

S REVIEW ARTICLE

Current concepts in subacromial impingement and the role of acromioplasty

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ABSTRACT

The treatment of subacromial impingement syndrome remains a controversial entity among both primary care physicians and orthopaedic surgeons. The initial management of this disorder is usually conservative with directed physiotherapy, subacromial corticosteroid injections and nonsteroidal anti-inflammatories representing the mainstays of treatment. Surgery in the form of arthroscopic bursectomy or acromioplasty is reserved for those patients who fail at least 6 months of conservative management. Traditionally, there have been few high-level studies to guide the practitioner concerning when conservative treatment has failed, which patients are candidates for surgical intervention or which surgical intervention may be most appropriate for a particular patient. However, recent studies have improved our knowledge base of the pathophysiology underlying this disorder and they provide some useful guidance on the conservative and operative management of this complex problem.

INTRODUCTION

Background

The subacromial space is bordered by the rotator cuff inferiorly, the coracoacromial (CA) ligament anteriorly, the acromion superiorly and the acromioclavicular (AC) joint medially. Pathology involving any of these structures can decrease the volume of this space and lead to impingement syndrome. This syndrome first gained notoriety in 1972 when Neer described 46 patients who were treated successfully with anterior and undersurface acromial resection along with the attached CA ligament [1]. This treatment supplanted previous, highly invasive treatments, such as lateral, subtotal or even total acromionectomy. Neer subsequently described three separate gradations of this impingement syndrome. Stage I denotes oedema and haemorrhage in the bursa. Stage II denotes fibrosis and tendinitis within the bursa and rotator cuff. Stage III denotes partial- and full-thickness tearing and degeneration of the rotator cuff tendon [2]. The focus of this review addresses Neer stage II, or classic, impingement syndrome.

Epidemiology

The overall incidence of shoulder pain in the general population is unclear. A systematic review of the literature and analysis of 18 epidemiological studies estimated the lifetime prevalence of shoulder pain ranging from 6.7% to 66.7%, notably with an

increasing prevalence being associated with increasing age [3]. In primary health care, subacromial impingement is considered the second most common complaint, trailing only back and neck pain, and accounting for 44% to 65% of all shoulder complaints [4].

Incidence of acromioplasty

When first described in 1972, anterior acromioplasty successfully treated 50 shoulders in 46 patients over a 5-year period [1]. Subsequent to this initial description, acromioplasty has been on a steady rise, with relatively recent precipitous climbs. A review of the Rochester Epidemiology Project servicing Olmstead County, Minnesota, USA demonstrated an almost six-fold increase in the incidence of acromioplasty, from 3.3 per 100,000 between 1980 and 1985 to 19.0 per 100,000 between 2000 and 2005 [5]. Similarly, a review of the New York Statewide Planning and Research Cooperative System ambulatory surgery database showed a 254% increase in the volume of acromioplasties from 1999 to 2008, representing an increase from 30.0 to 101.9 per 100,000. A review of the American Board of Orthopaedic Surgery database for Part 2 candidates demonstrated a 142% increase in acromioplasty (Current Procedural Terminology codes 23130 and 29826) from 1998 to 2008. This represented an increase from a mean of 2.6 to a mean of 6.3 per candidate [6]. The reasons for these increases are likely multifactorial, secondary not to only an increased recognition

for impingement syndrome pathology, but also to other patient and surgeon factors.

Aetiology

Contributing mechanisms for impingement can be divided into three categories: intrinsic, extrinsic and biological. Classically, Neer described an extrinsic mechanism for impingement, stemming from compression of the underlying rotator cuff by the anterolateral acromion and CA ligament arch [1]. Intrinsic processes related to ageing, decreased vascularity, systemic disease and overuse/overload may compromise rotator cuff tendon integrity. Other shoulder pathologies, such as abnormal glenohumeral translation, scapular protraction, posterior capsular tightness and scapular stabilizer weakness, can contribute to impingement [7]. Lastly, recent studies have focused on the altered biological environment of the subacromial bursa and the up-regulation of inflammatory mediators. Optimal treatment for impingement syndrome involves not only the correct identification of the underlying etiology, but also a reversal of this inflammatory cascade.

Extrinsic mechanisms

Three basic types of acromial morphology have been described based on the dissection of 140 shoulders in 71 cadavers. Flat (type I) occurred in 17%, curved (type II) occurred in 43% and hooked (type III) occurred in 40%. Interestingly, type III acromia comprised 70% of shoulders with complete rotator cuff tears. These morphological differences can best be identified on scapular Y or supraspinatus outlet radiographs [8]. An additional anterolateral acromial protuberance, independent of morphology and best seen on axillary radiographs, was identified in 113 of 165 (68%) patients with impingement syndrome who were undergoing shoulder arthroscopy. Although acromial morphology remains independent of age, this anterolateral protuberance is considered to represent an age-related reactive process creating or exacerbating impingement syndrome [9].

Pathological changes within the CA ligament may also contribute to impingement. In a comparison of 20 cadaveric shoulders (10 normal and 10 with rotator cuff tears), statistically significant decreases in strain and modulus of elasticity were observed in the lateral band of the CA ligament in the cuff tear shoulders. Geometrically, the length and thickness of these rotator cuff tear CA ligaments were also significantly increased [10]. Histologically, the CA ligament insertion underwent hypertrophic fibrocartilaginous proliferation, whereas the undersurface of the acromion remained normal in 50 patients who were treated for impingement syndrome [11]. The temporal relationship of these CA ligament changes to the onset of impingement syndrome and pain remains unknown.

The 'impingement arc' has traditionally referred to the range of elevation from approximately 60° to 120°, at which contact between the rotator cuff and the CA ligament arch-acromial undersurface occurs [2,7]. However, several laboratory investigations demonstrated CA ligament and acromial contact with the rotator cuff in normal shoulders throughout a much wider range

of motion. In seven normal cadaver shoulders, a flexible force sensor demonstrated contact occurring between the rotator cuff tendons and the CA arch during all motions [12]. This finding is further supported by magnetic resonance imaging (MRI) investigations of normal shoulders demonstrating contact between the rotator cuff and the CA arch with the arm in a neutral, adducted position [13]. Although such a study was limited by examination of supine shoulders, the total body of evidence corroborates normal contact throughout a wide range of shoulder motion. The addition of osteophytes, AC joint arthrosis, acromial morphology and CA ligament pathology potentially produces continuous compression on the underlying rotator cuff.

