Narrative Review

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

Jeremy S Lewis[1,2,3,4]

[1]Therapy Department, Chelsea and Westminster Hospital, [2]Physiotherapy Department, St George's Hospital, [3]Musculoskeletal Service, Central London Community Healthcare, [4]St George's University of London, London, UK

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 pathoaetiology. 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

Correspondence to: Dr J S Lewis, Therapy Department, Chelsea and Westminster Hospital NHS Foundation Trust, 369 Fulham Road, London SW10 9NH, UK. Email: jeremy.lewis@chelwest.nhs.uk

DOI 10.1179/1743288X11Y.0000000027

Physical Therapy Reviews ptr7245.3d 18/7/11 17:44:22
The Charlesworth Group, Wakefield +44(0)1924 369598 - Rev 7.51n/W (Jan 20 2003)

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.[5] This hypothesis was supported by Bigliani *et al.*[14] who described three distinct morphological variations of acromial shape. Bigliani *et al.*[14] 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[4,5] presenting his model, surgeons were performing complete acromionectomies and lateral acromioplasties to alleviate the symptoms. Basing his argument on intra operative and cadaver observations, Neer[4,5] 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).[15] Ketola *et al.*[16] 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£3500. 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.*[16] 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,[16–18] 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,[5] 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.*[19] reported that 39 (91%) partial thickness tears in 43 athletes were on the inferior (articular or joint) side of the supraspinatus tendon with only 4 (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.*[20] reported that in a study of 249 cadavera, 13% ($n=33$) 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.*[21] 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.[22] This study did not find a correlation between anatomical bony variations and tendon failure and argued that mechanical abrasion may not play an important part

Physical Therapy Reviews ptr7245.3d 18/7/11 17:44:23
The Charlesworth Group, Wakefield +44(0)1924 369598 - Rev 7.51n/W (Jan 20 2003)

in the initial pathogenesis of degenerative rotator cuff tears. Ellman[23] 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[23] 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[10] 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.*[24] 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.[25] 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.[25] 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.[25] Supporting this finding, Bey *et al.*[26] 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,[25] Bey *et al.*[26] 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.[27–30] 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.[25,26,31] Recent reviews on the rotator cuff exploring these and related issues have been published.[32,33]

More than 70 years ago, Lindblom and Palmer[34] 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[35,36] 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

Physical Therapy Reviews ptr7245.3d 18/7/11 17:44:23
The Charlesworth Group, Wakefield +44(0)1924 369598 - Rev 7.51n/W (Jan 20 2003)

pathology. However, intrinsic overload in the form of downhill eccentric running for 4, 8 and 16 weeks did. The rats subject to overuse (running) demonstrated an increase in cross-sectional area and reduced maximal strain at all time points. A combination group (allograft and overload) lead to the greatest change, suggesting that compression potentiated overload even though compression alone did not produce pathology.[37–39] These findings suggest that external compression is insufficient to cause pathology unless there is a concomitant history of tendon overload, suggesting that the primary pathoaetiology occurs within the tendon. It is accepted that due to differences in morphology and biomechanics, caution is necessary with direct translation from animal studies. Evolution of the human upper limb may place biomechanical constraints on the modern shoulder which make it less capable of sustaining positions of elevation.[40] If this is the case, then work, recreational and sporting activities performed above 90° of elevation may selectively affect the weaker, more vulnerable joint side fibres without the need for acromial compression.

Acromial shape

Based on a study of 140 shoulders in 71 cadavers, Bigliani *et al.*[14] 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.[41] 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.*[41] 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.[42] Confirming this, after a study of 523 people undergoing arthroscopic or open shoulder surgery, Gill *et al.*[43] 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 varia>tion as described by Bigliani.[14,44] 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[45] 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.[46,47] 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.*[47] demonstrated *in vivo* (n=5) that tension in the coracoacromial ligament increased as the arm was abducted. In an additional study[48] 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[14,44] may not be inherited, but may result from increased strain in the ligament disproportionally 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.[49–53] Evidence for chronic strain exists, with free nerve endings and neovascularity observed in coracoacromial ligament samples from people undergoing subacromial decompression.[54] This suggests that the ligament may be a potential source of symptoms. The coracoacromial ligament limits superior translation of the humeral head[55–57] and as acromioplasty has

Physical Therapy Reviews ptr7245.3d 18/7/11 17:44:24
The Charlesworth Group, Wakefield +44(0)1924 369598 - Rev 7.51n/W (Jan 20 2003)

been associated with increased anterosuperior translation of the humeral head,[52,57–59] 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.[60] It is therefore surprising that the coracoacromial ligament, which provides a stabilization role by preventing superior translation of the humeral head,[61] 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[62–65] 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 syndrome.[63,66–68] 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[69–76] 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.[66,68] 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[45,70,77,78] 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;[69] however, these also require clinical validation.

