Narrative Review

Subacromial impingement syndrome: a musculoskeletal condition or a clinical illusion?

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Background: Subacromial impingement syndrome is considered by many to be the most common of the musculoskeletal conditions affecting the shoulder. It is based on a hypothesis that acromial irritation leads to external abrasion of the bursa and rotator cuff.

Objectives: The aim of this paper is to review the evidence for the acromial irritation theory and in doing so challenge the rationale for subacromial decompression.

Major findings: There is a body of evidence that suggests there is a lack of concordance regarding (i) the area of tendon pathology and acromial irritation, (ii) the shape of the acromion and symptoms, (iii) the proposal that irritation leads to the development of tendinitis and bursitis, and (iv) imaging changes and symptoms and the development of the condition. In addition, there is no certainty that the benefit derived from the surgery is due to the removal of the acromion as research suggests that a bursectomy in isolation may confer equivalent benefit. It is also possible that the benefit of surgery is due to placebo or simply enforces a sustained period of relative rest which may allow the involved tissues to achieve relative homeostasis. It is possible that pathology originates in the tendon and as such surgery does not address the primary pathoetiology. This view is strengthened by the findings of studies that have demonstrated no increased clinical benefit from surgery when compared with exercise. Additionally, exercise therapy is associated with a substantially reduced economic burden and less sick leave.

Conclusion: As there is little evidence for an acromial impingement model, a more appropriate name may be ‘subacromial pain syndrome’. Moreover, surgery should only be considered after an appropriate period of appropriately structured rehabilitation.

Keywords: Shoulder, Subacromial impingement syndrome, Subacromial bursa, Acromioplasty, Subacromial decompression, Rotator cuff tendinopathy, Shoulder posture

Background

The shoulder complex has a range of movement that exceeds any other joint in the body and its main function is to position the hand to affect functional activities ranging from the performance of high powered explosive activities, such as throwing baseballs, to positioning the hand, often within the field of vision, to perform highly complex prehensile tasks. The shoulder is also used to place the hand so that the upper limb may be used for weight bearing. Musculoskeletal pathology involving the shoulder is common, has the potential to adversely affect upper limb function and is associated with substantial morbidity that increases with age.1–3 Of the wide spectrum of musculoskeletal disorders affecting the shoulder, subacromial impingement syndrome is considered to be one of the most common.4,5 This condition is well recognized clinically, presenting as antero-lateral shoulder pain experienced when the arm is elevated.4–7 Although numerous historic references to subacromial pathology exist,8–13 Neer argued that abrasion by the under surface of the anterior margin of the acromion onto the soft tissues located anatomically in the space between the humeral head and acromion leads to the symptoms experienced in subacromial impingement syndrome.4 He stated that this compression occurred principally in forward elevation and described a clinical test, the ‘(Neer) impingement sign’ to reproduce the associated symptoms.5 The test involves restricting scapular movement and forcing the arm into flexion while the shoulder remains internally rotated.5 According to Neer, this manoeuvre causes the greater...
tuberosity to impinge against the acromion. He argued that 95% of rotator cuff tears are initiated by impingement and that trauma may enlarge a tear but is rarely the principal factor. Neer described three stages of the impingement process. The first occurs in people under 25 years of age and is associated with tendinous oedema and haemorrhage, and does not require surgery. The second involves tendinitis and occurs in people aged 25 to 40 and bursectomy and coracoacromial ligament division should be considered after 18 months of conservative treatment. Neer stated that in this group an acromioplasty is not usually required. The third stage occurs in people over 40 years of age and is associated with bone spurs and tendon rupture and requires anterior acromioplasty. Neer stated that the reason rotator cuff tears develop in some people and not others is principally due to the shape of the acromion. This hypothesis was supported by Bigliani et al. who described three distinct morphological variations of acromial shape. Bigliani et al. argued that as a result of the shape and the damage it would cause, those with a Type III or hooked acromion were more likely to experience subacromial impingement syndrome and suffer a rotator cuff tear. Prior to Neer presenting his model, surgeons were performing complete acromioplasties and lateral acromioplasties to alleviate the symptoms. Basing his argument on intra operative and cadaver observations, Neer asserted that removal of the inferior aspect of the anterior acromion had greater efficacy. To augment the procedure, he suggested that a partial resection of the coracoacromial ligament together with surgery to remove a hypertrophic acromioclavicular joint may be required to arrest the impingement process.

