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(i) Degenerative rotator cuff disease and impingement

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Abstract

Impingement and tears of the rotator cuff are common. 2.5% of the population of the United Kingdom seek advice for a shoulder complaint each year. 70% of these referrals are due to rotator cuff related pathology.

Codman and Neer both initially postulated that impingement and tendinopathy were due to extrinsic factors with the antero-lateral acromion 'impinging' on the superior surface of the rotator cuff. This view has subsequently been modified and the general consensus now agrees that it is a multifactorial condition with both extrinsic, intrinsic and environmental factors all playing a role.

A clear history and examination findings are crucial to exclude subtle causes of impingement that are atypical, e.g. instability. This review clearly describes the general shoulder examination and specific tests. The benefits and disadvantages of ultrasound and magnetic resonance imaging are discussed in the diagnosis of rotator cuff pathology.

The treatment options for proven impingement vary from analgesia and physiotherapy, injection therapy to surgical intervention, both open and arthroscopic. The treatment needs to be tailored to the individual patient and is often dictated by the initial response to injections and radiological findings as well as examination findings.

Outcomes of arthroscopic subacromial decompression are favourable in most studies and evidence does suggest that the surgery reduces the incidence of rotator cuff tears when compared to an unoperated cohort. There are however contradictory studies claiming physiotherapy is comparable to subacromial decompression.

Keywords impingement; injection; rotator cuff; shoulder

Introduction

The shoulder has the greatest range of motion of any joint in the human body. Whilst this affords many benefits, such as the ability to accurately place and manipulate the hand in a vast space, it does come at a cost. The shoulder is inherently unstable and requires bony, muscle and ligamentous support to remain congruent.

Humans have evolved to undertake many overhead activities in everyday life, in the workplace and in the sporting arena. The relatively short lever arm of the shoulder muscles acting on the significantly long lever arm of the upper limb, often with extra load in the hand, leads to very high loads through the tendons and large reaction forces across the joint surfaces.

Shoulder pain is common. One in 40 of the United Kingdom population seeks advice from primary care services each year for new and recurrent shoulder complaints.[1] The majority of these presentations, and up to 70% of all referrals to shoulder clinics, are related to rotator cuff pathology.[2]

Primary impingement of the rotator cuff tendons was historically thought to be due to external factors, such as the prominence of the anterior-inferior acromion and the development of bony spurs extending into the coraco-acromial ligament, particularly anterior degenerative spurs. Clinical and basic science research over the years has now led to the widely held consensus that rotator cuff tendinopathy is a multifactorial pathology with intrinsic, extrinsic and environmental factors all contributing to the development of the condition.

Anatomy

The rotator cuff is essential for normal function of the glenohumeral joint. The function of the rotator cuff is twofold, to rotate the glenohumeral joint and to centre the humeral head within the glenoid to allow the efficient function of other muscle groups, for example the deltoid.

The 'dynamic' rotator cuff comprises four muscles, and its function in stabilizing the shoulder is complemented by the static structures, including the labrum, acromion, coracoid and coraco-acromial ligament.

The supraspinatus (SSP) muscle arises from the supraspinous fossa of the scapula. It is a fusiform muscle, passing anterolaterally under the acromion, where it inserts into the 'footprint' of the superior greater tuberosity over an area of approximately 23 × 16 mm.[3] In one-third of people the SSP tendon also partly inserts into the lesser tuberosity.[4] It is innervated by the suprascapular nerve (C5 and C6) having arisen from the upper trunk of the brachial plexus and passing below the transverse scapular ligament in the suprascapular notch. This is an area of potential compression of the nerve. The function of the supraspinatus is reported as the initiation of forward elevation and abduction over the first 15°. It is also vital in stabilizing the humeral head in the glenoid. This function is demonstrated in large and massive cuff tears, where the humeral head begins to sublux superiorly under the action of the deltoid.

The infraspinatus (ISP) arises from the infraspinous fossa and passes laterally inserting into the postero-superior aspect of the greater tuberosity into a footprint of approx. 29 × 19 mm^3. It is also supplied by the suprascapular nerve (C5 and C6). The main function of the infraspinatus muscle is external rotation of the glenohumeral joint along with teres minor, and again, stabilizing the humeral head.

Teres minor (TM) originates from the upper two-thirds of the dorsal, axillary border of the scapula. It passes laterally just inferior to the lower border of infraspinatus. It inserts into the inferior aspect of the greater tuberosity and is the second of only two external rotators of the glenohumeral joint. It is innervated by the axillary nerve (C5 and C6).

The subscapularis (SSC) muscle is a large triangular muscle that arises from the subscapular fossa. It passes laterally and

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anteriorly to the capsule of the shoulder joint, with which it blends, and inserts into the lesser tuberosity over an area of 40 × 20 mm^3. It is innervated by the upper and lower subscapular nerves (C5 and C6 respectively). The function of the subscapularis is internal rotation of the glenohumeral joint and humeral head stabilization.

The rotator cuff muscles and tendons form the boundaries of a number of crucial anatomical landmarks. These include the quadrilateral space that contains the axillary nerve and the posterior circumflex vessels. The boundaries are teres minor superiorly, teres major inferiorly, the shaft of humerus laterally and long head of triceps medially.