Tendinitis

Implicit within the three-stage impingement model presented by Neer[4,5] 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.[79] Similarly, no inflammatory cells were identified in bursal specimens ($n=8$) also taken during surgery for rotator cuff tendinopathy.[80] 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.[81] There is distinct need for robust evidence from appropriately designed research to better understand if inflammation is part of the continuum of pathoaetiology of tendon and bursal pathology.[33] 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.[13,82] The SAB is innervated anteriorly by the lateral pectoral nerve and posteriorly by the suprascapular nerve.[83] The identification of mechanoreceptors and free nerve endings (Aδ and C) in bursal tissue suggests the SAB has a role in

Physical Therapy Reviews ptr7245.3d 18/7/11 17:44:24
The Charlesworth Group, Wakefield +44(0)1924 369598 - Rev 7.51n/W (Jan 20 2003)

proprioception zand nociception.[84] 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 injections[99] 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] Neer[5] 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 bursectomy 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 Lewis[33] 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 osteoarthrosis 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. Neer[5] 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

Physical Therapy Reviews ptr7245.3d 18/7/11 17:44:25
The Charlesworth Group, Wakefield +44(0)1924 369598 - Rev 7.51n/W (Jan 20 2003)

contentious,[69,111–113] 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.[41–43] 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.[114] Milgrom *et al.*[115] 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.*[116] 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.[117,118] These examples of extremely high levels of function may be achieved even in the presence of structural pathology. Miniaci *et al.*[119] 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.[120] In addition, the sourcil (eyebrow) sign observed radiologically as sclerosis on the under surface of the acromion and considered to be an indictor 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.[121] Lewis *et al.*[122] 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[114–116,119,122]) 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.[69,111,112] 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.[16–18] In addition, surgery carries risks, such as infections, and is substantially more expensive.[16,123] 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.*[50] 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.[37] 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

Physical Therapy Reviews ptr7245.3d 18/7/11 17:44:25
The Charlesworth Group, Wakefield +44(0)1924 369598 - Rev 7.51n/W (Jan 20 2003)

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 aggrecan[129] 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 pathoaetiology 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 Purdam[125] 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 pressure[46] 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 is 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

Physical Therapy Reviews ptr7245.3d 18/7/11 17:44:25
The Charlesworth Group, Wakefield +44(0)1924 369598 - Rev 7.51n/W (Jan 20 2003)

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.

