# Subacromial Impingement Syndrome

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# 41.1 Subacromial Impingement

Fifty percent of the general population experience shoulder pain every year [1]. Subacromial impingement syndrome (SAIS) is the most common disorder of the shoulder, accounting for 44–65% of all complaints of shoulder pain [2].

In a Dutch study, the incidence of new cases of rotator cuff tendonitis in general practice was found to be around 3.2–4.2 per 1,000 personyears, and the corresponding incidence of shoulder pain (all causes) was 11.2 per 1,000 personyears [3]. Its prevalence is especially high in sports with overhead activity, such as swimming, volleyball, handball, and badminton. These overhead athletes have a high demand for optimal shoulder performance, and dynamic stability is required in order to prevent injury [4].

Shoulder impingement results from an "inflammation and degeneration of the anatomical structures in the region of the subacromial space" [5].

For many years, it has been thought that the anatomical basis was a mismatch between the structures in the subacromial space (Table 41.1).

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P. Consigliere, MD, MCh (Orth) Berkshire Independent Hospital and Ashford and St. Peter's Hospitals, Surrey, UK e-mail: paoloconsigliere@gmail.com Neer applied the phrase "impingement syndrome" in 1972 when he described the mechanism involved in this disorder [6]. It has been described as a chronic repetitive mechanical process in which the conjoint tendon of the rotator cuff undergoes repetitive compression and microtrauma as it passes under the coracoacromial arch [7]. As the arm is abducted or rotated, the subacromial space width changes and the cuff become increasingly compressed (Fig. 41.1a, b). The supraspinatus is in closest contact to the anterior inferior border of the acromion in 90° of abduction with 45° internal rotation [8].

In athletes where repetitive overhead activity is required, the act of throwing may subsequently lead to the pathological process outlined by Neer. Secondary impingement is usually associated with repetitive overhead activity resulting in glenohumeral instability [9, 10] (Table 41.2).

A recent study [13] focuses attention on the role of degeneration of the rotator cuff tendons, eventually giving rise to the development of tears.

A direct relationship between the anatomical substrate, functional load, and pain is not always explicitly present.

 Table 41.1
 Neer's classification: stages of subacromial impingement [33]

Stage 1: edema and hemorrhage, age <25, reversible Stage 2: fibrosis and tendinitis, age 25–40, recurrent pain with activity

Stage 3: bone spurs and tendon rupture, age >40, progressive disability

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Fig. 41.1 Anatomy of the subacromial space. (a) Humeral head, rotator cuff, subacromial bursa, acromion. (b) Impingement mechanism during abduction of the humerus

 
 Table 41.2
 Jobe's classification (1989): stages of subacromial impingement in athletes

Stage 1: pure impingement with no instability Stage 2: primary instability with capsular and labral injury with secondary impingement which can be internal impingement or subacromial Stage 3: primary instability because of generalized ligamentous laxity with secondary impingement Stage 4: pure instability and no impingement

The supraspinatus tendon is often the most involved tendon of the rotator cuff in shoulder impingement. It mainly derives its blood supply from the anterior circumflex humeral and suprascapular arteries. Within the supraspinatus tendon, near its insertion at the greater tuberosity, there is an avascular area also called "critical" zone. It is here that "impingement" usually occurs, and this zone has been found to increase in size with advancing age [14].

From the analysis of the recent literature [15], it seems likely that the imbalance and fatigue of the rotator cuff muscles (depressant action on the humeral head) lead to proximal migration of the humeral head during shoulder abduction/ rotations.