The triangular space contains the scapular circumflex artery and radial nerve. It comprises the teres minor and major, superiorly and inferiorly respectively, and long head of triceps laterally.

The long head of biceps tendon has also been found to play an important role in subacromial impingement. The long head of biceps tendon arises from the supraglenoid tubercle within the glenohumeral joint and the initial 2–3 cm of its course is intra-articular. It exits the shoulder joint as it passes into the bicipital groove of the proximal humerus. The groove is formed by the posterior aspect of the lesser tuberosity and the anterior aspect of the greater tuberosity.

Pathophysiology

The specific cause of rotator cuff degeneration remains unclear. Initially many believed that it was purely a mechanical process resulting from extrinsic wear of the bursal surface of the tendon beneath the superior arch of the acromion and many studies support this theory. Fu et al. attributed impingement to extrinsic forces in the majority of non-atheletes.[5] Wuelker et al. presented convincing cadaveric biomechanical evidence[6] and Neer presented his surgical outcome results based on this theory.[7] In 1983 Neer described the three stages of impingement (Table 1). In 1986 Bigliani classified the morphology of the shape of the acromion into three types, Type I is flat, Type II is curved and Type III has an anterior beak or hook[8] (Figure 1).

However not all surgeons were convinced that extrinsic factors were the only cause of impingement and cuff degeneration, so there were alternative contemporary theories. Rathburn and Macnab postulated that microvascular insufficiency[9] leads to areas of prolonged hypoxia and, as a result, degeneration. Uhtoff and Sarkar suggested microdegeneration of the collagen within tendon[10] and this has prompted a greater interest in intrinsic biological factors operating in the cuff.

Neer's stages of impingement (1983)

Stage I	Oedema and haemorrhage of cuff and bursa. Reversible
Stage II	Irreversible. Fibrosis and tendinitis of rotator cuff tendons
Stage III	Partial or full thickness tears of rotator cuff tendons. Irreversible without intervention

Table 1

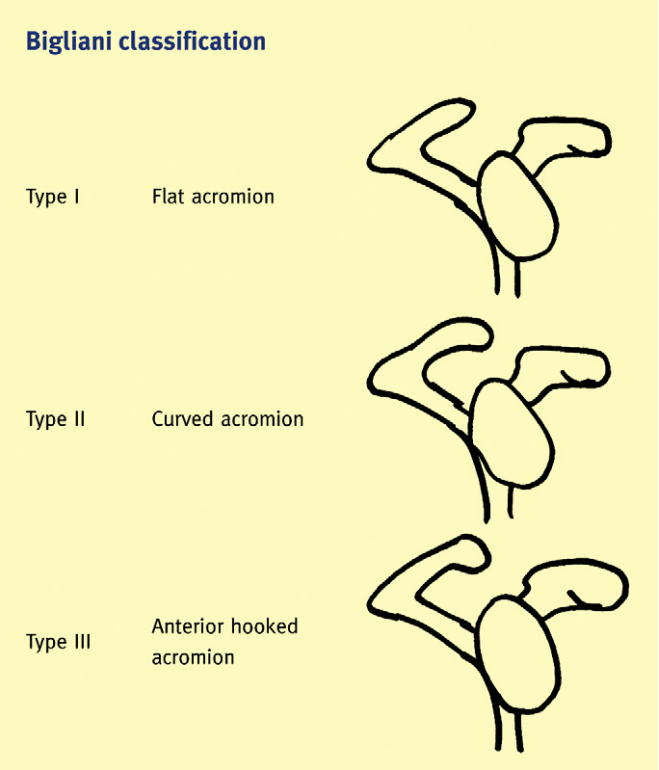

Figure 1

Intrinsic factors

Muscle dysfunction: Nirschl[11] postulated that impingement may be due to muscle dysfunction. This occurs in athletes and overhead manual workers secondary to overload of the supraspinatus tendon. When the arm is in the overhead position, the supraspinatus experiences eccentric contraction to balance the internal rotation and adduction of the arm. In a repetitive activity, this may lead to overload, due to the difference in lever arm length as previously described.[12]

It has also been suggested that as the muscle fatigues in overuse, (i) the function of the rotator cuff to centralize the humeral head in the glenoid and (ii) proprioceptive feedback from the tendon mechanoreceptors are both diminished, and the result can be upward subluxation under the coraco-acromial arch, possibly resulting in impingement.[13,14]

Overuse: impingement due to overuse is a diagnosis of exclusion.[12] It is more common in younger patients who partake in repetitive overhead manual work or competitive sports men and women, particularly, throwing athletes. In these cases, the microtrauma suffered by the tendon outweighs the microrepair. This in turn results in chronic inflammation of the rotator cuff. Chronic inflammation of the tendons causes friction and wear as they pass through the limited aperture of the subacromial arch.[15–17]

Degenerative tendinopathy: a study of 200 cadaveric shoulders demonstrated that all shoulders with abnormal acromial morphology (see below) demonstrated some damage to the bursal surface of the cuff. However as all tendons with evidence of degeneration did not have a corresponding acromial abnormality, the authors concluded that there must be an underlying degenerative element within the tendon.[18]

