subscapularis and the infraspinatus is maintained. The fine muscle balance helps the centre of rotation of the shoulder joint to remain constant. Thus, conditions characterised by either deltoid or supraspinatus dysfunction may result in abnormal loading mechanics at the glenohumeral joint [8].

In addition to the rotator cuff the static stabilisers like the coraco-acromial arch and the long head of biceps tendon have been implicated in preventing the superior migration of the humeral head in absence of rotator cuff function[9, 10].

The rotator cuff insertion has been further studied and is made up of five layers. Layer one consists of the fibres of the coraco-humeral ligament. Layer two is made up of the tendon fibres, which are uniformly oriented. Layers three consist of the thick tendon fascicles but are not uniformly oriented. Layer four is made of thick bands of collagen tissue that are directed perpendicular to the tendinous orientation. It is also called the pericapsular band, rotator cable or the transverse band [11]. Layer four is the capsular layer where the fibres are randomly oriented.

Because of the altered fibre orientation in the tendon itself and the different layers significant shear forces exist and may have a role in rotator cuff tear. The major arterial supply to the rotator cuff is derived from the ascending branch of the anterior humeral circumflex artery, the acromial branch of the thoracoacromial artery, as well as the suprascapular and posterior humeral circumflex arteries[12].

Recent studies, however, have shown that in symptomatic patients of impingement syndrome there is neovascularisation at the critical zone of the rotator cuff secondary to mechanical compression[12]. There is an intact vascularisation of the intact rotator cuff even in the critical zone[13]. It has been also observed that the tissue adjacent to the tear (<2.5mm) is viable in terms of vasculature histologically[14]. Vascular studies have not been consistent in their findings, and hyperaemic response at the edge of a tear has also been seen[15].

AETIOLOGY:

The rotator cuff problems range from impingement to rotator cuff damage of variable severity. Neer proposed a continuum from chronic bursitis, to partial tears, to complete tears and then to massive tears[16]. The evidence is mounting that such progress depends on many factors i.e. extrinsic morphology causing impingement, intrinsic factors within the tendon as well as genetic factors[6]. While the sequence of events remain unknown, it is becoming clearer that tissue viability may have a role in planning treatment. It is uncertain if it is possible to predict the progress through these stages in particular patients.

The extrinsic theory to the causation of rotator cuff tears indicates that it could be the subacromial side (external impingement) caused by acromial spur or the coracoacromial ligament. Neer believed that the rotator cuff pathology was always secondary to the mechanical compression caused by the anterior acromion of the osteophytes of the acromio-clavicular joint[17]. Moreover the anatomical morphology correlated with the disease process (13, 14). This proposed a progression from chronic bursitis to partial tears to complete tears and then to massive tears.

In an animal model Mehta et al demonstrated that the strain was equal on the bursal side as well as the articular side of the rotator cuff but the stress concentration was more at the critical zone in the subscapularis muscle which were potentiating the articular, bursal and intratendinous tears. He demonstrated that the articular side tears were more common (17).

The intrinsic theory, on the other hand, is because of the changes in the cuff itself leading to degenerative tears. Vascularity abnormalities are often attributed to the “critical zone” [4]. This zone is present when the head of the humerus occludes vascularity of the supraspinatus tendon during shoulder movements. Studies have demonstrated a zone of hypovascularity at the level of critical zone of supraspinatus tendon, which is around 8 mm proximal to its insertion in the humerus[13], but it is not known whether the presumed hypoperfusion within this area results in failure of the cuff. Rotator cuff degeneration is often the combination of factors. This degeneration initially develops in the supraspinatus tendon near its anterior insertion. Supraspinatus tears tend to cascade posteriorly into the infraspinatus then anteriorly across the bicepital groove into the subscapularis. This“cascading effect” increases the load on neighboring, unaffected fibres[18].

Internal impingement is a different entity altogether where impingement occurs when the cuff is pinched between the humeral head and the posterosuperior labrum whilst the arm is rotated in extreme abduction, and externally rotated as seen in the quarterback position in overhead athletes[19, 20]. Varying degrees of glenohumeral instability, posterior capsular contracture, and scapular dyskinesis may play a role in the development of symptomatic internal impingement. This leads to tear over the articular side of the posterior rotator cuff. This condition is far from clear with Burkhart suggesting that internal impingement is not pathologic; it is a natural restraint to hyperexternal rotation, in fact the loss of internal impingement is pathologic leading to "dead arm" of the throwing athlete that may be caused by superior labrum anterior and posterior lesions or by scapular dyskinesis[21]. It is worth remembering, however that that all shoulders exhibit internal impingement in the fully abducted and externally rotated position and that therefore internal impingement should ordinarily not be considered pathologic[22].