Although this is probably the most accredited theory [21], other authors focused their attention also on the cinematic of the scapula in the last years. Ludewing et al. studied the role of the periscapular muscles during shoulder abduction. An imbalance/fatigue of periscapular muscles (scapular dyskinesia) may play a role in shoulder impingement. In patients with SAIS, a late activation of the lower trapezius (LT) and the serratus anterior (SA) and an early recruitment of the upper trapezius fibers (UT) lead to a narrowing of the subacromial space during shoulder abduction [16, 17]. A review of the literature on these topics (Struyf et al.) confirmed that patients with shoulder impingement have a decreased upward scapular rotation, a decreased posterior tilt, and a decreased external rotation [19, 20], due to an imbalance of the periscapular muscles. J. Lewis, instead, studied the role of the posture in patients with SAIS. "Forward head and shoulder posture" (increased thoracic kyphosis and forward shoulder posture) can't be considered the cause of subacromial impingement, but patients with an altered posture experienced pain at a reduced range of movement (ROM) during abduction/forward flexion of the shoulder [18].

Outcomes of arthroscopic decompression in these patients are not reported in the literature as most of these patients respond to an appropriate rehabilitation program focused on recovery of the scapulothoracic rhythm and correction of posture. However, good outcomes are observed in patients in which physiotherapy failed (minimum 6 months of physiotherapy) to regain a balance in periscapular muscles and improve symptoms. To explain these positive results, the authors conjectured that a deafferentation of the subacromial area allows a successful proprioceptive reeducation of the shoulder movements with a modification of nociceptive stimuli (all the neurological causes of scapula dyskinesia must be excluded before arthroscopic subacromial decompression (ASD) is performed).

## 41.1.1 Diagnosis: Clinical

A recent systematic review concluded that several clinical shoulder tests have sufficient sensitivity but inadequate specificity [22]. Neer's sign and Hawkins' impingement tests, in particular, have been found useful in confirming SAIS but poor at ruling out pathology [23, 24]. Neer's and Hawkins' tests were found to have a sensitivity and specificity of 79% and 53% and 79% and 59%, respectively. No shoulder tests can by themselves confirm impingement [22].

A recent prospective study of patients admitted for surgery, however, found Neer's sign, the painful arc test, and the external rotation resistance test to be excellent screening tools to rule out SAIS [25].

The fundamental tests to rule out SAIS are:

- *Painful arc test*: The patient is instructed to actively elevate the arm in the scapular plane and then slowly reverse the motion. The test is considered positive if the patient has pain between 60 and 120° of abduction.
- *Neer's sign*: Subject is sitting. The scapula is fixed/stabilized in a depressed position, while the shoulder is maximally forwardly flexed.
- *Hawkins' test*: Subject is sitting on the examination table. The shoulder is placed in 90° of forward flexion and passively internally rotated as far as possible (elbow flexed 90°).
- *Yocum test:* The hand of the painful shoulder is placed on the opposite shoulder; the arm is flexed at 90° in the coronal plane. The elbow is pushed downward, while the patient is making active resistance. This is another test with high sensitivity but low specificity.
- *Jobe's sign*: The examiner passively elevates the patient's shoulder to 90° of elevation with internal rotation. The examiner then applies a downward pressure against the arm. A posi-

tive test is the provocation of pain and abnormal weakness.

- *External rotation resistance test*: Shoulder pain during forced external rotation of the shoulder against resistance (arm adducted, elbow flexed 90°). A modification of this test can also be used; the lack test is performed bringing the forearm of the patient to 60° of external rotation, asking the patient to hold the position. The test is positive if the patient can't hold the position. It reveals a weakness/ tear of the posterosuperior RC.
- *Posterior impingement sign*: Patient with the shoulder in 90° of abduction and elbow in 90° of flexion. Examiner stabilizes elbow and applies external rotation (ER) force to maximum ER.
- *Coracoid impingement test*: Pain directly over the coracoid with arm passively adducted across chest (distinguish from acromioclavicular joint (ACJ) scarf test in which the pain is felt in the ACJ).

Visual assessment of the ROM is appropriate only for distinguishing between the affected and the contralateral side. Even when using a goniometer, which can increase the reliability of the measurements, the measurement error remains high; this is useful to detect capsular stiffness, particularly of the posteroinferior area, that might generate upward translation of the humeral head and secondary subacromial impingement.