Many studies have implicated degeneration of the rotator cuff tendons as the primary aetiology of shoulder impingement. It is thought that the resulting dysfunction of the tendons may reduce their ability to centre the humeral head in the glenoid and therefore lead to cephalad subluxation and hence impingement.[10]

Recent histological studies from biopsies at surgery have shown that the cellularity of the rotator cuff decreased as the tear size increased.[19] The authors found that there was a small increase in macrophage, mast cell and leucocyte populations as a small tear appeared, but then as the tear propagated these population sizes all diminished below the levels of control tendons with no tears. The same phenomenon was also seen with respect to tendon vascularity and neoangiogenesis.

Cell populations and cell type are also affected by degeneration and tearing of the tendon. The numbers of tenocytes within the tendon decreases and they are replaced by increasing numbers of chondrocytes in degenerate tendon. Deposition of Type II cartilage within the collagen fibres of the tendon has been demonstrated.

There is almost certainly a genetic element. The incidence of cuff tears in the siblings of patients with the same diagnosis was 59% whereas the incidence in matched controls with no siblings with a similar history was only 25%.[20]

The role of hypoxia and apoptosis (programmed cell death) in rotator cuff degeneration and subsequent tears has been investigated. It has been that, when compared to controls with no impingement, there was evidence of relative hypoxia in intact but impinging tendon and smaller cuff tears. There was also evidence of increased levels of pro-apoptotic proteins in partial and small tears but this reduced in massive tears.[21]

Extrinsic

Acromial shape: the shape and inclination of the acromion have been postulated as the main extrinsic cause of impingement for many years.[22,23] Neer reported in 1972 that shape and angle of the anterior acromion (particularly, an anterior spur) were causative for subacromial impingement.[7] It was based on this fact that he developed his treatment in the form of an anterior acromioplasty. The spur is thought to arise as a result of repetitive microtrauma due to traction, at the insertion of the coraco-acromial ligament.[12]

As previously mentioned Bigliani classified the shape of the acromion into three simple and common morphologies (Figure 1).[8] It was reported that the incidence of full thickness rotator cuff tears was higher in patients with Type III, hooked acromions and indeed 80% of later patients with a visible tendon tear on arthrogram had Type III morphology.[12] More recently, however the interobserver reliability of the Bigliani classification using standard shoulder 'outlet' views has been bought into doubt.[24,25] The difficulty may arise due to the radiographic projection angle rather than the classification being flawed.[26]

Glenohumeral instability: underlying ligamentous laxity and resulting instability must be in the forefront of the surgeon's mind when a young patient (<35 years) presents with the clinical signs and symptoms of impingement. It is uncommon, but not impossible, for this age group to develop true primary subacromial impingement and it is more commonly the result of subtle instability of the glenohumeral joint. The rotator cuff muscles, as mentioned previously are important dynamic stabilizers of the glenohumeral joint and electromyography shows that they are active throughout elevation of the upper limb.[27]

The subtle movements of the humeral head within the glenoid can dramatically affect the biomechanics of the shoulder, particularly in overhead activities related to occupation and sport, leading to secondary impingement of the rotator cuff tendons.[5,28]

This theory is supported in numerous research studies that have shown that young athletes with impingement type symptoms are not improved with surgical acromioplasty alone.[28,29] The treatment of these patients must include correction of the underlying cause of the problem.

Scapulothoracic dysrhythmia: scapular dysrhythmia can present as a 'secondary' clinical sign due to an underlying pathology. Athletes, particularly those engaged in swimming and throwing sports, may develop subtle dysfunction of the scapulothoracic muscles that in turn causes 'winging' or abnormal protraction of the scapula on the thoracic wall.[5,30–32]

Scapular dysrhythmia or scapular muscle weakness will lead to an increase in the forces across the glenohumeral joint causing secondary extrinsic subacromial impingement. Abnormal function of the long thoracic nerve (supplying serratus anterior) or the spinal accessory nerve (supplying trapezius) may also result in scapular winging and secondary impingement.

Acromioclavicular joint (ACJ) degeneration: the role of the ACJ in impingement is unclear, but degenerative change in this joint is a common accompanying feature. Large inferior osteophytes arising from the ACJ can occasionally be seen to indent the superior surface of the rotator cuff tendons and subacromial bursa and in these cases it may play a role.[7,33–35] The joint, however, should only be resected in the presence of symptoms and degenerative change.[12]

Impingement by the coraco-acromial ligament: impingement against the anterior edge of the acromion is an accepted cause of subacromial impingement. The coraco-acromial ligament is another structure that has been implicated in the development of extrinsic impingement,[7,15,33,36,37] and the name 'snapping shoulder' attached to this condition.[38]

Several studies, cadaveric and surgical, have shown enlargement and thickening within the tendon[39,40] and it is accepted that in the presence of swollen and inflamed subacromial structures, a prominent and unyielding coraco-acromial ligament can lead to extrinsic impingement of the rotator cuff tendons.