Intrinsic mechanisms

The vascularity of the rotator cuff tendons and its role in impingement syndrome has been proposed to be associated with localized ischaemia and age-related degenerative changes. Codman first described a watershed area of decreased vascularity near the tendon insertion of the rotator cuff [14]. Subsequent studies further defined this natural zone of hypovascularity as extending from the musculotendinous junction laterally to approximately 5 mm from the tendon-bone insertion [15]. On the other hand, Neer noted increased haemorrhage and oedema in the tendons of impingement syndrome patients [2]. A recent systematic review of rotator cuff real-time *in vivo* vascularity studies supports an articular-sided watershed zone and a continuum of vascularity from Stage I (hypovascular) to Stage II (hypervascular) to Stage III (hypovascular). Additionally, it was concluded that the relationship between hypovascularity, age and degeneration has not clearly been elucidated by the current body of literature [16].

Rotator cuff atrophy, weakness and dysfunction can lead to uncoupling and pathological glenohumeral translation. When this occurs in the superior direction, a decreased acromiohumeral distance can lead to increased pressure within the subacromial space and subsequent impingement syndrome. Piezo-electric pressure transducer measurements in 14 patients with impingement syndrome compared to eight normal controls demonstrated increased pressure in the impingement group, with the highest pressures recorded anterolaterally underneath the acromion and additional significant increases with shoulder abduction [17]. After bursectomy, subacromial pressures as measured by pressure-sensitive measurement films under the acromion subsequently decreased from 1.43 MPa to 1.14 MPa [18]. Muscle imbalance, specifically of the internal and external rotators and adductors, may also play a role in rotator cuff impingement. Clinical and isokinetic testing of shoulders with impingement syndrome demonstrated weakness in adduction and both external and internal rotation compared to asymptomatic shoulders. Additionally, symptomatic patients demonstrated higher abduction: adduction and abduction: internal rotation strength ratios compared to the asymptomatic cohort [19]. Taken together, these findings suggest that the successful treatment of impingement syndrome requires restoration of muscle balance and rotator cuff coupling.

Table 1 Summary of the increased biochemical, genetic and immunological changes seen in the subacromial bursa in patients with impingement syndrome

Study	Up-regulated factors
Sakai et al. [20]	IL-1 β , TNF- α , TGF- β , FGF
Gotoh et al. [21,25]	IL-1, substance P
Yoshida et al. [22]	IL-8
Yanagisawa et al. [23]	VEGF
Blaine et al. [24]	TNF, IL-1 α , IL-1 β , IL-6, MMP-1, MMP-9, COX-1, COX-2
Hyvonen et al. [26]	Tenascin-C
Hyvonen et al. [26]	Increased type I and type III collagen
Tomonaga et al. [27]	Increased type I and type III collagen
Soifer et al. [28]	Increased number of bursal free nerve endings

COX, cyclooxygenase; FGF, fibroblast growth factor; IL, interleukin; MMP, matrix metalloproteinase; TGF, transforming growth factor; TNF, tumour necrosis factor; VEGF, vascular endothelial growth factor.

Biological mechanisms

Recent work has focused on the increased expression of inflammatory factors within the subacromial bursa environment. Increased expression of interleukin (IL)-1 β , tumour necrosis factor- α , transcription growth factor- β , fibroblast growth factor, IL-8, vascular endothelial growth factor, tenascin-C and substance P have all been demonstrated [20–25]. Increased collagen type I and type III production occurs within the bursa [26,27]. Other studies have noted an increased number of free nerve endings [28] (Table 1). Despite the increased knowledge of altered biology, the factors triggering this inflammatory cascade remain unknown, as well as how to definitively halt its progression.

In summary, it is likely that impingement syndrome results from extrinsic compression to an already constrained subacromial space combined with intrinsic rotator cuff dysfunction and pathological initiation of the inflammatory cascade within the subacromial bursa. The temporal relationship of these events is currently unknown, and successful treatment must likely address all three aetiologies.

DIAGNOSIS OF IMPINGEMENT SYNDROME

History

Impingement syndrome patients typically complain of the insidious onset of pain, often exacerbated by repetitive overhead activities or minor trauma. Pain is often dull and aching, with sharpness experienced in the upper ranges of motion and may be referred down the lateral aspect of the arm. Patients often complain of pain waking them or keeping them awake at night. A thorough history should include hand dominance, occupation, recreational activities and previous treatment, including medications, injections and physical therapy. Neurological symptoms, neck pain and pending worker's compensation claims should also be investigated.

Physical examination

A careful, detailed physical examination of the shoulder not only evaluates for impingement syndrome, but also rules out other aetiologies of shoulder pain. Inspection should begin with the shoulders exposed, noting scapular and shoulder positioning. Palpation for tenderness includes the AC joint for arthritis and the acromion for an irritable os acromiale. Scapular kinematics should be observed for asymmetry or winging. The glenohumeral joint should be evaluated for range of motion, crepitus, instability and rotator cuff strength. Full cervical spine and distal motor and sensory examination should be included.

Provocative manoeuvres are often used in the physical examination of the shoulder, with several receiving emphasis in impingement syndrome. The Neer sign describes increased pain with forward flexion with internal rotation [2]. The Hawkins–Kennedy sign describes increased pain with passive internal rotation in 90° of abduction [29]. The painful arc sign brings the arm forward in the plane of the scapula from adduction to full elevation and is considered positive if there is pain or painful catching from 60° to 120°. The Neer test describes the removal of pain in forward flexion with internal rotation after injection of lidocaine into the subacromial bursa [30].

Eight different provocative physical examination tests of the shoulder were studied in a cohort comprising 913 patients who underwent shoulder arthroscopy over an 11-year period. All patients had pre-operative evaluation within 1 month before surgery utilizing these eight different tests. Within the group, 359 patients had positive impingement tests, a history of pain in the deltoid region or radiating down the arm, and at least temporary resolution of the pain with a subacromial injection with local anaesthetic (positive Neer test). Of those, 72 patients had no evidence of rotator cuff disease at the time of arthroscopy and were determined to have subacromial impingement alone. Within this group, the Neer sign had the highest sensitivity at 85.7%, followed by the Hawkins–Kennedy sign at 75.7%, and the painful arc sign at 70.6%. No other test demonstrated > 50% sensitivity. Despite their higher sensitivity, the specificity of these three tests was below 50% [30]. A recent systematic review of physical examination tests used in the clinical examination of shoulder pain found only 17 'high-quality' studies (> 60% methods score) out of 714 studies reviewed. Additionally, the reviewers found generally poor intra- and/or inter-examiner reliability among those high-quality studies [31].