In selecting an outcome instrument, it is important for the scale to have been validated in the language of the patients and the examiner. The simple shoulder test (SST) and the Oxford Shoulder Score (OSS) are instruments with relatively few questions and are easy to use. The Dutch Shoulder Disability Ouestionnaire (DSDQ) with 16 questions is a medium-length questionnaire and is also easy to use [13]. The Constant-Murley Shoulder Outcome Score (CS) is still probably the most commonly used outcome measure for assessing the outcomes of the treatment of shoulder disorders including subacromial impingement [26]. It has the benefits of including an objective measurement of strength, and in this it differs from other scores, in combination with pain score, functional assessment,

and range of motion. Several critics have been moved to this test about sensibility, interobserver reliability, etc. The Western Ontario Rotator Cuff Index (WORC) was developed for use as a primary outcome measure in clinical trials evaluating treatments and was comprehensively tested during development. The final set of questions was selected by determining the most important factors from a patient's perspective [27].

# 41.1.2 Investigation: Instrumental, Radiological

X-rays are the basis for investigating the painful shoulder and a shoulder impingement syndrome. They may demonstrate subacromial sclerosis or spurs (hooked acromion) and anomalies of the acromion (os acromiale), sclerosis of the greater tuberosity and undersurface of the acromion, and reduced subacromial space with a proximal migration of the humeral head if associated with a torn or dysfunctional rotator cuff. They are also important in the differential diagnosis of SAIS as could demonstrate calcifying tendinitis, fractures, and neoplasm. Both anteroposterior and axillary views should always be asked (Fig. 41.2). Ultrasound (U/S) has been widely used for the evaluation of the shoulder, mainly for rotator cuff pathology. Ultrasonographic evaluation for rotator cuff tears were first described by Crass and Middleton in 1984 [28]. It has been shown to be a sensitive and accurate method of identifying patients with subacromial bursitis or full-thickness tears of the rotator cuff, and dynamic ultrasound can help confirm, but not exclude, a clinical diagnosis of impingement.

Although relatively inexpensive and noninvasive evaluation tool, the main issues regarding ultrasound relate to the interobserver variability in the demonstration of rotator cuff tears (Table 41.3).

Magnetic resonance (MR) arthrography is the most sensitive and specific technique for diagnosing both full- and partial-thickness rotator cuff tears that can be combined with subacromial impingement. U/S and MRI are comparable to each other in both sensitivity and specificity in

 Table 41.3
 Milgrom's ultrasound classification: grading of impingement changes

Stage 1: bursal thickness 1.5-2.0 mm

- Stage 2: bursal thickness over 2.0 mm
- Stage 3: partial- or full-thickness tear of the rotator cuff



Fig. 41.2 Shoulder X-ray with signs of impingement. (a) Anteroposterior view, (b) axillary view

 Table 41.4
 Bigliani classification: grading of acromion deformities

Type 1: acromion is flat in shape
Type 2: more curved acromion, which lies parallel to
the humeral head
Type 3: the edge of the acromion is hooked

the diagnosis of full-thickness rotator cuff tears but are rarely requested if just SAIS is suspected [13].

Although the indication for acromioplasty is based on clinical evaluation of the patient, it is generally supported by typical changes in acromial morphology on standard radiographs [6, 7, 96]. The most common classification is the one by Bigliani et al., but several attempts have been made to classify the acromial morphology (Table 41.4) [7].

Bigliani et al. [7] and Kitay et al. [97] described the *acromial slope* (AS; Fig. 41.3a), and Kitay et al. [97] and Aoki et al. [96] described the *acromial tilt* (AT; Fig. 41.3b). Other authors have focused on the lateral rather than the anterior extension of the acromion: Banas et al. [98] described the frontal plane slope of the acromion on MRI and found a lower *lateral acromial angle* (LAA; Fig. 41.3c) in patients with rotator cuff disease; Nyffeler et al. [99] observed that the acromion, of patients with a rotator cuff tear, appeared to have a more lateral extension than that of patients with an intact cuff and described the *acromion index* (AI; Fig. 41.3d) [29].