Os acromiale: originally described in 1863, an os acromiale is a mobile segment of the acromion due to failure of fusion of the distal acromial epiphysis. The prevalence is between 1 and 15% of the normal population.[41,42] Due to the anterior pull of the deltoid, the relatively mobile section of the acromion can lie in a 'flexed' position and cause impingement on the cuff surface or the traction of the coraco-acromial ligament can displace the os into a similar positon.[41]

The classification of os acromiale morphology is shown in Figure 2.

Treatment of a symptomatic os acromiale is tailored to the size of the fragment. Small fragments are excised, larger fragments are reduced and fixed using a tension band system or screws.[43,44]

Sub-coracoid impingement: sub-coracoid impingement is an uncommon diagnosis but one the shoulder surgeon should consider.[45,46] The patient generally describes pain over the anterior aspect of the shoulder that often radiates down into the upper arm. The pain is often made worse by forward elevation and particularly internal rotation.[12] The theory is that the pain is caused by the impingement of the subscapularis tendon beneath the coracoid arch.

Diagnosis on clinical grounds alone is difficult but MRI scanning has helped in the diagnosis of this condition. Evidence of inflammation within the subscapularis tendon or beneath the bony architecture of the coracoid in conjunction with the above symptoms should make the examiner consider this diagnosis. It has been demonstrated that the interval between the coracoid and lesser tuberosity in patients with sub-coracoid impingement was about half (6 mm *vs.* 11 mm) that of patients without evidence of the condition.[45]

Treatment of subacromial impingement was initially recommended to include excision of the coracoid process and transposition of the conjoint tendon.[47] However, as arthroscopic techniques have improved, an arthroscopic coracoidplasty can now be undertaken to increase the space for the subscapularis tendon to pass.

Internal impingement (postero-superior impingement): internal or postero-superior impingement occurs most commonly in overhead workers and throwing athletes.[48,49] The symptoms are felt as pain in the 'cocking' phase of the throwing action. In this position the shoulder is in maximal abduction, external rotation and extension and the articular side surface of the rotator cuff tendons impinge on the postero-superior aspect of the glenoid. This may be exacerbated in anterior instability, as clearly this is also the position of maximal anterior translation of the humeral head.[50]

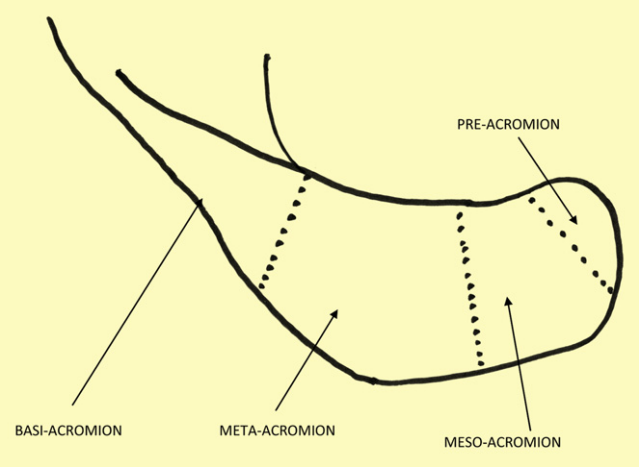

Figure 2 Diagram showing areas of failed apophyseal fusion and os acromiale morphology.

During repeated loading cycles, attritional wear may occur leading to tendinitis and eventually a possible tear and labral damage. This can occur more quickly as muscle fatigues.[12]

Presentation

Patients are normally in their forties or fifties (average of 51.5 years for tendinopathy[4]) and complain of pain. The pain has often been present for many months after an insidious onset and has not settled despite analgesia and physiotherapy. Patients may recall a specific incident when they have injured the shoulder that precipitated the start of the pain. Care must be taken in diagnosing younger patients that present with similar symptoms with impingement syndrome, as they may have impingement as a result of underlying instability. Clearly, treating the impingement and not the underlying cause is unlikely to improve their symptoms. The pain is generally felt in the upper arm and may be worse at night and on reaching or elevation and rotational movements. They may complain of a generalized background ache with severe exacerbations on certain movements, which may assist the diagnosis. Codman and Neer both clearly described the classic presentation of impingement and cuff lesions. Common features include patients undertaking repeated over-head activity, a 'painful arc' between 70 and 120° of elevation, tenderness over the supraspinatus tendon and crepitus.[4]

Pain may not be the only complaint. Patients may also be experiencing stiffness and weakness of the shoulder. These two symptoms may be as a result of pain inhibition but if they persist, after subacromial injection, the examiner should consider other pathologies such as (i) arthritis or frozen shoulder if stiffness remains, and (ii) rotator cuff tears or neurological lesions if weakness persists.

A detailed history of neurological symptoms must also be taken. In neural compression pain may be experienced around the shoulder. The patient may also experience symptoms of weakness in the shoulder, elbow or hand, and they may also report altered sensation or numbness in the upper limb. It will then be important to delineate any dermatomal pattern to the sensory changes or neurological deficit.