References

1 Brox JI. Regional musculoskeletal conditions: shoulder pain. Best Pract Res Clin Rheumatol 2003;**17**:33–56.
2 van der Heijden GJ. Shoulder disorders: a state-of-the-art review. Baillieres Best Pract Res Clin Rheumatol 1999;**13**:287–309.
3 Taylor W. Musculoskeletal pain in the adult New Zealand population: prevalence and impact. NZ Med J 2005;**118**: U1629.
4 Neer CS, 2nd. Anterior acromioplasty for the chronic impingement syndrome in the shoulder: a preliminary report. J Bone Joint Surg Am 1972;**54**:41–50.
5 Neer CS, 2nd. Impingement lesions. Clin Orthop Relat Res 1983;**173**:70–7.
6 Bigliani LU, Levine WN. Subacromial impingement syndrome. J Bone Joint Surg Am 1997;**79**:1854–68.
7 Michener LA, Walsworth MK, Doukas WC, Murphy KP. Reliability and diagnostic accuracy of 5 physical examination tests and combination of tests for subacromial impingement. Arch Phys Med Rehabil 2009;**90**:1898–903.
8 Adam R, Todd, RB. Shoulder joint. In: Todd RB, editor. Cyclopaedia of anatomy and physiology. Vol. 4. London: Longman; 1852. p. 571–621.
9 Meyer A. The minuter anatomy of attrition lesions. J Bone Joint Surg Am 1931;**13**:341–60.
10 Codman E. The shoulder: rupture of the supraspinatus tendon and other lesions in or about the subacromial bursa. Boston: Thomas Todd Company; 1934. p.18–31, , 65–107.
11 Bosworth D. Analysis of 28 consecutive cases of incapacitating shoulder lesions radically explored and repaired. J Bone Joint Surg Am 1940;**22**:369–92.
12 McLaughlin H, Asherman EG. Lesions of the musculotendinous cuff of the shoulder. IV. Some observations based upon the results of surgical repair. J Bone Joint Surg Am 1951;**33**:76–86.
13 Diamond B. The obstructing acromion: underlying diseases, clinical development, and surgery. Springfield: Charles C. Thomas; 1964. p. 53–120.
14 Bigliani LU, Morrison DS, April EW. The morphology of the acromion and its relationship to rotator cuff tears. Ortho Trans 1986;**10**:228.
15 Vitale MA, Arons RR, Hurwitz S, Ahmad CS, Levine WN. The rising incidence of acromioplasty. J Bone Joint Surg Am 2010;**92**:1842–50.
16 Ketola S, Lehtinen J, Arnala I, Nissinen M, Westenius H, Sintonen H, *et al*. Does arthroscopic acromioplasty provide any additional value in the treatment of shoulder impingement syndrome? a two-year randomised controlled trial. J Bone Joint Surg Br 2009;**91**:1326–34.
17 Haahr JP, Ostergaard S, Dalsgaard J, Norup K, Frost P, Lausen S, *et al*. Exercises versus arthroscopic decompression in patients with subacromial impingement: a randomised, controlled study in 90 cases with a one year follow up. Ann Rheum Dis 2005;**64**:760–4.
18 Haahr JP, Andersen JH. Exercises may be as efficient as subacromial decompression in patients with subacromial stage II impingement: 4–8-years' follow-up in a prospective, randomized study. Scand J Rheumatol 2006;**35**:224–8.
19 Payne LZ, Altchek DW, Craig EV, Warren RF. Arthroscopic treatment of partial rotator cuff tears in young athletes. A preliminary report. Am J Sports Med 1997;**25**:299–305.
20 Fukuda H, Mikasa M, Yamanaka K. Incomplete thickness rotator cuff tears diagnosed by subacromial bursography. Clin Orthop Relat Res 1987;**223**:51–8.
21 Ozaki J, Fujimoto S, Nakagawa Y, Masuhara K, Tamai S. Tears of the rotator cuff of the shoulder associated with pathological changes in the acromion. A study in cadavera. J Bone Joint Surg Am 1988;**70**:1224–30.
22 Loehr JF, Uhthoff HK. The pathogenesis of degenerative rotator cuff tears. Orthop Trans 1987;**11**:237.
23 Ellman H. Diagnosis and treatment of incomplete rotator cuff tears. Clin Orthop Relat Res 1990;**254**:64–74.
24 Hashimoto T, Nobuhara K, Hamada T. Pathologic evidence of degeneration as a primary cause of rotator cuff tear. Clin Orthop Relat Res 2003;**415**:111–20.
25 Nakajima T, Rokuuma N, Hamada K, Tomatsu T, Fukuda H. Histological and biomechanical characteristics of the supraspinatus tendon. J Shoulder Elbow Surg 1994;**3**:79–87.
26 Bey MJ, Song HK, Wehrli FW, Soslowsky LJ. Intratendinous strain fields of the intact supraspinatus tendon: the effect of glenohumeral joint position and tendon region. J Orthop Res 2002;**20**:869–74.
27 Walch G, Boileau P, Noel E, Donell ST. Impingement of the deep surface of the supraspinatus tendon on the posterosuperior glenoid rim: an arthroscopic study. J Shoulder Elbow Surg 1992;**1**:238–45.
28 Jobe CM, Sidles JA. Evidence for a superior glenoid impingement upon the rotator cuff [abstract]. J Shoulder Elbow Surg 1993;**2**(Suppl):S19.
29 Riand N, Levigne C, Renaud E, Walch G. Results of derotational humeral osteotomy in posterior glenoid impingement. Am J Sports Med 1998;**26**:453–9.
30 Edelson G, Teitz C. Internal impingement in the shoulder. J Shoulder Elbow Surg 2000;**9**:308–15.
31 Nakajima T, Hughes RE, An KN. Effects of glenohumeral rotations and translations on supraspinatus tendon morphology. Clin Biomech (Bristol, Avon) 2004;**19**:579–85.
32 Lewis JS. Rotator cuff tendinopathy. Br J Sports Med 2009;**43**:236–41.
33 Lewis JS. Rotator cuff tendinopathy: a model for the continuum of pathology and related management. Br J Sports Med 2010;**44**:918–23.
34 Lindblom K, Palmer I. Ruptures of the tendon aponeurosis of the shoulder joint. Acta Chir Scand 1939;**82**:133–42.
35 Clark JM, Harryman DT, 2nd. Tendons, ligaments, and capsule of the rotator cuff. Gross and microscopic anatomy. J Bone Joint Surg Am 1992;**74**:713–25.
36 Fallon J, Blevins FT, Vogel K, Trotter J. Functional morphology of the supraspinatus tendon. J Orthop Res 2002; **20**:920–6.
37 Soslowsky LJ, Thomopoulos S, Tun S, Flanagan CL, Keefer CC, Mastaw J, *et al*. Neer Award 1999. Overuse activity injures the supraspinatus tendon in an animal model: a