However, in the last years, this classification started to be discredited. Some authors [11, 12] reported that there are no close correlations between type III acromions and cuff ruptures and that spurs on the acromion deep surface, found mainly in old people, can't be related without any doubt with subacromial impingement.

## 41.1.3 Treatment Indications

Improved function can be obtained through reduction of inflammatory edema, strengthening of the muscles, which act as depressors and stabilizers of the humeral head, or by removing the inflamed and fibrotic tissue in the subacromial bursa and a part of the acromion itself. Treatment options, hence, can be conservative or surgical. Conservative management includes exercise therapy, ultrasound treatment, and subacromial injections.

Studies show that conservative management of shoulder impingement syndrome resolves the problem in 70–90% of patients [32]. In symptomatic patients, a course of conservative management is widely accepted as first-line management, but the time frame for this is variable and a point of controversy. Furthermore, the condition is often treated conservatively in the primary healthcare sector by general practitioners or physiotherapists [33]. Most surgeons generally tend to observe patients for a 6-month period before considering surgery; however, based on individual patient factors, this can vary.

*Exercise* is seen to be an effective treatment for SAIS [34, 35]. Several authors reported in the past that physiotherapy aimed at strengthening the muscular motors and stabilizing the shoulder joint renders satisfactory results especially in patients aged under 60 and represents a costeffective treatment. Different exercise regimens include supervised exercise, home exercise programs, and exercise associated with manual therapy.

*Massage* (myofascial trigger points in the shoulder muscles or soft tissue) appears to be more effective than placebo or no treatment in reducing pain and improving shoulder function in patients with shoulder pain. However, manual joint mobilizations have no added benefit to a program of active exercises in reducing pain and improving shoulder function.

Subacromial injections can be used to treat SAIS. A rotator cuff tear (RCT) showed that methylprednisolone conferred significant benefits on patients' symptoms and was effective in improving range of abduction at 6 weeks postin-jection [36]. Literature reported that subacromial steroids were better than placebo in improving the range of abduction. The authors reported that the duration of benefit of subacromial corticosteroid injections appears to be from 3 to 38 weeks [37]. However, a RCT by Crawshaw et al. concluded that corticosteroid injections combined with exer-





**Fig. 41.3** Overview of parameters of acromial morphology. (a) Acromial slope AS (d) according to Bigliani et al. (1986) and Kitay et al. [97]; (b) acromial tilt AT (b) according to Kitay et al. [97] and Aoki et al. [96]; (c) lat-

[98]; (d) acromion index (AI) according to Nyffeler et al.[99], [12]

cise were only successful in achieving short-term benefit, and [2] long-term results (2-year followup) showed no differences with manipulation and physiotherapy (half of the patients experienced recurrent complaints). In a recent study, 232 participants were randomized and divided in two groups: injection plus exercise and exercise only groups resulted similarly effective at 12 weeks, with no differences at week 24 [37]. *Oral NSAIDs* appear to be more effective than placebo in reducing pain in the first 1–2 weeks, but don't control pain in a long term [13].

*Laser treatment* (of all types) appears to be more effective than placebo or ultrasound treatment in reducing pain after 2–4 weeks [13].

Acupuncture treatment appears to have a good effectiveness in pain management but not a long-term effectiveness [13].



When conservative treatments fail, surgical procedures are recommended. However, there isn't a consensus in the literature of the indications with regard to the age of the patient. Some authors say that patients, suffering from isolated subacromial impingement and resistant to conservative therapies, benefit substantially from surgical decompression if they are young (<60 years). In fact, although physical demands decrease notably in older population, it still remains unclear if surgery leads to better results in these patients [61]. Other authors had different results and reported that in elderly patients (>60 years), arthroscopic decompression leads to better outcomes than conservative treatment, while in young patients, it is possible to achieve equivalently good to excellent results for both treatment regimens [64].