Occupation and a history of other activities are crucial, as impingement may be more common in people who undertake a lot of overhead activity or throwing. It is also important to question the patient about previous treatment and any response they may have had to non-steroidal anti-inflammatories, subacromial injections and physiotherapy for example.

Examination and specific tests

Physical examination should confirm what has already been postulated after a full and thorough history. A systematic 'Look, Feel, Move' approach should reliably elicit the clinical signs present.

Inspection

The patient should be stripped to the waist to fully expose the anterior and posterior aspects of the shoulder, torso and upper limb. Scars, sinuses, muscle wasting, swelling and asymmetry can then clearly be visualized.

Palpation

Palpation alone has a limited role in shoulder examination. The landmarks that should be palpated and can yield useful diagnostic information include the acromioclavicular joint (ACJ), the long head of biceps tendon (LHB) and the antero-lateral rotator cuff tendons.

The ACJ is a common pain generator in all age groups from the third decade onwards. Pain is generally felt directly over the joint and palpation can reproduce the pain. Cross body adduction or 'the scarf test' are reliable in diagnosing the condition.

The LHB can be palpated in the bicipital groove on the anterior aspect of the shoulder but generally the dynamic tests (see below) of the LHB are more reliable. The LHB is frequently pathological in patients with impingement. The tendon should specifically be tested during examination and inspected at the time of surgery.

The rotator cuff tendons, particularly the supraspinatus tendon, can be palpated at the antero-lateral corner of the acromion. In some patients with tendinopathy and cuff tears this may elicit discomfort and if a full thickness cuff tear is present it is described that a defect may be physically palpable.

Movement

The majority of the examination of the shoulder takes place with the patient and examiner standing.

Examination of the shoulder should involve a thorough examination of the movements of the cervical spine. In the presence of degenerative changes of the cervical spine, nerve root compression or radiculopathy of the C5 nerve root may present as pain over the shoulder. Flexion, extension, lateral flexion and rotation of the cervical spine can easily be undertaken.

With the patient stood facing away from you, scapular movement, symmetry and rhythm can be assessed. Ask the patient to slowly elevate their arms in front of them to approximately 90°. The arms are then lowered again. Observation of static and dynamic winging of the scapula can be present in many pathologies but give a general clue that something is amiss. Early scapulothoracic movement of one side may represent stiffness within the glenohumeral joint resulting in compensatory movement of the scapula.

Active and passive range of motion can then be assessed. Examination of flexion, abduction, internal and external rotations is usually sufficient to assess function, but extension and adduction can be assessed, and also contribute to more specific tests, as described in Table 2.

Active flexion is crucial in impingement. As described by Neer,[7] as the arm is elevated in the scapular plane the patient experiences pain, usually between 70 and 120°. A loss of active flexion or weakness of flexion in the presence of full passive flexion suggests a tear of the rotator cuff tendons. Stiffness may represent impingement or a tear or glenohumeral pathology which will become more evident during the rest of the examination.

The passive range of external rotation is an excellent indicator of glenohumeral pathology. It is best assessed initially with the patient stood with their elbows tucked into the waist and flexed to right angles. The range of rotation is measured from the neutral position with the forearms pointing directly forwards. Reduced external rotation may occur in rotator cuff pathology due to pain inhibition but it is more likely to represent osteoarthritis or frozen shoulder.

Internal rotation is normally assessed by asking the patient to 'reach up their back' and the vertebral level reached can be compared to the unaffected side.

The strength generated during flexion/elevation and rotational movements is important, particularly in rotator cuff disorders. Profound weakness may well reflect a tear of the cuff, due to pain inhibition, or less commonly a neurological deficit.

If a patient less than 35 years old presents with signs and symptoms of impingement, then the examiner must consider subtle instability of the glenohumeral joint as the underlying

Specific tests of impingement

Neer's sign	Pain on elevation of arm in plane of scapula. Pain normally felt between 70 and 120° of elevation.
Jobe's test	Arm elevated to approx. 60°. Arm placed into full internal rotation and elevation resisted by examiner. The same is then repeated with the arm in full external rotation. A positive test evokes pain on IR but the pain is less when repeated in ER.
Hawkins—Kennedy test	The arm is placed into 90° of elevation in the scapular plane. The elbow is flexed to 90° also and the examiner passively internally rotates the shoulder. A positive test elicits pain as the greater tuberosity is rotated beneath the acromion.
Neer's test	Neer's test. If there is a positive sign, local anaesthetic is infiltrated into the subacromial bursa. After a few minutes the sign is repeated. A positive result is the abolition of pain or increased range of motion and strength.
Yergasson's test (LHB)	Speed's test is undertaken with the shoulder in neutral and the elbow flexed at 90° at the waist. The forearm is actively held in full supination as the examiner tries to pronate the forearm. The test is positive when pain is felt over the anterior shoulder over the LHB. The thumb of the examiner's other hand may be used to palpate the tendon.
Speed's test (LHB)	The shoulder is elevated in the scapular plane to approximately 30°. The elbow is extended and forearm supinated. Resisted elevation of the shoulder is undertaken. The test is positive when pain is felt over the anterior shoulder over the LHB. The thumb of the examiner's other hand may be used to palpate the tendon.