Neer’s impingement model has been widely embraced by surgeons, sports physicians and physical therapists. So much in fact, that the percentage of acromioplasties performed in New York State (USA) alone has increased 254% in the 10 years from 1996 to 2006. The number of procedures has increased from 30 per 100 000 people (5571 operations) to 102 per 100 000 (19 743 operations). Ketola et al. reported that the average cost of an acromioplasty and post-surgical rehabilitation in Finland was €2961 (equivalent to GB£2479, US$4017). In London, UK, an average price for a series of quotations for private subacromial decompression was GB£3300. In New York State, a figure of US$4860 has been given. If this is a representative amount, then staggeringly, the total cost of performing acromioplasties in New York State alone would be in the order of US$95 959 980. These figures are not definitive and costs in some centres may be less and in others higher. Nonetheless this is of substantial economic burden and healthcare concern, as Ketola et al. have stated:

‘Arthroscopic acromioplasty provides no clinically important effects over a structured and supervised exercise programme alone in terms of subjective outcome or cost-effectiveness when measured at 24 months. Structured exercise treatment should be the basis for treatment of shoulder impingement syndrome, with operative treatment offered judiciously until its true merit is proven’.

This begs the question that if surgery aiming to remove the cause of the impingement irritation (i.e. the acromion) is no more clinically effective than a substantially less expensive structured rehabilitation programme, then is the original hypothesis correct, is the procedure valid, or is there an alternative explanation for the symptoms?

Objectives

The aim of this paper is to review the evidence for the acromial irritation theory and in doing so challenge the rationale for the surgical removal of the inferior aspect of the anterior acromion to remove the source of symptoms.

Objective Findings

Area of pathology

If 95% of rotator cuff failure is caused by mechanical irritation by the under surface of the acromion or coracoacromial ligament, then this should result in abrasion to the superior (bursal side) surface of the rotator cuff, especially the supraspinatus. Published research disputes this. Payne et al. reported that 39% partial thickness tears in 43 athletes were on the inferior (articular or joint) side of the supraspinatus tendon with only 4% or 9% on the superior or bursal side. In this series, 100% of those with non-traumatic shoulder pain had articular side tears. Fukuda et al. reported that in a study of 249 cadavera, 13% demonstrated partial thickness tears. Of the partial thickness tears 82% were either joint side or intra-tendinous (n=27) and only 28% (n=6) were isolated to the upper bursal/acromial side. Ozaki et al. examined 200 shoulders from 100 cadavera and reported that a partial thickness tear was observed in 69 specimens and that the majority involved the deeper articular side of the tendon. They argued that the prevalence of tears increased with age and occurred due to intrinsic degeneration and not external (acromial) irritation. In a study of 306 rotator cuff specimens (from 153 cadavera) the prevalence of partial thickness tears was 32%, with histological and scanning electron microscopy sections demonstrating that the majority were either intra-substance or occurred on the articular side of the supraspinatus tendon, near the insertion. This study did not find a correlation between anatomical bone variations and tendon failure and argued that mechanical abrasion may not play an important part
in the initial pathogenesis of degenerative rotator cuff tears. Ellman reported that partial thickness tears were found in 15% (n=20) of people undergoing arthroscopic subacromial decompression (n=130). He reported that when the findings of his observational work was combined with the findings of five other studies a total sample of 160 partial thickness tears was produced, and of these the location was reported in 126 cases. From these 126 cases, 76% (n=96) had articular side tears, 14% (n=17) had bursal side, and 10% (n=13) had both. This repeated and consistent finding that the predominance of partial thickness tears occurs on the deeper articular side of the tendon substantially challenges the hypothesis that 95% of rotator cuff tears are caused by acromial abrasion. Furthermore, Codman already in 1934 observed that the rotator cuff degenerates within the substance of the tendon or frequently along the inferior margin of the tendon, the side opposite the acromion, calling partial thickness tears in this region, 'rim rents'. He stated, ‘…I am confident that these rim rents account for the great majority of sore shoulders. It is my unproved opinion that many of these lesions never heal, although the symptoms caused by them usually disappear after a few months’. The weight of evidence supports Codman’s early observations that although the acromion may be involved, the tendon may structurally fail without direct mechanical irritation from the overlying acromion. Supporting this contention, Hashimoto et al. observed diffuse degenerative changes involving tendon thinning, fibre disorientation, myxoid and hyaline degeneration, calcification, and chondroid metaplasia to be more prominent in the middle and deeper rotator cuff tendon layers, suggesting intrinsic tendon failure. Variations observed in the morphology of the supraspinatus tendon support these findings. In a histological and biomechanical investigation of 20 normal rotator cuff tendons, the deeper, non-acromial, side fibres were reported to have a smaller cross-sectional area than the superior acromial side fibres. In addition, when stretched to the point of rupture, the deeper fibres were found to be more vulnerable to tensile load than the bursal side fibres, with the deeper fibres failing at approximately half the tensile load of the failure point of the upper acromial side fibres. Supporting this finding, Bey et al. in a study of seven cadaveric shoulders reported that when placed at 15, 30, 45 and 60° of glenohumeral abduction, strain within the supraspinatus tendon increased with increasing joint elevation. At 60° elevation there was no significant difference in strain between the superior, middle and inferior portions of the tendon. However and importantly, as the inferior fibres are comparatively weaker, Bey et al. argued that the fibres in the inferior region are relatively more susceptible to failure in elevation. This is of clinical relevance as many vocational activities and sporting pursuits involve placing the shoulder in elevation for prolonged periods. As significantly more strain is placed on the inferior fibres at 45 and 60° abduction than at 15 and 30°, and the deeper side fibres are relatively weaker and fail earlier than the larger acromial side fibres, it is arguable that the acromion has little or nothing to do with the failure, which potentially may result from the deeper side fibres passing their physiological failure point.