histologic and biomechanical study. J Shoulder Elbow Surg 2000;**9**:79–84.
38 Perry SM, McIlhenny SE, Hoffman MC, Soslowsky LJ. Inflammatory and angiogenic mRNA levels are altered in a supraspinatus tendon overuse animal model. J Shoulder Elbow Surg 2005;**14**(Suppl):S79–83.
39 Carpenter JE, Flanagan CL, Thomopoulos S, Yian EH, Soslowsky LJ. The effects of overuse combined with intrinsic or extrinsic alterations in an animal model of rotator cuff tendinosis. Am J Sports Med 1998;**26**:801–7.
40 Lewis J, Green A, Yizhat Z, Pennington D. Subacromial impingement syndrome: Has evolution failed us? Physiother 2001;**87**:191–8.
41 Worland RL, Lee D, Orozco CG, SozaRex F, Keenan J. Correlation of age, acromial morphology, and rotator cuff tear pathology diagnosed by ultrasound in asymptomatic patients. J South Orthop Assoc 2003;**12**:23–6.
42 Snow M, Cheong D, Funk L. Subacromial impingement: is there correlation between symptoms, arthroscopic findings and outcomes? Shoulder Elbow 2009;**1**:89–92.
43 Gill TJ, McIrvin E, Kocher MS, Homa K, Mair SD, Hawkins RJ. The relative importance of acromial morphology and age with respect to rotator cuff pathology. J Shoulder Elbow Surg 2002;**11**:327–30.
44 Bigliani LU, Ticker JB, Flatow EL, Soslowsky LJ, Mow VC. The relationship of acromial architecture to rotator cuff disease. Clin Sports Med 1991;**10**:823–38.
45 Edelson JG, Taitz C. Anatomy of the coraco-acromial arch. Relation to degeneration of the acromion. J Bone Joint Surg Br 1992;**74**:589–94.
46 Werner CM, Blumenthal S, Curt A, Gerber C. Subacromial pressures in vivo and effects of selective experimental suprascapular nerve block. J Shoulder Elbow Surg 2006; **15**:319–23.
47 Chambler AF, Bull AM, Reilly P, Amis AA, Emery RJ. Coracoacromial ligament tension in vivo. J Shoulder Elbow Surg 2003;**12**:365–7.
48 Chambler AF, Pitsillides AA, Emery RJ. Acromial spur formation in patients with rotator cuff tears. J Shoulder Elbow Surg 2003;**12**:314–21.
49 Sarkar K, Taine W, Uhthoff HK. The ultrastructure of the coracoacromial ligament in patients with chronic impingement syndrome. Clin Orthop Relat Res 1990 **254**:49–54.
50 Shalabi A, Kristoffersen-Wiberg M, Aspelin P, Movin T. Immediate Achilles tendon response after strength training evaluated by MRI. Med Sci Sports Exerc 2004;**36**:1841–6.
51 Sharkey NA, Marder RA. The rotator cuff opposes superior translation of the humeral head. Am J Sports Med 1995;**23**:270–5.
52 Lazarus MD, Yung SW, Sidles JA, Harryman DT. Anterosuperior humeral head displacement: limitation by the coracoacromial arch. J Shoulder Elbow Surg 1996;**5**:S7.
53 Thompson WO, Debski RE, Boardman ND, 3rd, Taskiran E, Warner JJ, Fu FH, *et al*. A biomechanical analysis of rotator cuff deficiency in a cadaveric model. Am J Sports Med 1996;**24**:286–92.
54 Tamai M, Okajima S, Fushiki S, Hirasawa Y. Quantitative analysis of neural distribution in human coracoacromial ligaments. Clin Orthop Relat Res 2000;**373**:125–34.
55 Flatow EL, Wang VM, Kelkar R. The coracoacromial ligament passively restrains anterosuperior humeral subluxation in the rotator cuff deficient shoulder. Orthop Trans 1996;**21**:229.
56 Fagelman M, Sartori M, Freedman KB, Patwardhan AG, Carandang G, Marra G. Biomechanics of coracoacromial arch modification. J Shoulder Elbow Surg 2007;**16**:101–6.
57 Chen J, Luo CF, Luo ZP. Initiatory biomechanical study on humeral head migration after coracoacromial ligament cut. Arch Orthop Trauma Surg 2009;**129**:133–7.
58 Harryman DT, 2nd, Lazarus MD, Yung SW, Sidles JA, Matsen FA. Anterosuperior humeral displacement: limitation by the coracoacromial arch. J Shoulder Elbow Surg 1996;**5**:S29.
59 Su WR, Budoff JE, Luo ZP. The effect of coracoacromial ligament excision and acromioplasty on superior and anterosuperior glenohumeral stability. Arthroscopy 2009;**25**:13–8.
60 Samuelsson K, Andersson D, Karlsson J. Treatment of anterior cruciate ligament injuries with special reference to graft type and surgical technique: an assessment of randomized controlled trials. Arthroscopy 2009;**25**:1139–74.