Table 2

cause. If this is the case a full assessment of the stability of the shoulder should be undertaken.

Radiology

Plain radiography

Plain radiographs are useful in the diagnosis of impingement, despite the fact that the tendons themselves are not visualized, there are often clues to the underlying disease process. At least two views of the shoulder are required. A standard antero-posterior view, lateral scapula, outlet view or axial views are all commonly utilized.

In the presence of impingement alone the antero-posterior radiograph alone may demonstrate subtle signs. The 'sourcil sign' (French for 'eyebrow' (Figure 3)) is present when there is increased sclerosis of the undersurface of the acromion. Degenerative changes of the ACJ may lead to large inferior osteophytes that may also lead to impingement. In the presence of a rotator cuff tear or cuff insufficiency there may be evidence of subtle or profound cephalad subluxation of the humeral head in the glenoid.

The outlet view of the shoulder may show anterior spurs on the inferior acromion and also allow the surgeon to assess the shape of the acromion according to the Bigliani classification (Figure 1).

Ultrasonography

Ultrasound (USS) assessment of the rotator cuff tendons is now generally accepted to be comparable to magnetic resonance imaging (MRI) in the diagnosis of full thickness tendon tears. In a recent meta-analysis it has also been reported to be comparable in the diagnosis of partial thickness tears.[51] Anecdotal evidence, however, suggests that USS is not as reliable in diagnosing partial thickness tears and tendinopathy.

USS clearly has the benefit that it is a dynamic imaging modality and the patient can place the arm into the painful range of motion whilst the ultrasonographer looks for evidence of impingement or catching of the bursa or rotator cuff tendons beneath the acromion. USS can also clearly visualize the presence of calcific deposits within the substance of the tendon, a relatively common cause of impingement-type symptoms (Figure 4).

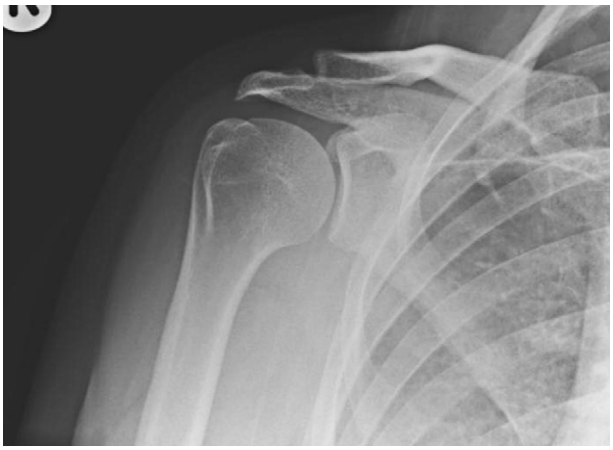

Figure 3 AP radiograph of the right shoulder showing sclerosis of the undersurface of the acromion — 'sourcil sign'. Note also the large antero-lateral bony spur on the acromion.

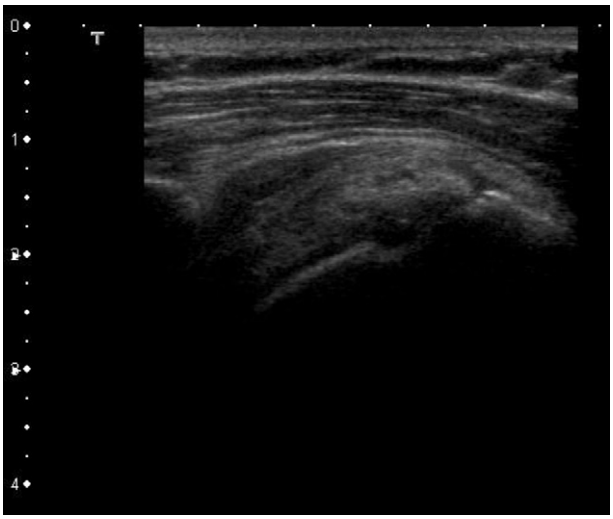

Figure 4 USS image of calcific deposit in supraspinatus tendon. From scale on left of image, deposit appears approx. 10 mm × 6 mm.

Magnetic resonance imaging

MRI is a reliable imaging method for the diagnosis of rotator cuff tears, both full and partial thickness. It is as reliable at detecting tendinopathies and tendinitis as USS, due to its ability to demonstrate inflammatory processes and oedema within the tendon. It has been shown in canine subjects that MRI is accurate in the preoperative diagnosis of abnormalities within the rotator cuff tendons and the long head of biceps tendon.[52] MRI is a reliable method of diagnosing tendinopathy of the rotator cuff, however the reliability is significantly better in the hands of a senior, suitably trained radiographer[53] (Figure 5).

Injection therapy

The use of injection therapy is common in the shoulder. Local anaesthetic and steroid injections can be used therapeutically but also as a diagnostic tool. Relief from symptoms after an injection into the subacromial space is reassuring evidence that it is the contents of this space, i.e. the tendons or the subacromial bursa, as opposed to the glenohumeral joint or cervical spine, that is the root of the problem.