History and physical examination can often distinguish subacromial impingement from two other shoulder impingement syndromes: coracoid and internal impingement. Most commonly affecting overhead athletes, internal impingement results from posterior capsular tightness causing abnormal contact between the posterosuperior labrum and the articular undersurface of the rotator cuff when the arm is in the abducted, externally rotated position. Patients may complain of a 'dead arm' and the posterior capsular tightness manifests in an internal rotation loss of > 25° compared to the contralateral arm. Patients with coracoid impingement often complain of anterior shoulder pain, presumably secondary to pathological contact between the subscapularis and coracoid process. Tenderness at the tip of the coracoid and a

positive cross-body adduction test, as well as decreased distance between the coracoid and anterior humerus on axial imaging, helps confirm the diagnosis [32].

Imaging

Impingement syndrome is a clinical and not a radiographic diagnosis. Beginning with plain radiographs, imaging may show subtle signs of impingement. A true anteroposterior view of the glenohumeral joint (Grashey view) should be evaluated for glenohumeral arthritis and AC joint disease including inferior osteophytes, as well as other occult suggestions of impingement disease, such as greater tuberosity sclerosis or excrescence formation, decreased acromiohumeral distance [33] and increased acromial index [34]. The axillary view should be evaluated for presence of an os acromiale in addition to presence of an anterolateral protuberance [9]. Scapular-Y can be used to assess the size of the supraspinatus fossa and the acromial morphology [8].

The greatest utility of advanced imaging such as ultrasound, MRI and computed tomographic arthrography lies in diagnosis or exclusion of other aetiologies for shoulder pain, such as chondral lesions, biceps and labral pathology, as well as partial- or full-thickness rotator cuff tears. Imaging cannot independently or reliably be utilized for the diagnosis of impingement syndrome.

NON-OPERATIVE MANAGEMENT

Once impingement syndrome is diagnosed, treatment aims at relieving pain and correcting the underlying aetiology. Treatment aims to combat intrinsic changes and uncoupling weakness within the rotator cuff, as well as halt the inflammatory cascade leading to the increased production of patient pain. Non-operative or conservative management often begins with physical therapy coupled with oral nonsteroidal anti-inflammatory drugs (NSAIDs). Subacromial injection can be utilized to combat extreme flares and acute exacerbations of pain. The injection may allow brief relief to facilitate participation in a structured physical therapy programme.

Physical therapy

The positive effect of structured exercise on patients with impingement syndrome has been demonstrated by several randomized clinical trials in the literature and appears to be the most appropriate first line of treatment (Table 2). A comprehensive treatment consisting of exercises, hot packs, soft tissue mobilization and education combined with a supervised shoulder exercise programme outperformed comprehensive treatment alone when measured by a visual analogue scale (VAS) for pain in a small group of 14 patients measured at only 24 hours [35]. In a group of 50 patients, an exercise programme plus manual physical therapy demonstrated similar decreases in VAS pain but increased strength at 8 weeks compared to an exercise programme alone at 2 months [36]. In the two largest studies, each with 60 patients, comparison of a home exercise programme with formal physical therapy demonstrated improved VAS pain and Constant scores at 3 months compared to a functional brace, with no statistically significant differences [37],

and progressive resistance training resulted in significant improvement in the 36-Item Short Form Health Survey, Disorders of the Arm, Shoulder, Hand (DASH) score and Cybex strength testing compared to no treatment [38]. A Cochrane Database review in 2003 concluded that, despite small sample sizes and variable methodological quality, exercise demonstrated effective short-term recovery and longer-term functional benefit in the setting of rotator cuff disease [39].

NSAIDs

These medications exert their effect on the cyclo-oxygenase pathway, which inhibits production of the downstream inflammatory mediator prostaglandin. They are available over-the-counter and stronger formulations are prescription only. Many patients present having used or tried a short-course of these medications and they have demonstrated improved efficacy in randomized, blinded clinical trials compared to placebo for shoulder impingement [40].

Corticosteroid injections

Injections into the subacromial bursa are often utilized to either manage an acute exacerbation of pain or complement a course of physical therapy. Transient hyperglycaemia, skin hypopigmentation, subcutaneous fat necrosis and infection are rare complications, with most injections being well-tolerated by patients. The location of the injection often varies based on provider preference and experience. Some guidance stems from a systematic review evaluating subacromial injection accuracy via imaging with the following combined accuracies: anterolateral 84% (range 75% to 88%), posterior 76% (range 75% to 76%), anteromedial 63% (one study), lateral 55% (range 29% to 70%) [41]. The choice of corticosteroid is also often dependent on physician or hospital system factors, with triamcinolone, methylprednisolone and betamethasone being commonly utilized (Table 3) with equivalent results [42]. Although some randomized, blinded clinical trials have demonstrated statistically significant improvements in pain and range of motion compared to placebo [42,43], others have not supported this result (Tables 4 and 5). Fifty-eight patients were randomized to subacromial betamethasone plus xylocaine injection or xylocaine injection alone, with both receiving the same guided therapy regimen. In this well-powered study of 58 patients, assessment of active range of motion, Neer sign, Western Ontario Rotator Cuff (WORC), DASH and American Shoulder and Elbow Surgeons (ASES) scores demonstrated an improvement of both groups over baseline, although no significant differences at all time points up to 6 months [44]. Another Cochrane Database review in 2003 demonstrated a small benefit for subacromial steroid injection over placebo in some trials, although no benefit over NSAIDs after pooling the results of three trials [48].