61 Hockman DE, Lucas GL, Roth CA. Role of the coracoacromial ligament as restraint after shoulder hemiarthroplasty. Clin Orthop Relat Res 2004;**419**:80–2.
62 Ayub E. Posture and the upper quarter. In: Donatelli R, editor. Physical therapy of the shoulder. 2nd ed. Melbourne: Churchill Livingstone; 1991. p. 81–90.
63 Grimsby O, Gray J. Interrelation of the spine to the shoulder girdle. In: Donatelli R, editor.Physical therapy of the shoulder. 3rd ed. New York: Churchill Livingstone; 1997. p. 95–129.
64 Gray J, Grimsby O. Interrelationship of the spine, rib cage, and shoulder. In: Donatelli R, editor. Physical therapy of the shoulder. 4th ed. Edinburgh: Churchill Livingston; 2004. p. 133–85.
65 Ludewig PM, Cook TM. Alterations in shoulder kinematics and associated muscle activity in people with symptoms of shoulder impingement. Phys Ther 2000;**80**:276–91.
66 Kebaetse M, McClure P, Pratt NA. Thoracic position effect on shoulder range of motion, strength, and three-dimensional scapular kinematics. Arch Phys Med Rehabil 1999;**80**:945–50.
67 Sahrmann S. Diagnosis and treatment of movement impairment syndromes. London: Mosby; 2002. p. 193–261.
68 Bullock MP, Foster NE, Wright CC. Shoulder impingement: the effect of sitting posture on shoulder pain and range of motion. Man Ther 2005;**10**:28–37.
69 Lewis JS. Rotator cuff tendinopathy/subacromial impingement syndrome: is it time for a new method of assessment? Br J Sports Med 2009;**43**:259–64.
70 Lewis JS, Green A, Wright C. Subacromial impingement syndrome: the role of posture and muscle imbalance. J Shoulder Elbow Surg 2005;**14**:385–92.
71 Lewis JS, Valentine RE. The pectoralis minor length test: a study of the intra-rater reliability and diagnostic accuracy in subjects with and without shoulder symptoms. BMC Musculoskelet Disord 2007;**8**:64.
72 Grimmer K. An investigation of poor cervical resting posture. Aust J Physiother 1997;**43**:7–16.
73 McClure PW, Bialker J, Neff N, Williams G, Karduna A. Shoulder function and 3-dimensional kinematics in people with shoulder impingement syndrome before and after a 6-week exercise program. Phys Ther 2004;**84**:832–48.
74 Griegel-Morris P, Larson K, Mueller-Klaus K, Oatis CA. Incidence of common postural abnormalities in the cervical, shoulder, and thoracic regions and their association with pain in two age groups of healthy subjects. Phys Ther 1992;**72**:425–31.
75 Lederman E. The fall of the postural-structural-biomechanical model in manual and physical therapies: Exemplified by lower back pain. J Bodyw Mov Ther 2011;**15**:131–8.
76 Raine S, Twomey LT. Head and shoulder posture variations in 160 asymptomatic women and men. Arch Phys Med Rehabil 1997;**78**:1215–23.
77 Singer KP, Goh S. Anatomy of the thoracic spine. In: Giles LGF, Singer KP, editors. The clinical anatomy and management of thoracic spine pain. Vol 2. Oxford: Butterworth Heinemann; 2000. p. 17–33.
78 Lewis JS, Valentine RE. Clinical measurement of the thoracic kyphosis. A study of the intra-rater reliability in subjects with and without shoulder pain. BMC Musculoskelet Disord 2010;**11**:39.
79 Fukuda H, Hamada K, Yamanaka K. Pathology and pathogenesis of bursal-side rotator cuff tears viewed from en bloc histologic sections. Clin Orthop Relat Res 1990;**254**:75–80.
80 Sarkar K, Uhthoff HK. Ultrastructure of the subacromial bursa in painful shoulder syndromes. Virchows Arch A Pathol Anat Histopathol 1983;**400**:107–17.
81 Santavirta S, Konttinen YT, Antti-Poika I, Nordstrom D. Inflammation of the subacromial bursa in chronic shoulder pain. Arch Orthop Trauma Surg 1992;**111**:336–40.
82 Standring S. Gray's Anatomy. 39th ed. Edinburgh: Elsevier Churchill Livingstone; 2005. p. 817–49.
83 Aszmann OC, Dellon AL, Birely BT, McFarland EG. Innervation of the human shoulder joint and its implications for surgery. Clin Orthop Relat Res 1996;**330**:202–7.
84 Ide K, Shirai Y, Ito H, Ito H. Sensory nerve supply in the human subacromial bursa. J Shoulder Elbow Surg 1996;**5**:371–82.
85 Gotoh M, Hamada K, Yamakawa H, Yanagisawa K, Nakamura M, Yamazaki H, *et al*. Interleukin-1-induced subacromial synovitis and shoulder pain in rotator cuff diseases. Rheumatology (Oxford) 2001;**40**:995–1001.