It is common practice to infiltrate the subacromial space with 40 mg of steroid, e.g. triamcinalone or methylprednisolone, diluted in up to 10 ml in local anaesthetic. Strict sterile techniques should be undertaken. The risk of 'septic bursitis' is exceeding low with very few reported cases,[54,55] however the morbidity associated with it is significant.

Injections of local anaesthetic can make up part of a standard shoulder assessment as in Neer's test (Table 2) to assess for an immediate reaction of reduced pain or increased strength. If the patient responds well to the local anaesthetic or in the longer term, the steroid, then it confirms that the subacromial space is the source of the symptoms and occasionally patients can be cured with a single injection.

Treatment

Treatment of impingement of the shoulder clearly depends on the underlying aetiology. If subtle instability is the underlying cause, physiotherapy to enhance dynamic stability and proprioception

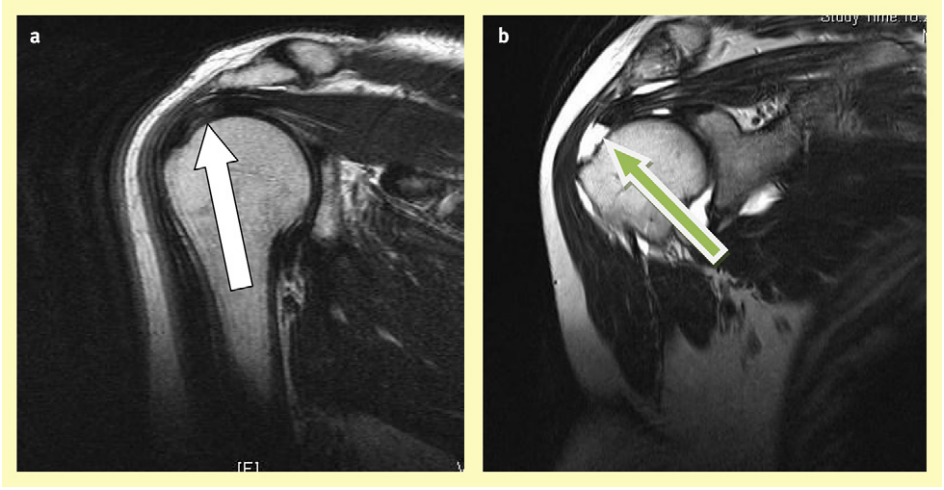

Figure 5 Two images from shoulder MRI. Image **a**, on the left demonstrates increased signal (white arrow) within the supraspinatus tendon. This represents the tendinopathy within the substance of the tendon. Image **b** on the right clearly shows a full thickness tear of the supraspinatus tendon (green arrow).

would be the likely first line of treatment. This section however, will concentrate on the treatment of a tendinopathic tendon with evidence of an extrinsic bony spur from the acromion.

Some patients will gain good long-term relief from a subacromial injection and need no further treatment. Others will gain excellent symptomatic relief for months at a time but the symptoms predictably return. In these cases it may be acceptable to repeat the injections if there is prolonged relief. Up to three injections annually are generally accepted as the maximum 'safe' dose as it has been shown that repeated exposure to steroid leads to a reduction in the tensile strength of the collagen fascicles within the tendon and therefore increased risk of rupture.[56]

There is evidence that physiotherapy can improve the symptoms in impingement. The treatment concentrates on strengthening the rotator cuff muscles, particularly the anterior subscapularis and posterior infraspinatus. The theory is to maximize the effect of these muscles as a 'force couple' to centre the humeral head more inferiorly in the glenoid. This in turn increases the subacromial space and hence reduces the pressure on the superior rotator cuff.

For recurrent or resistant impingement in a suitable patient with a clear history, clinical examination findings and ideally radiological evidence, surgery may be considered. Numerous operative procedures have been described over the years with the majority of the early descriptions involving an open procedure. Early reports suggested the excision of the entire acromion or the majority of the anterior process[57] but the results of this procedure were poor in the long-term. The operation of acromionectomy resulted in defunctioning of the deltoid and if a rotator cuff tear developed later the outcome was disastrous. Neer described limited resection of the anterior-inferior third of the acromion (acromioplasty) and excision of the coraco-acromial ligament at its