Alternative treatment regimens

Alternative treatment regimens for impingement syndrome have included ice, heat, massage, acupuncture, iontophoresis, phonophoresis and laser therapy. Few blinded, randomized clinical trials have been conducted and the results obtained have been mixed and should be interpreted with caution (Table 6). A recent

Table 2 Summary of randomized, blinded, prospective clinical trials comparing different physical therapy regimens in the treatment of impingement syndrome in the absence of rotator cuff tear

Study	Number (n)	Intervention	Outcome measures	Results
Conroy and Hayes [35]	14	Joint mobilization + comprehensive treatment (hot packs, stretching, education) versus comprehensive treatment alone	VAS pain, aROM, function	Improved VAS pain, but not aROM or function at 24 hours
Bang and Deyle [36]	52	Exercise alone versus manual physical therapy + exercise	Strength, VAS pain, FAQ	Significantly decreased pain, increased strength and function with manual therapy at 2 months
Walther et al. [37]	60	Home exercise versus functional brace versus physical therapy	VAS pain, Constant score	Nonstatistically significant reduction of pain in all 3 groups at 12 weeks
Lombardi et al. [38]	60	Progressive resistance training versus no treatment	SF-36, DASH, ROM, Cybex	Statistically significant improvement in pain and function at 2 months

aROM, active range of motion; DASH, Disabilities of the Arm, Shoulder and Hand; FAQ, functional assessment questionnaire; SF-36, 36-Item Short Form Health Survey; VAS, visual analogue scale.

Table 3 Equivalent dosages of corticosteroids commonly used in subacromial injections

Steroids	Equivalent dosages (mg)
Methylprednisolone (Depo-Medrol)	30
Triamcinolone (Kenalog)	30
Betamethasone (Celestone)	6

American Academy of Orthopaedic Surgeons (AAOS) task force determined that there was no conclusive evidence for or against the usage of alternative treatment modalities for patients with impingement syndrome in the absence of a full-thickness rotator cuff tear [54].

Despite these numerous clinical investigations involving a multitude of different treatment regimens, no clear guidelines exist regarding type, timing or duration of treatment. Predictors for the failure of non-operative treatment are also not clearly defined by the current body of impingement literature.

OPERATIVE MANAGEMENT

Indications

Following a 6-month programme of dedicated nonsurgical treatment and continued symptomatology, operative intervention can be discussed. Neer first demonstrated success with open anterior and lateral acromionectomy [1]. In 1991, Ellman first reported good or excellent results for up to 5 years in 65 (89%) patients who underwent arthroscopic subacromial decompression with acromioplasty [55].

Classically, subacromial decompression consists of three distinct steps: (1) bursectomy, (2) CA ligament recession and (3) anterolateral acromioplasty. Subacromial decompression with

acromioplasty provides several theoretical advantages. Technically, it improves the space to work in the subacromial space, which may be beneficial during rotator cuff repair. Mechanically, it offloads the rotator cuff by removing the prominent anterolateral acromion, which contacts the cuff during the 60° to 120° impingement arc. Biologically, the exposed bone surface provides a potential source for mesenchymal stem cells to potentiate rotator cuff healing. Lastly, neurologically, the required bursectomy removes the nociceptive pain fibres from the subacromial space.

Contraindications

Although no absolute contraindications exist, care should be taken in patients with massive or irreparable rotator cuff tears to avoid anterior humeral escape, in elderly or smaller patients with a diminutive acromion to avoid acromial fracture, and in those patients with asymptomatic os acromiale to avoid conversion to a painful condition.

Acromioplasty versus non-operative management

Several randomized controlled prospective trials have investigated the difference between acromioplasty and non-operative physical therapy or exercise treatment for impingement syndrome. At both 6 months and 30 months, 125 patients randomized to physical therapy and arthroscopic acromioplasty demonstrated improved Neer scores, decreased sick leave and decreased analgesic intake compared to placebo, although no statistically significant difference existed between the two groups [56,57]. Similarly, 90 patients randomized to physical therapy and arthroscopic acromioplasty demonstrated improvement in Constant, VAS pain and functional questionnaire scores at both 24 months and 48 months, again with no difference between treatment groups [58,59]. Another study reporting similar results in 134

Table 4 Summary of randomized, blinded, prospective clinical trials comparing different corticosteroid injections to placebo in the treatment of impingement syndrome in the absence of rotator cuff tear

Study	Number (n)	Intervention	Outcome measures	Results
Blair et al. [43]	40	Triamcinolone + lidocaine versus lidocaine alone	Pain, aROM	Steroid: 15/18, lidocaine: 4/19 improved at 30 weeks
Plafki et al. [42]	50	Bupivacaine versus triamcinolone versus dexamethasone	Patte score	Bupivacaine group aborted because of no effect, 50% resolution in other groups
Alvarez et al. [44]	58	Betamethasone + xylocaine versus xylocaine	WORC, ASES, DASH, aROM, Neer sign	No difference between groups at all time points, both groups improved from baseline
Ekeberg et al. [45]	106	Bursal triamcinolone versus systemic triamcinolone	SPADI, WORC	Small statistically significant difference favouring bursal injection

aROM, active range of motion; ASES, American Shoulder and Elbow Surgeons; DASH, Disabilities of the Arm, Shoulder and Hand; SPADI, Shoulder Pain and Disability Index; WORC, Western Ontario Rotator Cuff.

Table 5 Summary of randomized, blinded, prospective clinical trials comparing different treatment regimens to each other in the treatment of impingement syndrome in the absence of rotator cuff tear

Study	Number (n)	Intervention	Outcome measures	Results
Hardy et al. [46]	40	Indomethacin versus triamcinolone injection	Pain relief	19/30 (63%) complete relief, no difference between groups at 6 weeks
Adebajo et al. [40]	60	Oral diclofenac versus triamcinolone injection versus placebo	Pain, abduction, functional survey	Triamcinolone superior to diclofenac, both far superior to placebo at 4 weeks
Karthikeyan et al. [47]	58	Bursal tenoxicam versus bursal methylprednisolone	Constant score, DASH, Oxford Shoulder Score	Statistically significant greater improvement in Constant and DASH with steroids at 6 weeks

DASH, Disabilities of the Arm, Shoulder and Hand.

randomized patients at 24 months conducted a cost analysis revealing that the €1100 of additional cost for acromioplasty yielded equivalent clinical results to physical therapy patients [60]. The results are summarized in Table 7.