NATURAL HISTORY:

MRI and ultrasound studies have shown an overall prevalence of the full thickness rotator cuff tear is from 15% to 23%, 4-13% of the people below 59 years and 28-51% of the people between 60 and 80 years of age [23, 24]. A cadaveric study determined the incidence to be 5% to 30% (20). Although the incidence is known there is very little understanding about the symptoms associated with the extent of the size of the tear, which may range from patients having full range of movement to having considerable limitation and even pseudo-paralysis of the shoulder. There is strong correlation of tears associated with increasing age, which was demonstrated by a longitudinal study using ultrasound techniques. Although some tears progress from asymptomatic to symptomatic, this cannot be accurately predicted[25].

DIAGNOSIS

CLINICAL

HISTORY

Causes of shoulder pain should be thought of in terms of pain from the shoulder itself, and pain from the surrounding anatomical structures. These are summarized in the table

Shoulder	Degenerative Arthritis of glenohumeral joint	Inflammatory
		Osteoarthritis
		Post Traumatic
	Frozen shoulder	
	Subacromial Impingement	
	Acromio clavicular joint degeneration	
	Instability	
	Rotator cuff tears	
	Calcific tendonitis	
	Infection	
	Trauma, i.e. fractures, dislocation	
Referred Pain	Neck and C spine	
	Myocardial iscaemia	
	Intra-abdominal pain - subdiaphragmatic	
	Malignancy – lung apex	
	Generalized conditions i.e. polymyalgia, Myalgic Encephalopathy	

Table 1 : A list of common causes of painful shoulder is presented. Whilst not comprehensive, history should focus on these and will diagnose the problem in the majority.

EXAMINATION

The shoulder possibly has more described tests for it than any other area of the body. 112 tests have been listed[26] in a series.

The common ones for the rotator cuff are listed below

Test	Pathophysiology	Perform
Hawkins-Kennedy Test	Traps the rotator cuff between the greater tuberosity and the coraco-acromial arch	With both shoulder and elbow at 90^{o} flexion, the arm is stabilized and moved into internal rotation causing pain
Neer Impingement sign	Elevation of the arm causes critical areas to pass under the coraco-acromialligament or anterior acromion. Internal rotation increases the sensitivity.	The examiner performs maximal passive forward flexion with internal rotation whilst stabilising the scapula.
Neer Impingement test	Local anesthetic abolishes pain	Subacromial injection of local anaesthetic, about 10 mls of bupivacaine (0.5%) commonly used
Empty Can Test[27]	test integrity of the supraspinatus tendon, minimize deltoid contribution by elevating in scapular plane and in internal rotation	Patient resists downward pressure exerted by examiner at patients elbow
Full Can Test	45^{o} external rotation, otherwise as above	Less pain inhibition
Codman's Sign[4]	the arm can be passively abducted without pain, but when support of the arm is removed and the deltoid contracts suddenly, causing pain	Passive elevation, then ask patient to hold limb abducted

For diagnosing tendinopathies with or without tears, active unresisted external rotation for the infraspinatus and the lift off test for the subscapularis were specific but lacked sensitivity.[28]

INVESTIGATIONS:

X rays

These should include AP, axillary as well as supraspinatus outlet views. The AP view will show the most common abnormaliites of the shoulder, but the other views are required to fully appreciate the skeletal morphology.

Figure 1 : Supraspinatus outlet view, demonstrating an acromial spur

Figure 2 - supraspinatus and subscapularis calcification. Subscapularis calcification is rare but well described [29]

The outlet view is perhaps the most useful for problems in and around the bursa, although controversy exists in classifying the different types[30]. The supraspinatus outlet view (Figure 1) reveals acromial morphologic traits, acromial slope, subacromial excrescences, and inferior osteophytes at the acromioclavicular joint (Figure 3) [31]

Figure 3 : ACJ Degeneration with osteophytes on the lateral clavicle

. The axillary view best demonstrates the glenohumeral relationship and has a role in assessing glenoid wear in degenerative disease.

As the type of tear and the size of tear vary in individual patients, investigations aid in defining the anatomy of the lesion aiding the surgeon in his decision.

Ultrasonography has been shown to have high sensitivity and specificity to full thickness tears. When an investigator has comparable experience with both imaging tests, the decision regarding which test to perform for rotator cuff assessment does not need to be based on accuracy concerns[32]. Recent work points to this being as good as if not better than MRI for cuff problems [33]. The advantage is that of a dynamic investigation which can correlate imaging with patients symptoms. Many shoulder surgeons are now performing the ultrasound examinations in their clinic on their own [34]

MRI has been regarded as a procedure of choice in for shoulder imaging technique and has been extensively studied (sensitivity 95% to 100% and specificity 88% to 95%) in diagnosis of rotator cuff tear[35-37] MR imaging helps in evaluating the muscle belly of the rotator cuff especially for the evidence of fatty infiltration which may suggest a chronic tear or a massive tear suggesting that their repair may not result in optimal functional recovery[38].

MR (as well as CT) arthrograms have been used for cuff problems, this has the additional benefit of forcing the contrast agent into small tears. but the introduction of 3.0 Tesla MRI scanners, which have very high specificity and sensitivity in diagnosing partial and full thickness tears, may not justify their use[39, 40].