86 Gotoh M, Hamada K, Yamakawa H, Inoue A, Fukuda H. Increased substance P in subacromial bursa and shoulder pain in rotator cuff diseases. J Orthop Res 1998;**16**:618–21.
87 Gotoh M, Hamada K, Yamakawa H, Yanagisawa K, Nakamura M, Yamazaki H, *et al*. Interleukin-1-induced glenohumeral synovitis and shoulder pain in rotator cuff diseases. J Orthop Res 2002;**20**:1365–71.
88 Sakai H, Fujita K, Sakai Y, Mizuno K. Immunolocalization of cytokines and growth factors in subacromial bursa of rotator cuff tear patients. Kobe J Med Sci 2001;**47**:25–34.
89 Yanagisawa K, Hamada K, Gotoh M, TokunagaT, Oshika Y, Tomisawa M, *et al*. Vascular endothelial growth factor (VEGF) expression in the subacromial bursa is increased in patients with impingement syndrome. J Orthop Res 2001;**19**:448–55.
90 Hyvonen P, Melkko J, Lehto VP, Jalovaara P. Involvement of the subacromial bursa in impingement syndrome of the shoulder as judged by expression of tenascin-C and histopathology. J Bone Joint Surg Br 2003;**85**:299–305.
91 Voloshin I, Gelinas J, Maloney MD, O'Keefe RJ, Bigliani LU, Blaine TA. Proinflammatory cytokines and metalloproteases are expressed in the subacromial bursa in patients with rotator cuff disease. Arthroscopy 2005;**21**:1076.
92 Henkus HE, de Witte PB, Nelissen RG, Brand R, van Arkel ER. Bursectomy compared with acromioplasty in the management of subacromial impingement syndrome: a prospective randomised study. J Bone Joint Surg Br 2009;**91**:504–10.
93 Henkus HE, Cobben LP, Coerkamp EG, Nelissen RG, van Arkel ER. The accuracy of subacromial injections: a prospective randomized magnetic resonance imaging study. Arthroscopy 2006;**22**:277–82.
94 Naredo E, Cabero F, Beneyto P, Cruz A, Mondejar B, Uson J, *et al*. A randomized comparative study of short term response to blind injection versus sonographic-guided injection of local corticosteroids in patients with painful shoulder. J Rheumatol 2004;**31**:308–14.
95 Chen MJ, Lew HL, Hsu TC, Tsai WC, Lin WC, Tang SF, *et al*. Ultrasound-guided shoulder injections in the treatment of subacromial bursitis. Am J Phys Med Rehabil 2006;**85**:31–5.
96 Alvarez CM, Litchfield R, Jackowski D, Griffin S, Kirkley A. A prospective, double-blind, randomized clinical trial comparing subacromial injection of betamethasone and xylocaine to xylocaine alone in chronic rotator cuff tendinosis. Am J Sports Med 2005;**33**:255–62.
97 Akgun K, Birtane M, Akarirmak U. Is local subacromial corticosteroid injection beneficial in subacromial impingement syndrome? Clin Rheumatol 2004;**23**:496–500.
98 Plafki C, Steffen R, Willburger RE, Wittenberg RH. Local anaesthetic injection with and without corticosteroids for subacromial impingement syndrome. Int Orthop 2000;**24**:40–2.
99 Ekeberg OM, Bautz-Holter E, Tveita EK, Juel NG, Kvalheim S, Brox JI. Subacromial ultrasound guided or systemic steroid injection for rotator cuff disease: randomised double blind study. BMJ 2009;**338**:273–6.
100 Burns TP, Turba JE. Arthroscopic treatment of shoulder impingement in athletes. Am J Sports Med 1992;**20**:13–6.
101 Checroun AJ, Dennis MG, Zuckerman JD. Open versus arthroscopic decompression for subacromial impingement. A comprehensive review of the literature from the last 25 years. Bull Hosp Jt Dis 1998;**57**:145–51.
102 Ellman H, Kay SP. Arthroscopic subacromial decompression for chronic impingement. Two- to five-year results. J Bone Joint Surg Br 1991;**73**:395–8.
103 Chin PY, Sperling JW, Cofield RH, Stuart MJ, Crownhart BS. Anterior acromioplasty for the shoulder impingement syndrome: long-term outcome. J Shoulder Elbow Surg 2007;**16**:697–700.
104 McClelland D, Paxinos A, Dodenhoff RM. Rate of return to work and driving following arthroscopic subacromial decompression. ANZ J Surg 2005;**75**:747–9.
105 Charalambous CP, Sahu A, Alvi F, Batra S, Gullett TK, Ravenscroft M. Return to work and driving following arthroscopic subacromial decompression and acromio-clavicular joint excision. Shoulder Elbow 2010;**2**:83–6.
106 Moseley JB, O'Malley K, Petersen NJ, Menke TJ, Brody BA, Kuykendall DH, *et al*. A controlled trial of arthroscopic surgery for osteoarthritis of the knee. N Engl J Med 2002;**347**:81–8.