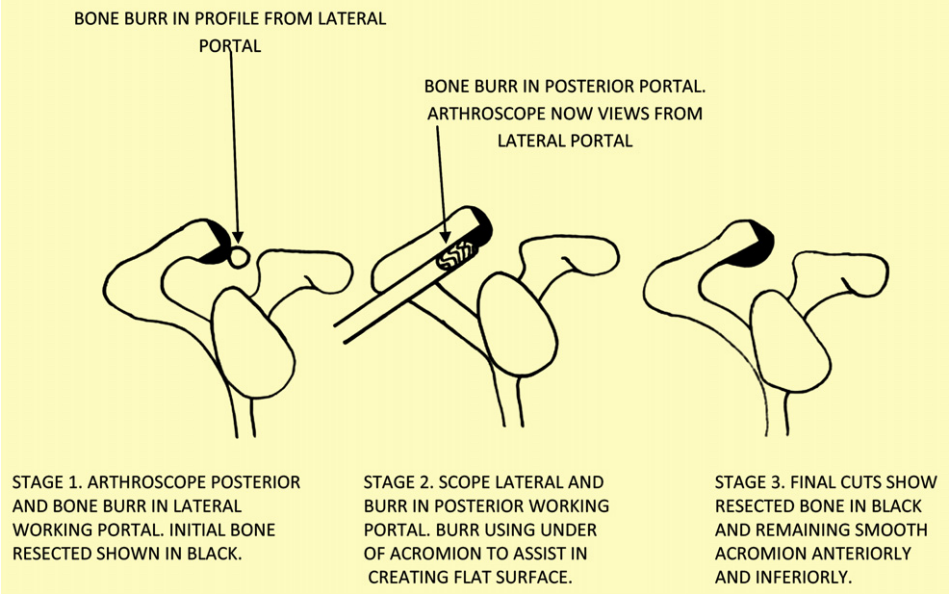

Figure 6

insertion.[7] The description again was of an open procedure, but this was the surgical management of choice for many years.

Arthroscopic subacromial decompression has gained favour since it was first described by Ellman.[58] The surgical technique involves excision of the inflamed bursa, division of the coracohumeral ligament and removal of the anterior acromion and any other spurs using a high-speed burr. The completed acromioplasty must meet the same criteria as the open procedure, i.e. cover the full width of the acromion and all spurs must be removed. The 2–5-year results were good with the vast majority reporting satisfactory or excellent outcomes and very few complications.[59]

There is convincing long term evidence supporting the use of surgery in the management of subacromial impingement. The long-term follow-up of 105 shoulders between 8 and 11 years post-subacromial decompression reveals favourable outcomes with statistically significant improvement in range of motion, strength, pain and functional outcome scores.[60]

Recent meta-analyses, however, have questioned the efficacy of the surgical management of impingement. There is evidence that conservative treatment, including anti-inflammatories, injection therapy and physiotherapy can offer as good an outcome as arthroscopic subacromial decompression, although more high quality randomized controlled trials are needed to confidently draw this conclusion.[61,62]

Subacromial decompression has been shown to reduce the prevalence of rotator cuff tears. Patients that underwent subacromial decompression were investigated 15 years post-decompression for partial and full thickness rotator cuff tears. The prevalence in the study group was only 18%, approximately half of the reported prevalence in a similar age group of over 60 years, 40%.[63]

Subacromial decompression is a successful surgical procedure when undertaken by an experienced surgeon on the 'right' patient. The initial management of the patient should include an accurate and detailed history and full clinical assessment (Table 2) to eliminate other underlying pathologies that are causing 'secondary' impingement.

Various surgical techniques have been described for arthroscopic subacromial decompression. Many surgeons perform the technique with the arthroscope in the standard posterior portal for viewing and the 'working' portal laterally. Radiofrequency ablation[64] is commonly used in conjunction with a soft tissue resector to clear the bursa and release the coraco-acromial ligament from the undersurface of the acromion. It is crucial to adequately clear the soft tissue to be able to fully visualize and assess the contour of the acromion and hence the surgical resection once undertaken. In longer procedures, such as a rotator cuff repair, this clearance is even more important as over time the residual soft tissues can swell due to saline being pumped through the space resulting in irretrievable loss of view.

An alternative surgical technique, and one favoured by the senior author, is the so-called, 'cutting block' technique. This requires an initial resection of the anterior spur and front edge of the acromion as described above. The arthroscope is then switched to the lateral portal and the posterior portal is used as the 'working' portal. This allows the surgeon to use the posterior aspect of the undersurface of the acromion as a guide to the level of resection of bone required to produce a flat undersurface in two planes (Figure 6).

Summary

Rotator cuff tears and tendinopathy are a common presentation to the specialist shoulder surgeon. Impingement was initially considered to be a problem arising from the antero-lateral acromion causing extrinsic pressure on the superior surface of the supraspinatus tendon.

More recent research has presented convincing evidence that it is in fact a multifactorial condition that involves both extrinsic factors, such as compression of the tendon, intrinsic factors such as hypoxia and collagen degeneration and environmental factors, such as occupation and sporting activities.

The standard orthopaedic practice of 'look, feel, move' should be utilized in making the diagnosis. A clear idea of the diagnosis should be apparent from the history and examination alone in the majority of patients using the specific tests seen in Table 2. The use of injection therapy and specific radiology are used as adjuncts in the confirmation of surgeons' ideas.

It is a common diagnosis from the late fourth decade onwards. Making the diagnosis in younger patients should be undertaken with caution and only having ruled out more common conditions such as subtle instability.

Once the diagnosis has been confirmed, the treatment is tailored to the patient. Analgesia and physiotherapy to improve muscle tone and proprioception may be all that some patients require, however some may benefit from a single or repeated injections depending upon the length of benefit each affords. Many patients, however, come to surgical decompression. This can be carried out as an open procedure but generally the arthroscopic technique has gained favour. The basic premise, however, remains the same, complete decompression of the tendon over the entire depth and width of the acromion, excision of the bursa and division of the coraco-acromial ligament. ◆

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