Open versus arthroscopic acromioplasty

Arthroscopic acromioplasty provides several advantages over open acromioplasty, such as the direct evaluation of the glenohumeral joint and any underlying pathology and maintenance of deltoid acromial origin integrity. Several studies have compared the results of open versus arthroscopic acromioplasty. A recent systematic literature review uncovered four Level I and one Level II well-designed, randomized, controlled prospective trials comparing open to arthroscopic acromioplasty along with appropriate patient follow-up. Although one study demonstrated significant flexion and abduction strength loss in the open group, the other studies demonstrated no significant difference in VAS pain scores, University of California at Los Angeles (UCLA) shoulder scores, range of motion and strength [65]. A recent meta-analysis demonstrated no significant differences between arthroscopic

and open acromioplasty in clinical outcomes or complications; however, the open procedure was associated with longer hospital stays and a greater length of time until return to work [66].

Acromioplasty versus bursectomy

Although the subacromial bursa may provide a gliding surface and potential blood supply to the underlying tendons, bursectomy for impingement syndrome removes some of the biological mediators of the inflammatory cascade and the increased nociceptive pain fibres of the inflamed tissue. A prospective trial randomizing 57 patients with impingement syndrome in the absence of a full-thickness rotator cuff tear to bursectomy plus acromioplasty compared to bursectomy alone demonstrated no significant difference in Constant score, Simple Shoulder Test and VAS pain at mean 30 months of follow-up [61]. It was found that the type of acromion and severity of symptoms had a greater influence on clinical outcome than type of treatment (Table 7).

Table 6 Summary of randomized, blinded, prospective clinical trials comparing the effectiveness of alternative treatments for impingement syndrome in the absence of rotator cuff tear

Study	Number (n)	Intervention	Outcome measures	Results
Johansson et al. [49]	85	Acupuncture versus ultrasound	Constant, AL, and UCLA scores	Larger improvement with acupuncture at 12 months
Paoloni et al. [50]	53 (57 shoulders)	Topical glyceryl trinitrate + home programme versus placebo + home programme	Pain, ROM, strength	Improved ROM, 50% complete resolution of pain at 24 weeks with patches
Aktas et al. [51]	46	Pulsed electromagnetic field + therapy versus sham + therapy	VAS pain, Constant score, disability questionnaire	Improvement in all measures, but no difference between groups at 3 weeks
Engebretsen et al. [52]	104	Extracorporeal shockwave treatment versus supervised exercise	SPADI	Statistically significant greater improvement with supervised exercise at 18 weeks
Yeldan et al. [53]	67	Low-level laser therapy + exercise versus sham + exercise	DASH, SDQ, dynamometer	No difference between groups at 3 weeks

AL, Adolfsson–Lysholm; DASH, Disabilities of the Arm, Shoulder and Hand; ROM, range of motion; SDQ, Shoulder Disability Questionnaire; SPADI, Shoulder Pain and Disability Index; UCLA, University of California at Los Angeles; VAS, visual analogue scale.

Acromioplasty with rotator cuff repair

During rotator cuff repair, CA ligament recession and acromioplasty not only improve the arthroscopic working area, but also provide theoretic advantages of exposure to mesenchymal stem cells from exposed cancellous bone and mechanical offloading of the repaired tendon. Despite these theoretic advantages, the current literature demonstrates no advantage to subacromial decompression including acromioplasty in the setting of rotator cuff repair. Three independent randomized trials comparing at least 40 patients in each group each demonstrated no significant differences in postoperative ASES scores at 15 months, Constant and DASH scores at 24 months, or WORC and ASES scores at 24 months between groups with and without subacromial decompression including acromioplasty in the setting of rotator cuff tears [62–64] (Table 7). The literature review of a recent AAOS task force supported this conclusion [54].

Long-term results

Both open and arthroscopic acromioplasty maintain good long-term results. Thirty-five open acromioplasty patients followed for a mean of 25 years demonstrated satisfaction in 88% and comparable Simple Shoulder Test and ASES scores with respect to the opposite, unaffected side. The rate of re-operation was 8.6% [67]. A comparison between prospective cohorts undergoing arthroscopic and open acromioplasty with a minimum follow-up of 12 years showed good or excellent results in 77% of patients. Both cohorts demonstrated improved UCLA scores, with a significantly increased change from baseline in the arthroscopic compared to the open group [68].

Complications

Although generally a safe, effective treatment method, arthroscopic subacromial decompression can have several complications in addition to those of general shoulder arthroscopy [69]. Technical error with a failure to remove sufficient anterolateral acromion, failure to recess enough CA ligament or failure to remove under-surface AC osteophytes can result in treatment failure. Extensive resection can result in deltoid detachment, acromial fracture [70], superomedial humeral head escape [71] or AC joint instability [72]. Heterotopic ossification is also a rare but reported complication [73].

Additionally, certain subgroups of patients have traditionally reported inferior results and must be managed carefully to avoid an adverse outcome. Os acromiale, a fibrocartilaginous tissue connection of the distal acromion to the scapular spine, is an anatomic variant described in 8% of the population and, when symptomatic, can commonly cause impingement syndrome. Arthroscopic subacromial decompression with acromioplasty has shown mixed results when treating impingement secondary to an unstable os acromiale [74]. Patients with pending workers' compensation claims have traditionally demonstrated poorer clinical results and an increased time for return to full-duty work [75].

Preferred technique

In our practice, the diagnosis of impingement syndrome combines a highly suggestive history and physical examination with radiographs and advanced imaging, ruling out other concomitant pathologies. Nonsurgical treatment is preferred, and our standard

Table 7 Summary of randomized, blinded, prospective clinical trials comparing the effectiveness of arthroscopic acromioplasty to other treatments for impingement syndrome in the absence of rotator cuff tear

Study	Number (n)	Intervention	Outcome measures	Results
Brox et al. [56,57]	125	Arthroscopic acromioplasty versus exercises versus placebo	Neer score, sick leave, analgesic intake	Significant improvement with acromioplasty and exercises, no difference between the two at 6 and 30 months
Haahr et al. [58,59]	90	Arthroscopic acromioplasty versus physiotherapy	Constant, VAS pain, functional questionnaire	Both groups improved, no difference between them at 12 and 48 months
Ketola et al. [60]	140	Arthroscopic acromioplasty versus supervised exercises	VAS pain	No clinically important difference at 24 months
Henkus et al. [61]	57	Arthroscopic bursectomy versus acromioplasty	Constant score, SST, VAS pain	No statistically significant difference at 30 months
Gartsman and O'Connor [62]	93	Rotator cuff repair + acromioplasty versus repair alone	ASES shoulder score	No difference at 12 months
Milano et al. [63]	80	Rotator cuff repair + acromioplasty versus repair alone	Constant score, DASH, Work-DASH	Subacromial decompression did not affect outcome at 6 months
MacDonald et al. [64]	86	Rotator cuff repair + acromioplasty versus repair alone	WORC, ASES shoulder score	No difference at 24 months