As there is a lack of concordance between the area of structural failure observed in the supraspinatus tendon and the area predicted by an acromial irritation model, others have suggested that the observed joint side structural pathology is better explained by external impingement between the superior aspect of the glenoid fossa and the humeral head. This alternative external irritation model has been referred to as superior, postero-superior and internal impingement. However, robust evidence required to support this model of impingement is lacking, and as such it is possible that the deep side tendon failure described in this model might not result from external (extrinsic) impingement but may result because of the heterogeneity of the fibre distribution of the upper and lower aspects of the supraspinatus tendon, together with the disparity of tendon loading patterns during movement. Recent reviews on the rotator cuff exploring these and related issues have been published.

More than 70 years ago, Lindblom and Palmer suggested that during shoulder abduction uneven loads may be placed on the upper and lower aspects of the tendon resulting in intratendinous shearing, which may play a part in rotator cuff degeneration and tears. This is relevant as the supraspinatus tendon is made up of structurally independent parallel fascicles and movement will potentially lead to different length tension relationships occurring within and between the different fascicles. For example, at the extreme of shoulder horizontal abduction the anterior part of the tendon may be relatively lengthened and the posterior shortened, whereas at the extreme of horizontal adduction this pattern may be reversed. As a greater range of movement is required of the shoulder than any other joint in the body it is conceivable that internal tendon shearing may result that may predispose pathology without the need for external compression on either the superior or inferior aspects of the tendon. In support of this, external irritation, in the form of an Achilles tendon allograft wrapped around the left acromion, in rats, did not lead to rotator cuff
These findings suggest that if this is the case, then work, argued that three distinct shapes of the acromion existed. These morphological variations included a Type I (flat), Type II (curved) and Type III or hooked acromion. If the acromion is responsible for 95% of rotator cuff pathology and is the causative mechanism of pathology in impingement syndrome, then a definitive relationship between acromial shape, pathology and symptoms should exist with a Type II or III more likely to predispose pathology. However, research evidence has failed to demonstrate this.