107 Dimond EG, Kittle CF, Crockett JE. Comparison of internal mammary artery ligation and sham operation for angina pectoris. Am J Cardiol 1960;**5**:483–6.
108 Cobb LA, Thomas GI, Dillard DH, Merendino KA, Bruce RA. An evaluation of internal-mammary-artery ligation by a double-blind technic. N Engl J Med 1959;**260**:1115–8.
109 Hawkins RJ, Kennedy JC. Impingement syndrome in athletes. Am J Sports Med 1980;**8**:151–8.
110 Zaslav KR. Internal rotation resistance strength test: a new diagnostic test to differentiate intra-articular pathology from outlet (Neer) impingement syndrome in the shoulder. J Shoulder Elbow Surg 2001;**10**:23–7.
111 Lewis JS, Tennent TD. How effective are diagnostic tests for the assessment of rotator cuff disease of the shoulder? In: MacAuley D, Best TM, editors. Evidenced based sports medicine. 2nd ed. London: Blackwell Publishing; 2007. p. 327–59.
112 Hegedus EJ, Goode A, Campbell S, Morin A, Tamaddoni M, Moorman CT, 3rd, *et al*. Physical examination tests of the shoulder: a systematic review with meta-analysis of individual tests. Br J Sports Med 2008;**42**:80–92.
113 Schellingerhout JM, Verhagen AP, Thomas S, Koes BW. Lack of uniformity in diagnostic labeling of shoulder pain: time for a different approach. Man Ther 2008;**13**:478–83.
114 Sher JS, Uribe JW, Posada A, Murphy BJ, Zlatkin MB. Abnormal findings on magnetic resonance images of asymptomatic shoulders. J Bone Joint Surg Am 1995;**77**:10–5.
115 Milgrom C, Schaffler M, Gilbert S, van Holsbeeck M. Rotator-cuff changes in asymptomatic adults. The effect of age, hand dominance and gender. J Bone Joint Surg Br 1995;**77**:296–8.
116 Frost P, Andersen JH, Lundorf E. Is supraspinatus pathology as defined by magnetic resonance imaging associated with clinical sign of shoulder impingement? J Shoulder Elbow Surg 1999;**8**:565–8.
117 Dillman CJ, Fleisig GS, Andrews JR. Biomechanics of pitching with emphasis upon shoulder kinematics. J Orthop Sports Phys Ther 1993;**18**:402–8.
118 Feltner M. Three-dimensional interactions in a two-segment kinetic chain. Part II: application to the throwing arm in baseball pitching. Int J Sports Biomech 1989;**5**:420–50.
119 Miniaci A, Mascia AT, Salonen DC, Becker EJ. Magnetic resonance imaging of the shoulder in asymptomatic professional baseball pitchers. Am J Sports Med 2002;**30**:66–73.
120 Connor PM, Banks DM, Tyson AB, Coumas JS, D'Alessandro DF. Magnetic resonance imaging of the asymptomatic shoulder of overhead athletes: a 5-year follow-up study. Am J Sports Med 2003;**31**:724–7.
121 Smith C, Deans V, Drew S. The sourcil sign: a useful finding on plain x-ray? Shoulder Elbow 2010;**2**:9–12.
122 Lewis JS, Raza SA, Pilcher J, Heron C, Poloniecki JD. The prevalence of neovascularity in patients clinically diagnosed with rotator cuff tendinopathy. BMC Musculoskelet Disord 2009;**10**:163.
123 Brox JI, Staff PH, Ljunggren AE, Brevik JI. Arthroscopic surgery compared with supervised exercises in patients with rotator cuff disease (stage II impingement syndrome). BMJ 1993;**9**:899–903.
124 Soslowsky LJ, Thomopoulos S, Esmail A, Flanagan CL, Iannotti JP, Williamson JD, 3rd, *et al*. Rotator cuff tendinosis in an animal model: role of extrinsic and overuse factors. Ann Biomed Eng 2002;**30**:1057–63.
125 Cook JL, Purdam CR. Is tendon pathology a continuum? A pathology model to explain the clinical presentation of load-induced tendinopathy. Br J Sports Med 2009;**43**:409–16.
126 Scott A, Cook JL, Hart DA, Walker DC, Duronio V, Khan KM. Tenocyte responses to mechanical loading in vivo: a role for local insulin-like growth factor 1 signaling in early tendinosis in rats. Arthritis Rheum 2007;**56**:871–81.
127 Corps AN, Robinson AH, Movin T, Costa ML, Hazleman BL, Riley GP. Increased expression of aggrecan and biglycan mRNA in Achilles tendinopathy. Rheumatology (Oxford) 2006;**45**:291–4.
128 Riley G. Tendinopathy–from basic science to treatment. Nat Clin Pract Rheumatol 2008;**4**:82–9.
129 Tsai WC, Tang FT, Hsu CC, Hsu YH, Pang JH, Shiue CC. Ibuprofen inhibition of tendon cell proliferation and upregulation of the cyclin kinase inhibitor p21CIP1. J Orthop Res 2004;**22**:586–91.