ASES, American Shoulder and Elbow Surgeons; DASH, Disabilities of the Arm, Shoulder and Hand; SST, Simple Shoulder Test; VAS, visual analogue scale; WORC, Western Ontario Rotator Cuff.

protocol addresses all three mechanisms simultaneously, via injection for biological, education for extrinsic and therapy for intrinsic mechanisms,

Once the diagnosis is made, patients are counselled regarding the aetiologies of impingement syndrome and are first recommended activity modification, specifically the avoidance of exacerbating overhead motions. A subacromial injection of a ropivacaine, lidocaine and corticosteroid mixture laterally is offered to temper the bursal inflammatory response. After allowing 7 days to 10 days of rest for the anti-inflammatory properties of the corticosteroid to take effect, therapy can begin. We prescribe a cuff-strengthening protocol with abduction less than 90° to restore the coupling action of the rotator cuff and to steer clear of the painful impingement arc. Patients continue therapy and return to the clinic in 6 weeks to 8 weeks for repeat evaluation.

Acromial morphology can help stratify patients who have continued pain. Despite repeat injections and diligent therapy, the extrinsic compression from a type III acromion may lead some patients to early failure of nonsurgical treatment. Patients with type I and II acromions are typically offered repeat injection at a minimum of 3 months from the previous injection and are recommended to continue therapy. A minimum of 6 months of failed non-operative treatment is preferred before operative subacromial decompression for isolated impingement syndrome.

Ultimately, accommodating a patient's unique set of occupational and athletic demands drives our treatment.

Subacromial decompression is typically performed with three successive operative steps: (1) adequate bursectomy, (2) CA ligament recession and (3) acromioplasty. The goal of operative acromioplasty is the conversion of a type II or III acromion into a flat, type I acromion. Additionally, lateral acromial overhang is resected back to deltoid tendinous origin and a flat acromial surface is achieved anteriorly from the medial AC joint to the anterolateral corner. With these principles, the average amount of bony resection varies significantly depending on acromial morphology; thus, standard bony resection amounts are not utilized.

Shoulder arthroscopy is performed using an indwelling, intrascapular extraarticular pain catheter with a pre-operative bolus. To avoid the morbidity and potential complications associated with general anaesthesia and intubation, intravenous sedation is preferred. The patient is placed in beach chair position with the arm prepped and connected to an arm-positioning device, thereby allowing rotational freedom throughout the procedure. Lateral decubitus positioning is another option and may be optimal in certain patients or situations.

A standard posterior viewing portal approximately 2 cm inferior and medial to the posterolateral acromial corner allows initial glenohumeral visualization. The anterior portal is localized via a

spinal needle through the rotator interval with slight positional changes depending on pathology. After completion of all intra-articular procedures, subacromial arthroscopy utilizes a lateral working portal. This portal is localized with a spinal needle lateral to the anterior third of the acromion and parallel to the acromial undersurface. Initial bursectomy is performed with an arthroscopic shaver, followed by CA ligament recession via electrocautery. Its attachment to the acromial undersurface is recessed beginning on the bone and proceeding laterally, anteriorly and then medially. Care is taken to preserve the white fibres of the deltoid origin because they guide the depth of bony resection.

Beginning laterally, a 5.5-mm arthroscopic burr is used to remove prominent anterolateral bone with care to preserve the aforementioned white deltoid fibre origin. Acromioplasty is finished by first switching the arthroscope to the lateral portal and the arthroscopic shaver to the posterior portal and then proceeding with a 'butcher-block' or 'cutting-block' technique to achieve a smooth, flat, type I acromion. The remaining arthroscopic procedures are then completed.

Postoperatively, isolated impingement patients without concomitant pathologies are placed in a bulky dressing for 48 hours and a simple sling for 2 days to 3 days. Once pain has decreased, they can begin light activity and supervised exercises as needed.

SUMMARY

Impingement syndrome is extremely common and yet considerable controversy exists regarding aetiology, diagnosis and treatment. Extrinsic, intrinsic, and biological aetiologies stem from anatomic compression, biomechanical imbalance and the bursal inflammatory cascade. History and physical examination help make the diagnosis, whereas additional imaging may rule out concomitant pathologies. The current body of impingement literature supports structured physical therapy as the mainstay of treatment, with NSAIDs and corticosteroid injections to facilitate therapy and control painful exacerbations. After 6 months of failed non-operative treatment, operative intervention can be considered, although the current literature does not conclusively demonstrate a clear benefit and the role of acromioplasty is not clear at this time. Additional high-level prospective, randomized, double-blind clinical trials are needed to further delineate this common clinical entity.