In a study of 59 people without shoulder pain the association between acromial morphology, age and rotator cuff tears was investigated. For people over the age of 50 years, a 40% prevalence of asymptomatic full thickness rotator cuff tears was identified in this investigation. Based on the substantial number of people with curved and hooked acromia who were entirely asymptomatic, Worland et al. concluded that, ‘Surgeons should interpret radiologically hooked or curved acromions as well as rotator cuff tears diagnosed with ultrasound or other modalities with caution’. In a study of 55 people who underwent arthroscopic subacromial decompression (anterolateral edge of the acromion resected together with release and resection of the coracoacromial ligament from the acromion), the association between preoperative pain, clinical signs (Hawkins test, Neer sign, Copeland impingement test) and satisfaction with the severity of rotator cuff and acromial lesions was investigated. At the 6 month follow-up no significant correlation between pain and satisfaction and the severity of structural pathology was identified. Confiming this, after a study of 523 people undergoing arthroscopic or open shoulder surgery, Gill et al. reported no significant association between acromial shape and rotator cuff pathology in people over 50 years of age (n=192). A highly significant correlation between age and rotator cuff pathology existed and the researchers argued that a Type III hooked acromial represents a degenerative process rather than a morphological variation as described by Bigliani. Although a relationship between rotator cuff tears and acromial degeneration appears to exist, this should be seen as an association, rather than the acromion being implicated in (i.e. the cause of) rotator cuff pathology.

An alternative explanation for the observed acromial spurs is possible. Edelson and Taitz observed degenerative spur formation on the acromial insertion of the coracoacromial ligament but not on the coracoid side in 18% of 200 scapulae. When compared with shoulder adduction, increasing ranges of shoulder elevation increase subacromial pressure. The coracoacromial ligament is more trapezoid in shape with a smaller area of insertion on the acromial side than the coracoid side. It is therefore possible that superiorly directed pressure from below the ligament will lead to relatively more tension on the acromial insertion of the ligament than on the coracoid side due to the smaller surface area of insertion on the former. This potential increased stress on the bone may lead to osteophyte formation. Supporting this hypothesis, Chambler et al. demonstrated in vivo (n=5) that tension in the coracoacromial ligament increased as the arm was abducted. In an additional study analysis of acromial bone spurs (n=15) suggested that the development of the spurs was a secondary phenomenon. These studies suggest that tension in the coracoacromial ligament is the probable mechanism of acromial bone spur formation and that acromial Type II (curved) and Type III (hooked) as described by Bigliani may not be inherited, but may result from increased strain in the ligament disproportionately affecting the acromial side.

Chronic strain in the coracoacromial ligament may result from changes in the rotator cuff tendons that may involve increased tendon volume, as well as from failure of the rotator cuff to stop superior translation of the humeral head during arm elevation. Evidence for chronic strain exists, with free nerve endings and neovascularity observed in coracoacromial ligament samples from people undergoing subacromial decompression. This suggests that the ligament may be a potential source of symptoms. The coracoacromial ligament limits superior translation of the humeral head and as acromioplasty has
been associated with increased anterosuperior translation of the humeral head, the procedure itself may be an iatrogenic cause of ligament strain.

Ligaments are structures that stabilize joint movement and if disrupted they are replaced in an attempt to recreate stability. Examples of this include patellar tendon and hamstrings tendon grafts for anterior cruciate knee ligament failure. It is therefore surprising that the coracoacromial ligament, which provides a stabilization role by preventing superior translation of the humeral head, has been extensively sacrificed to retard or stop the subacromial impingement, due to the belief that it is of relative structural unimportance, when there is no conclusive evidence to support the existence of primary external impingement from this structure. It would be hard to imagine that a surgeon would suggest, or a patient would agree to, having the anterior cruciate ligament removed to treat knee pain.

Posture and muscle imbalance

Physical therapists have also embraced the acromial irritation model and have argued that an increased kyphosis, a change in scapular position due to poor posture, uncontrolled scapular movement (dyskinesis), or an imbalance in muscle activity leads to subacromial impingement. Even though concepts relating to posture and muscle imbalance have existed for more than half a century it is surprising how little evidence there is to support (i) the existence of an ideal posture of the head, neck, thorax, (ii) the existence of an ideal scapular position (i.e. the basis for scapular setting exercises), (iii) that uncontrolled scapular movement and dyskinesis is always a primary problem, (iv) that postural deviations and muscle length tension changes alter scapular position in a consistent manner and significantly lead to a detrimental effect of movement and provoke impingement symptoms, (v) that rehabilitation can correct posture that is considered abnormal, and (vi) the idea that this correction leads to an improvement in function and a reduction in pain. There is evidence to challenge these concepts, which suggests that the certainty with which this aspect of clinical practice is taught to undergraduate and postgraduate students and imparted to patients and clients requires robust research enquiry. In addition, it is arguably inappropriate to suggest that an increased thoracic kyphosis leads to restricted shoulder movement and impingement based on studies that have unnaturally restricted thoracic spine movement. An assumption implicit within the postural-muscle imbalance model of assessment is that a forward head posture and increased thoracic kyphosis observed during static posture has a direct correlation on dynamic movement, and that all scapulae have the same geometric proportions and move in the same way on the same shaped rib cage and thorax. This is simply not correct, and as such, a one size fits all approach is unlikely to be appropriate. Variations in shoulder function may be dictated by variations in structure, and the differences observed between people with impingement syndrome may reflect a range of normal values and not deviations from one idealized normal posture. If this is correct it would not be possible to identify postural deviations that lead to subacromial impingement. Based on the uncertainty of the current models of postural and clinical assessment alternative models of assessment have been proposed; however, these also require clinical validation.