TREATMENT:

Treatment of rotator cuff problems need three elements to be considered

Structural	Acromion morphology, osteophytes etc
Biological	Inflammation
Functional	Movement pattern disorders

Table 2 – Treatment of rotator cuff problems are directed at three aspects

NONINVASIVE THERAPY

Treatment should begin with noninvasive therapy. This should commence with physiotherapy to assess and treat posture and muscle imbalances. It is important to remember that especially in the young, the impingement pain is usually caused by muscle imbalance around the shoulder girdle or by an underlying instability. The common pattern of a protracted scapula with overactive pectorals, which narrows the subacromial space [41] needs to be identified, as this will also lead to a significant reduction of cuff strength [42, 43]. Weak external rotators, and tight and overactive internal rotators are responsible for suboptimal function of the supraspinatus as well and need correction.

Prior to actual rotation of the shoulder joint, normal recruitment of the rotator cuff and biceps is characterized by a non-specific presetting phase which is mainly directed at enhancing the joint 'stiffness' and hence its stability. Once movement is in progress, the EMG patterns of these muscles become movement specific and are correlated with the resultant moment [44].

There are patterns of activation of muscles around the scapulohumeral articulation which are specific for certain activities[45]. These need to be recognized by the physiotherapists and the pattern needs to be optimized, i.e. ensuring that the scapular stabilisers activate before the rotator cuff. The throwing shoulder poses a specific challenge in this regard[22, 46, 47]. Clinicians treating this subgroup of patients need to be familiar with the work done by Stephen Burkhart, as referenced above.

Techniques used to achieve these goals are based on regular a regular exercise programme aiming to increase range of movement, correcting any movement pattern disorder, ensuring good core stability. Modalities such as EMG biofeedback[48], may well be useful as well. Ultrasound for short term pain relief may be appropriate but there is insufficient evidence for its longterm use.

INVASIVE TREATMENT

INJECTIONS

Injections of corticosteroids are simple office treatments for painful shoulders. Corticosteroids are potent anti-inflammatory and pain modulating drugs and may act through both local and systemic mechanisms. They may be effectively used with diagnostic tests. However, Insufficient evidence exists for the efficacy of corticosteroid injections in rotator cuff disease of the shoulder. Studies have shown that corticosteroid injections can be superior to physiotherapy in terms of the success of treatment; improvement in degree of lateral rotation; improvement in clinical severity; and in relief of the main complaint, pain, and disability[49]. There is evidence to suggest that steroid injections are effective in the short term but these are thought to be ineffective in the long term [50]. There is however, little agreement in the amounts injected [51] and also the injection route used. Other important issues that remain to be clarified include whether the accuracy of needle placement, anatomical site, frequency, dose and type of corticosteroid influences efficacy.

Although subacromial injections appear straightforward, more recent cadaveric, radiographic, and clinical studies have demonstrated variable accuracy rates [52]. The accuracy is, at best about 70%, in expert hands, which implies 30% of these injections are being administered in the surrounding tissues including the rotator cuff. The risk of infection, a rare but debilitating complication, needs to be taken into account. Corticosteroids may also adversely affect the collagen molecules in tendon and atricular cartilage. This may precipitate rotator cuff tears, by damaging the collagen structure and inhibiting a repair response.

Hyaluronan injections have recently been tried instead of steroids, to try and minimize the risks. Early results appear promising, but more widespread use will determine the efficacy of this agent in subacromial impingement.

Considerable placebo effects have also been seen with injection treatment, acupuncture, etc, and contradictory findings have been reported in studies regarding the effectiveness of steroids [53]. There is also evidence that systemic steroids and subacromial steroid have a similar efficacy [53].

Needling of calcific tendonitis is also an effective way of breaking up the calcium, and encouraging its resorption. Steroid injections may be counterproductive in this situation [54].

SURGERY

	Factor	Effect
History	Night pain	
	Problems with activities of daily living	
	Problems with work	
	Duration of symtoms	>6 months
	Patient expectations	
	Result of non operative therapy	
	General health	
Pathology	Morphological abnormality, i.e acromial spur	Consider early surgery
	Full thickness tear	Early surgery
	Partial thickness tear	Surgery often required – refer for opinion
	Movement pattern problem	Surgery may be ineffective in isolation
Others	Referred pains	Surgery may be ineffective
	Disability insurance patients	May bias surgical results

Table 3 : Factors to consider before advising surgery for rotator cuff problems

Surgery has a clear role in treatment of rotator cuff problems. For impingement problems, a subacromial decompression should be considered if there is no response to conservative management. This is a simple daycase procedure with consistent good results reported in the literature. It leads to minimal scarring, and rapid return to activities. In associated AC joint degeneration, this is often combined with an AC joint excision.

There is evidence that surgery for operative repair of rotator cuff tears has consistently shown a higher rate of pain relief in patients (85%) and a better return of strength. Non-operative treatment is expected to produce satisfactory relief of pain in only 50% of patients and no improvement in strength at long-term follow-up [55]. Impingement and AC joint problems also benefit from surgery.

The fact that patients try to get benefit from social insurance based on sickness disability significantly biased the outcome after ASD[56]

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