References

1. **Neer CS II.** Anterior acromioplasty for the chronic impingement syndrome in the shoulder: a preliminary report. *J Bone Joint Surg Am* 1972; 54A:41–50.
2. **Neer CS II.** Impingement lesions. *Clin Orthop* 1983; 173:70–7.
3. **Luime JJ, Koes BW, Hendriksen IJ, et al.** Prevalence and incidence of shoulder pain in the general population; a systematic review. *Scand J Rheumatol* 2004; 17:152–64.
4. **Michener LA, Walsworth MK, Burnet EN.** Effectiveness of rehabilitation for patients with subacromial impingement syndrome: a systematic review. *J Hand Ther* 2004; 17:152–64.
5. **Yu E, Cil A, Harmsen WS, et al.** Arthroscopy and the dramatic increase in frequency of anterior acromioplasty from 1980 to 2005: an epidemiologic study. *Arthroscopy* 2010; 26:142–7.
6. **Vitale MA, Arons RR, Hurwitz S, Ahmad CS, Levine WN.** The rising incidence of acromioplasty. *J Bone Joint Surg Am* 2010; 92:1842–50.
7. **Michener LA, McClure PW, Darduna AR.** Anatomical and biomechanical mechanisms of subacromial impingement syndrome. *Clin Biomech* 2003; 18:369–79.
8. **Bigliani LU, Morrison DS, April EW.** The morphology of the acromion and its relationship to rotator cuff disease. *Orthop Trans* 1986; 10:228.
9. **Gartsman GM.** Arthroscopic acromioplasty for lesions of the rotator cuff. *J Bone Joint Surg Am* 1990; 72:169–80.
10. **Soslowky LJ, An CH, Johnston SP, Carpenter JE.** Geometric and mechanical properties of the coracoacromial ligament and their relationship to rotator cuff disease. *Clin Orthop Relat Res* 1994; 304:10–17.
11. **Suenaga N, Minami A, Fukuda K, Kaneda K.** The correlation between bursoscopic and histologic findings of the acromion. *Arthroscopy* 2002; 18:16–20.
12. **Yamamoto N, Muraki T, Sperling JW, et al.** Contact between the coracoacromial arch and the rotator cuff tendons in nonpathologic situations: a cadaveric study. *J Shoulder Elbow Surg* 2010; 19:681–7.
13. **De Maeseeneer M, Van Roy P, Shahabpour M.** Normal MR imaging anatomy of the rotator cuff tendons, glenoid fossa, labrum, and ligaments of the shoulder. *Radiol Clin North Am* 2006; 44:479–8.
14. **Codman E.** *The shoulder: rupture of the supraspinatus tendon and other lesions in or about the subacromial bursa.* Boston, MA: Thomas Todd, 1934.
15. **Lohr JF, Uthoff HK.** The microvascular pattern of the supraspinatus tendon. *Clin Orthop Relat Res* 1990; 254:35–8.
16. **Hegedus EJ, Cook C, Brennan M, et al.** Vascularity and tendon pathology in the rotator cuff: a review of literature and implications of rehabilitation and surgery. *Br J Sports Med* 2010; 44:838–47.
17. **Hyvonen P, Lantto V, Jalovaara P.** Local pressures in the subacromial space. *Int Orthop* 2003; 27:373–7.
18. **Machida A, Sugamoto K, Miyamoto T, et al.** Adhesion of the subacromial bursa may cause subacromial impingement in patients with rotator cuff tears: pressure measurements in 18 patients. *Acta Orthop Scand* 2004; 75:1109–13.
19. **Burnham RS, May L, Nelson E, Steadward R, Reid DC.** Shoulder pain in wheelchair athletes: the role of muscle imbalance. *Am J Sports Med* 1993; 21:238–42.
20. **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.
21. **Gotoh M, Hamada K, Yamakawa H, et al.** Interleukin-1-induced glenohumeral synovitis and shoulder pain in rotator cuff diseases. *J Orthop Res* 2002; 20:1365–71.
22. **Yoshida M, Funasaki H, Kajitani K, Fujii K.** Pathologic gene expression in adhesive subacromial bursae of human shoulder. *Clin Orthop Relat Res* 2003; 412:57–64.
23. **Yanagisawa K, Hamada K, Gotoh 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.
24. **Blaine TA, Kim YS, Voloshin I, et al.** The molecular pathophysiology of subacromial bursitis in rotator cuff disease. *J Shoulder Elbow Surg* 2005; 14(1 Suppl S):845–95.
25. **Gotoh M, Hamada Y, 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.
26. **Hyvonen P, Melkko J, Lehto VP, Jalovaara P.** Involvement of the subacromial bursa in impingement syndrome of the shoulder as judged by expression of tensacin-C and histopathology. *J Bone Joint Surg Br* 2003; 85-B:299–305.
27. **Tomonaga A, Hamada K, Gotoh M, et al.** Expression of procollagen alpha 1 type III mRNA in rotator cuff tears. *Tokai J Exp Clin Med* 2000; 25:125–34.
28. **Soifer TB, Levy HJ, Soifer FM, et al.** Neurohistology of the subacromial space. *Arthroscopy* 1996; 12:182–6.
29. **Hawkins RJ, Kennedy JC.** Impingement syndrome in athletes. *Am J Sports Med* 1980; 8:151–8.