Tendinitis

Implicit within the three-stage impingement model presented by Neer is the association between the mechanical abrasion caused by the acromion and the ensuing microtrauma within the tendon leading to tendon inflammation (tendinitis). The issue of tendon inflammation is controversial. Although histological studies have demonstrated substantial differences between normal tendon and pathological tendon, the evidence for the presence of cells classically associated with inflammation is not robust. No infiltration of neutrophils, lymphocytes or plasma cells were identified in specimens taken from 12 subjects with rotator cuff disease during surgery. Similarly, no inflammatory cells were identified in bursal specimens (n=8) also taken during surgery for rotator cuff tendinopathy. In another small study, people with constant shoulder pain were more likely to have lymphocyte infiltration in bursal tissue in comparison to people with pain only on movement who did not exhibit evidence of bursal inflammatory cells. There is distinct need for robust evidence from appropriately designed research to better understand if inflammation is part of the continuum of pathoanatomy of tendon and bursal pathology. Without this research an argument that acromial irritation and the ensuring microtrauma leads to bursal and tendon inflammation remains unsubstantiated.

The subacromial bursa

The subacromial bursa (SAB) separates the coracoacromial arch and deltoid above, and the rotator cuff tendons below. Together with the other bursae in the region, whose reported numbers ranging from 7/8 to 12, the SAB acts to reduce friction during movement. The SAB is innervated anteriorly by the lateral pectoral nerve and posteriorly by the suprascapular nerve. The identification of mechanoreceptors and free nerve endings (Aδ and C) in bursal tissue suggests the SAB has a role in...
propriocception and nociception.54 The presence of nociceptors is highly relevant as high concentrations of pro-inflammatory cytokines, pain mediating substances and matrix modifying proteins have been identified in bursal tissue of people whose shoulder pain is exacerbated by shoulder elevation.85–91 Higher shoulder pain scores were reported by those found to have higher concentrations of the cytokine interleukin-1beta and the neuropeptide, substance P, in their bursal tissue.86

One clinical trial randomized people diagnosed with subacromial impingement syndrome (n=57, mean age 47 years), to Group I: arthroscopic acromioplasty and bursectomy or Group 2: arthroscopic subacromial bursectomy alone. Good results were reported in both groups with no significant differences identified between the two groups at a mean follow-up of 2.5 years.92 These findings clearly suggest that the bursa is a significant pain generator and that the addition of an acromioplasty may be superfluous. Henkus et al.92 argued that subacromial impingement syndrome is largely an intrinsic degenerative condition rather than an extrinsic mechanical disorder. The importance of the SAB as a source of shoulder pain is reinforced by studies that have shown that injections reaching the SAB reduced pain while injections targeting other structures increased or did not change pain.93 This may be a reason why ultrasound guided injections appear to produce better outcomes than non-guided injections.94,95

It is also unclear whether treatment with corticosteroid and lidocaine is any more advantageous than lidocaine in isolation.96–99 Using analgesic injections as a control, systemic (gluteal) corticosteroid injections have been reported to be as effective as locally guided corticosteroid injections89 in the treatment of impingement syndrome. However, the certainty of this conclusion is questioned by studies that have shown no added long term effect of analgesic over steroid,96,97 and no one has yet shown the added benefit of the pharmacological substance injected over the mechanical stimulation of the dry needle, which in itself is frequently painful. The science supporting the use of injection therapy for impingement syndrome (timing, volume, medications used, direction of injection, post-injection advice, histological effect on tissues) is not robust and requires ongoing investigation. In the UK, many physical therapists perform injections and an increasing number are performing ultrasound guided injections. Alongside this change to scope of practice is a requirement for further research, which is essential to understand the histological and biochemical nature of bursal pathology and pain and the relationship between bursal pathology, tendon pathology and shoulder pain.