Physical Therapy Reviews ptr7245.3d 18/7/11 17:44:27
The Charlesworth Group, Wakefield +44(0)1924 369598 - Rev 7.51n/W (Jan 20 2003)

130 Scutt N, Rolf CG, Scutt A. Glucocorticoids inhibit tenocyte proliferation and Tendon progenitor cell recruitment. J Orthop Res 2006;**24**:173–82.
131 Ainsworth R, Lewis JS, Conboy V. A prospective randomized placebo controlled clinical trial of a rehabilitation programme for patients with a diagnosis of massive rotator cuff tears of the shoulder. Shoulder Elbow 2009;**1**:55–60.
132 Yiasemides R, Halaki M, Cathers I, Ginn KA. Does passive mobilization of shoulder region joints provide additional benefit over advice and exercise alone for people who have shoulder pain and minimal movement restriction? A randomized controlled trial. Phys Ther 2011;**91**:178–89.
133 Bang MD, Deyle GD. Comparison of supervised exercise with and without manual physical therapy for patients with shoulder impingement syndrome. J Orthop Sports Phys Ther 2000;**30**:126–37.
134 McClatchie L, Laprade J, Martin S, Jaglal SB, Richardson D, Agur A. Mobilizations of the asymptomatic cervical spine can reduce signs of shoulder dysfunction in adults. Man Ther 2009;**14**:369–74.

Physical Therapy Reviews ptr7245.3d 18/7/11 17:44:28
The Charlesworth Group, Wakefield +44(0)1924 369598 - Rev 7.51n/W (Jan 20 2003)