30. **Park HB, Yokota A, Gill HS, El Rassi G, McFarland EG.** Diagnostic accuracy of clinical tests for the different degrees of subacromial impingement. *J Bone Joint Surg Am* 2005; 87:1446–55.
31. **May S, Chance-Larsen K, Littlewood C, Lomas D, Saad M.** Reliability of physical examination tests used in the assessment of patients with shoulder problems: a systematic review. *Physiotherapy* 2010; 96:179–90.
32. **McFarland EG, Selhi HS, Keyurapan E.** Clinical evaluation of impingement: what to do and what works. *J Bone Joint Surg Am* 2006; 88:432–41.
33. **Saupe N, Pfirrmann CWA, Schmid MR, et al.** Association between rotator cuff abnormalities and reduced acromiohumeral distance. *AJR Am J Roentgenol* 2006; 187:376–82.
34. **Nyffler RW, Werner CML, Sukthankar A, Schmid MR, Gerber C.** Association of a large lateral extension of the acromion with rotator cuff tears. *J Bone Joint Surg Am* 2006; 88:800–5.
35. **Conroy DE, Hayes KW.** The effect of joint mobilization as a component of comprehensive treatment for primary shoulder impingement syndrome. *J Orthop Sports Phys Ther* 1998; 28:3–14.
36. **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.
37. **Walther M, Werner A, Stahlschmidt T, Woelfel R, Gohlke F.** The subacromial impingement syndrome of the shoulder treated by conventional physiotherapy, self-training, and a shoulder brace: results of a prospective, randomized study. *J Shoulder Elbow Surg* 2004; 13:417–23.
38. **Lombardi I Jr., Magri AG, Fleury AM, Da Silva AC, Natour J.** Progressive resistance training in patients with shoulder impingement syndrome: a randomized controlled trial. *Arthritis Rheum* 2008; 59:615–22.
39. **Green S, Buchbinder R, Hetrick S.** Physiotherapy interventions for shoulder pain. *Cochrane Database Syst Rev* 2003; 3:CD004258.
40. **Adebajo AO, Nash P, Hazleman BL.** A prospective double blind dummy placebo controlled study comparing triamcinolone hexacetonide injection with oral diclofenac 50mg TDS in patients with rotator cuff tendonitis. *J Rheumatol* 1990; 17:1207–10.
41. **Daley EL, Baja S, Bisson LJ, Cole BJ.** Improving injection accuracy of the elbow, knee, and shoulder: does injection site and imaging make a difference? A systematic review. *Am J Sports Med* 2011; 39:656–62.
42. **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.
43. **Blair B, Rokito AS, Cuomo F, Jarolem K, Zuckerman JD.** Efficacy of injections of corticosteroids for subacromial impingement syndrome. *J Bone Joint Surg Am* 1996; 78:1685–9.
44. **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.
45. **Ekeberg OM, Bautz-Holter E, Tveitå EK, et al.** Subacromial ultrasound guided or systemic steroid injection for rotator cuff disease: randomised double blind study. *Br Med J* 2009; 338:a3112.
46. **Hardy DC, Vogler JB III, White RH.** The shoulder impingement syndrome: prevalence of radiographic findings and correlation with response to therapy. *AJR Am J Roentgenol* 1986; 147:557–61.
47. **Karthikeyan S, Kwong HT, Upadhyay PK, et al.** A double-blind randomised controlled study comparing subacromial injection of tenoxicam or methylprednisolone in patients with subacromial impingement. *J Bone Joint Surg Br* 2010; 92:77–82.
48. **Buchbinder R, Green S, Youd JM.** Corticosteroid injections for shoulder pain. *Cochrane Database Syst Rev* 2003; 1:CD004016.
49. **Johansson KM, Adolfsson LE, Foldevi MO.** Effects of acupuncture versus ultrasound in patients with impingement syndrome: randomized clinical trial. *Phys Ther* 2005; 85:490–501.
50. **Paoloni JA, Appleyard RC, Nelson J, Murrell GA.** Topical glyceryl trinitrate application in the treatment of chronic supraspinatus tendinopathy: a randomized, double-blinded, placebo-controlled clinical trial. *Am J Sports Med* 2005; 33:806–13.
51. **Aktas I, Akgun K, Cakmak B.** Therapeutic effect of pulsed electromagnetic field in conservative treatment of subacromial impingement syndrome. *Clin Rheumatol* 2007; 26:1234–9.
52. **Engebretsen K, Grotle M, Bautz-Holter E, et al.** Radial extracorporeal shockwave treatment compared with supervised exercises in patients with subacromial pain syndrome: single blind randomised study. *Br Med J* 2009; 339:b3360.
53. **Yeldan I, Cetin E, Ozdincler AR.** The effectiveness of low-level laser therapy on shoulder function in subacromial impingement syndrome. *Disabil Rehabil* 2009; 31:935–40.
54. **Pedowitz RA, Yamaguchi K, Ahmad CS, et al.** Optimizing the management of rotator cuff problems. *J Am Acad Orthop Surg* 2011; 19:368–79.
55. **Ellman H, Kay SP.** Arthroscopic subacromial decompression for chronic impingement: two to five-year results. *J Bone Joint Surg Br* 1991; 73-B:395–8.
56. **Brox JI, Staff PH, Ljunggren AE, Brevik JI.** Arthroscopic surgery compared with supervised exercises in patients with rotator cuff disease (stage II impingement syndrome). *Br Med J* 1993; 307:899–903.
57. **Brox JI, Gjengedal E, Uppheim G, et al.** Arthroscopic surgery versus supervised exercises in patients with rotator cuff disease (stage II impingement syndrome): a prospective, randomized, controlled study in 125 patients with a 2 1/2-year follow-up. *J Shoulder Elbow Surg* 1999; 8:102–11.
58. **Haahr JP, Østergaard S, Dalsgaard J, 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.
59. **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.
60. **Ketola S, Lehtinen J, Arnala I, 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.
61. **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.
62. **Gartsman GM, O'Connor DP.** Arthroscopic rotator cuff repair with and without arthroscopic subacromial decompression: a prospective, randomized study of one-year outcomes. *J Shoulder Elbow Surg* 2004; 13:424–6.
63. **Milano G, Grasso A, Salvatore M, et al.** Arthroscopic rotator cuff repair with and without subacromial decompression: a prospective randomized study. *Arthroscopy* 2007; 23:81–8.
64. **MacDonald P, McRae S, Leiter J, Mascarenhas R, Lapner P.** Arthroscopic rotator cuff repair with and without acromioplasty in the treatment of full-thickness rotator cuff tears: a multicenter, randomized controlled trial. *J Bone Joint Surg Am* 2011; 93:1953–60.
65. **Barfield LC, Kuhn JE.** Arthroscopic versus open acromioplasty: a systematic review. *Clin Orthop Relat Res* 2007; 455:64–71.
66. **Davis AD, Kakar S, Moros C, et al.** Arthroscopic versus open acromioplasty: a meta-analysis. *Am J Sports Med* 2010; 38:613–8.
67. **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.
68. **Odenbring S, Wagner P, Atroshi I.** Long-term outcomes of arthroscopic acromioplasty for chronic shoulder impingement syndrome: a prospective cohort study with a minimum of 12 years' follow-up. *Arthroscopy* 2008; 24:1092–8.

69. **Marecek GS, Saltzman MD.** Complications in shoulder arthroscopy. *Orthopedics* 2010; 33:492–7.
70. **Coen MJ, Jobe CM, Pak K.** Acromial compromise with use of a transacromial portal; a biomechanical study. *J Shoulder Elbow Surg* 1995; 4:249–53.
71. **Wiley AM.** Superior humeral dislocation: A complication following decompression and debridement for rotator cuff tears. *Clin Orthop Relat Res* 1991; 263:135–41.
72. **Fischeer BW, Gross RM, McCarthy JA, Arroyo JS.** Incidence of acromioclavicular joint complications after arthroscopic subacromial decompression. *Arthroscopy* 1999; 15:241–8.
73. **Boynton MD, Enders TJ.** Severe heterotopic ossification after arthroscopic acromioplasty: a case report. *J Shoulder Elbow Surg* 1999; 8:495–7.
74. **Kurtz CA, Humble BJ, Rodosky MW, Sekiya JK.** Symptomatic os acromiale. *J Am Acad Orthop Surg* 2006; 14:12–19.
75. **Nicholson GP.** Arthroscopic acromioplasty: a comparison between workers' compensation and non-workers' compensation populations. *J Bone Joint Surg Am* 2003; 85:682–9.