Alternative explanations for the potential benefit of surgery
Success rates of 80–90% following subacromial decompression for impingement have been reported.100–103 Neer5 and those embracing his model argue that removing the acromion removes the source of irritation. Henkus et al.92 has clearly demonstrated this may not be the case, as isolated removal of the bursa has comparable effects to removing the acromion and bursa. However, in addition to the suggestion that bursctomy is more relevant than acromioplasty,92 other, additional explanations for the beneficial results reported following acromioplasty are entirely feasible. In Australia, non-manual workers take on average 6 weeks to return to work following an acromioplasty and 85% of manual workers take 3 months to return to employment, with driving commencing at 29 days post-surgery.104 Comparable findings from the UK suggest that non-manual workers return to work after 9 days, manual workers after 3 weeks and driving recommences after 13 days.105 These data clearly demonstrate that there is a prolonged period of substantial relative rest following the procedure and to date no study has compared surgery and the ensuing relative rest with comparable relative rest alone. This highly relevant issue of relative rest was suggested by Lewis33 as an essential component of treatment for rotator cuff tendinopathy in a reactive stage. This will be referred to again later in this paper. In addition, it is possible that subacromial decompression is a placebo. Moseley et al.106 reported that 180 people with painful knee osteoarthritis randomized to either (i) arthroscopic lavage, or (ii) arthroscopic debridement or (iii) placebo surgery (skin incisions) reported the same improvement at 2 year follow-up. This strongly suggests that the benefit reported for people undergoing arthroscopic surgery for painful degenerative knees may be entirely attributable to the placebo effect. This is not the first time that the benefit of surgery has been attributed to placebo.107,108 Additionally, to reduce the economic burden on healthcare systems, it would be very appropriate to recommend an appropriately structured period of relative rest and a supervised and graduated exercise programme before surgery is considered as non-surgical care is at least of equivalent clinical benefit.16–18

Clinical diagnosis
A diagnosis of subacromial impingement syndrome is initially made on the basis of clinical tests. Neer5 introduced the Neer impingement sign and others have proposed other tests to confirm or exclude impingement under the acromion.109,110 The clinical tests are often supported by imaging investigations. However, the ability for clinical tests and imaging investigations to enable a clinician to confirm a diagnosis of subacromial impingement syndrome is
contentious, as imaging investigations have consistently demonstrated structural pathology in a high percentage of people without symptoms.

As discussed earlier there is a poor correlation between acromial radiological changes and symptoms. In a study of 96 people without shoulder symptoms 28% or those aged between 40 and 60 years and 54% of those aged above 60 years had MRI evidence of a partial or full thickness rotator cuff tear. Milgrom et al. reported in a study of 90 people (age range 30 to 99 without shoulder symptoms) that the incidence of full thickness rotator cuff tears identified by ultrasound increased with advancing age and that after the 5th decade approximately 50% of people had asymptomatic full thickness tears that did not affect function. In a study that compared 42 people with impingement syndrome with 31 age matched symptom free controls, Frost et al. reported that 55% of the symptomatic group and 52% of the asymptomatic group had evidence of rotator cuff pathology on imaging. They also reported that pathology related to age and did not correlate with symptoms. Professional baseball pitchers have been reported to pitch up to 165 km/hour and demonstrate internal rotation velocities during pitching of 6100 to 6940°/second. These examples of extremely high levels of function may be achieved even in the presence of structural pathology. Miniaci et al. reported that 14 professional baseball pitchers without shoulder symptoms demonstrated rotator cuff changes in their throwing (79%) and non-throwing (86%) shoulders and labral changes (79%) in both shoulders. Professional baseball pitchers and tennis players without symptoms demonstrated partial and full thickness tears (40%), glenohumeral joint effusions (90%) and excess subacromial fluid (48%) in their dominant shoulder and remained asymptomatic at a 5 year follow-up. In addition, the sourcil (eyebrow) sign observed radiologically as sclerosis on the under surface of the acromion and considered to be an indicator of rotator cuff pathology due to increased pressure (impingement) was found not to correlate with clinical signs of impingement, rotator cuff tears, or age, and did not aid diagnosis in 175 people with shoulder pain. Lewis et al. demonstrated that neovascularity may be present in both the symptomatic and asymptomatic shoulders of people diagnosed with unilateral rotator cuff pathology.

These imaging studies demonstrate that high percentages of people without symptoms will have evidence of structural failure and at present there is no clinical certainty that imaging abnormalities are the cause of the presenting impingement symptoms. Observation of structural pathology identified in radiographs, ultrasound, MRI and arthroscopy are frequently employed as the gold standard comparator in studies designed to test the diagnostic accuracy, sensitivity, specificity, positive and negative predictive values, and positive and negative likelihood ratios. However, if the gold standard is not robust (i.e. people without shoulder symptoms have structural failure) then a concomitant high percentage of false positives would not provide the confidence required by a clinician to make a diagnosis with any certainty.

This is of major concern as surgery may be recommended to people diagnosed with impingement syndrome based on clinical tests and supported by imaging findings that are currently incapable of conclusively confirming such a diagnosis. The consequence of this is that for a substantial percentage of people the surgery may be unnecessary, inappropriate and unwarranted. This is clearly highlighted by the number of studies that do not show added benefit of surgery over non-surgical care. In addition, surgery carries risks, such as infections, and is substantially more expensive. Therefore, an appropriate and defensible argument is that until a robust method of confirming a diagnosis is obtainable, and until clear evidence concerning the pathology is available, surgery should only be offered after an appropriate period of appropriate non-surgical care.

Tendinopathy

Shalabi et al. performed an MRI investigation of the Achilles tendon (n=44 from 22 people, 30 symptomatic and 14 asymptomatic tendons) immediately before and within 30 minutes of an intense bout of concentric (bilateral heel raises) and/or eccentric (6 sets of 15 repetitions) gastrosoleus exercises. They reported a 12% increase in tendon volume in the eccentrically loaded symptomatic Achilles tendons and a 17% increase in the concentrically loaded tendons (mixed symptomatic and asymptomatic) Achilles tendons. There was a 20% increase in tendon volume in the concentrically loaded asymptomatic Achilles tendons. Rats subject to a tendon overload programme have also demonstrated an increase in rotator cuff cross-sectional area. Increased rotator cuff tendon volume as a result of unaccustomed activity or activity at an intensity that surpasses the physiological limit of the tendon (which will be highly variable between and within individuals) may lead to increased upward pressure on the acromion and coracoacromial ligament. This increased strain in the ligament is a possible aetiological mechanism for acromial spur formation and as such the acromial osteophyte may not be the primary problem but secondary to the increased tendon...
volume. If the increased volume and resulting ligamentous strain occurs at a subclinical level from bursts of activity or sport a spur may develop over time but may remain asymptomatic over a lifetime and explain the poor correlation between acromial shape and symptoms.41–43

Overuse tendinopathies involving the lateral epicondyle, patellar, adductor and Achilles tendons may occur without impingement from external structures such as adjacent bony surfaces, and this may also be the case for the rotator cuff. It is possible that the external irritation accentuates the tendon failure 37–39,124 but it is unlikely that it is the primary cause. The failure is likely to be due to a combination of factors including: relative overload, genetics, nutritional and life style variables,32 and the rotator cuff tendon failure may be seen as a continuum of pathology.33 The initial symptomatic stage of the continuum of pathology has been termed a reactive tendon,33,125 which may be characterized by increased tenocyte numbers.126 Increased expression of the large negatively charged proteoglycan aggrecan is observed in painful overuse tendinopathy.127,128 Due to its negative charge aggrecan attracts and retains water, which explains the swelling observed in acute Achilles tendinopathy.50 The non-steroidal anti-inflammatory drug, ibuprofen, appears to inhibit the synthesis of aggrecan129 and may be an appropriate treatment at this stage. Additionally, glucocorticoids have been shown to inhibit tenocyte proliferation,130 which may explain the benefit ascribed to corticosteroid injections for the shoulder in some people.98 However as stated, the long term efficacy and potential detrimental effects of corticosteroid injections for the shoulder require on going investigation.

The other pathological stages associated with rotator cuff tendinopathy (disrepair and degeneration)33 may have an associated element of reactivity. When reactivity is present tendon thickening and swelling is possible. If this pathoaeiology is accurate and if the pathology is correctly explained by intrinsic tendon failure as a consequence of relative overload then it simply may be the swollen tendon pushing up and not the acromion pushing down that is the cause of the problem. If this hypothesis is correct, then an acromioplasty will not treat the primary problem (i.e. intrinsic tendon failure) or provide appropriate initial management for the condition. If relative rest and appropriate reloading strategies are principal factors in tendon rehabilitation it is possible that a major benefit of an acromioplasty is enforced relative rest.104,105 If this is correct, the associate expense, potential risks and lack of appropriately targeted treatment question its utility as a first line treatment option. 

Surgery versus non-surgical management
As mentioned, reports of 80–90% success following subacromial decompression for impingement have been published.100–102 When acromioplasty was compared with conservative care (physiotherapy exercises and pain relief) surgery appeared to be no more beneficial clinically at 6, 12 or 48 months.16–18 As elucidated earlier there is no certainty that the benefit relates directly to the stated aim of the surgery (i.e. removal of the acromion) and benefit may be derived from the bursectomy, the period of post-surgical relative rest, and potentially placebo. Relative rest is of relevance as Cook and Purdam125 in a generic model of overuse tendinopathy, have suggested that tendon load management and reduction in frequency and/or intensity of tendon load is important during the reactive phase. Relative rest may also be important in the reactive stage of rotator cuff tendinopathy.33 Relative rest may allow the tendon to attain relative homeostasis, by reducing the up regulation of tenocytes that may be characteristic of a reactive tendon and thereby reduce the associated swelling before a graduated and appropriately constructed rehabilitation programme is instigated. It may be possibly to enhance the exercise prescription that has been utilized in clinical trials by more effectively targeting the stage of the rotator cuff tendinopathy.33,131 In addition, consideration of the varying effects exercise may have on subacromial pressure46 is relevant. To further reduce upward humeral head translation and tendon compression, avoidance of internal rotation in the early stages of rehabilitation may be appropriate. Although uncertainty exists,132 it may be possible to enhance the effect of exercise by including manual therapy in the treatment package.133,134 These issues relating to rehabilitation need to be appropriately scrutinized through robust research investigations.

Conclusion
Subacromial impingement syndrome is considered by many to be the most common of the musculoskeletal conditions affecting the shoulder. It is based on a hypothesis that acromial irritation leads to external abrasion of the bursa and rotator cuff. Subacromial decompressive surgery aims to remove the source of this irritation. There however a body of evidence that suggests there is a lack of concordance regarding (i) the area of tendon pathology and acromial irritation, (ii) the shape of the acromion and symptoms, (iii) the proposal that the irritation leads to the development of tendinitis and bursitis, and (iv) imaging changes and symptoms and the development of the condition. In addition, there is no certainty that any benefit derived from the surgery is due to the removal of the acromion as research suggests that a
Subacromial impingement syndrome

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bursectomy in isolation may confer equivalent benefit. It is also possible that the benefit of surgery is that it simply enforces a sustained period of relative rest which may allow the involved tissues to achieve relative homeostasis. It is possible that pathology originates in the tendon and as such surgery does not address the primary problem. This view is strengthened by the findings of studies that have demonstrated no increased clinical benefit from surgery when compared with exercise, with exercise therapy being associated with a substantially reduced economic burden and less sick leave. Evidence based healthcare involves the integration of clinical expertise, patient values and best research evidence. To provide the research evidence required, surgeons performing acromioplasties need to demonstrate that it is the acromioplasty that is beneficial and not the enforced reduction in activity or the possibility of placebo. As there is little evidence for an acromial impingement model a more appropriate name may be ‘subacromial pain syndrome’. Moreover, surgery should only be considered after an appropriate period of appropriately structured conservative treatment.

Acknowledgements

Aspects of the information contained in this paper were presented as a key note lecture entitled ‘Subacromial impingement syndrome. A musculoskeletal condition or a clinical illusion?’ at the 11th International Conference of Shoulder and Elbow Surgeons, Edinburgh, Scotland, UK, 5–8 September 2010.

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Subacromial impingement syndrome

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Subacromial impingement syndrome

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