Recently, Sforza et al. presented a study on 421 arthroscopic decompressions. They reported the correlation of the impingement symptoms and the age of the patients (Fig. 41.4).

They also reported the correlation of clinical results of subacromial decompression in different rotator cuff tear sizes. The results of their study showed worse outcomes in patients with RCT >3 cm (medium size) if compared with patients with RCT <3 cm that have undergone ASD alone. In these patients, an arthroscopic repair of the lesion would be recommended even in the older population [65].

In athletic or young individuals (<40 years), instead, especially with recent trauma and dysfunctional damaged rotator cuff, surgical intervention should be considered at an earlier stage as successful repairs allow this particular group to return to pre-injury level of function [87, 88].

Subacromial decompression doesn't seem to have an indication in frozen shoulder. A study presented in 2012 on 29 nondiabetic patients showed no further medium-term functional benefits in patients that underwent subacromial decompression in addition to a standard arthroscopic capsular release [94].

Regarding the need of performing an ASD in patients with a RC tear, recent literature reviews report that RC repair gives a new balance and strength to the RC muscles, which prevent superior migration of the humeral head and, thus, relieves the patient from impingement symptoms. However, some authors promote ASD as a source of growth/angiogenic factors (matrix metalloproteinases MMP-2 and MMP-9) that may improve the healing process of the tendons [89]. Moreover, ASD, increasing the subacromial space, allows a safer rehabilitation process avoiding conflicts between the acromial spur and the sutures applied to the rotator cuff.

In the past, patients with calcific tendonitis usually underwent to ASD, as literature reported that subacromial decompression was the primary procedure to perform in patients with subacromial impingement and calcific tendonitis. A study, dated 1998, reported good outcomes and a complete disappearance of the calcific deposits (postoperative shoulder X-rays) in 97% of the patients treated with ASD alone (calcific deposits were left untouched) [66]. Anyway recent studies report that patients treated by debridement of the calcific deposit alone without a concomitant subacromial decompression required a shorter time to return to unrestricted activity without pain and to the same function [67].

# 41.1.4 Surgical Techniques

With regard to surgery in shoulder impingement, the two structures that need to be addressed are the acromion and the rotator cuff itself. Neer was the first to popularize acromioplasty for the treatment of shoulder impingement (Fig. 41.5). He emphasized that resecting the anteroinferior portion of the acromion would increase the volume of the subacromial space and therefore decrease the degree of impingement of the supraspinatus tendon under the acromion. Neer, also, described the indications for acromioplasty as being long-term disability from chronic bursitis and either partial tears or complete tears of the supraspinatus [6–30].

Today, arthroscopic subacromial decompression (ASD) is the gold standard to surgically treat this pathology. The procedure includes debridement of the subacromial bursa, resection of the coracoacromial ligament, and the anteroinferior acromion, as well as any underhanging osteophytes from the acromioclavicular joint [39].

ASD nowadays is more spread and adopted because of less morbidity: the possibility to perform it through arthroscopic portals reduces infection rate and the risks of neurovascular damages; improvements gain in anesthesia play an important role in pain management and safety of the procedure. It can be performed in lateral or beach chair position. A standard glenohumeral arthroscopy is performed via the posterior portal. This enables assessment of the undersurface of the rotator cuff. The scope is then withdrawn and inserted into the subacromial bursa. The bursa itself is then carefully inspected. First, the bursal surface of the rotator cuff is inspected to confirm the presence of an impingement lesion of this area and subacromial surface ("kissing" lesion).

Many techniques were described to perform ASD. In 1994, Snyder presented the *cutting block technique* (Fig. 41.6) [40].

Through the posterior portal, a large full radius resector is passed for bursal resection,

Fig. 41.5 Open subacromial decompression

while the subacromial space is distended and visualized using the arthroscope in the lateral portal. Once the bursal tissue and periosteum of the acromion have been adequately resected to allow identification of the anatomic landmarks, the coracoacromial ligament is resected. In this technique, the posterior aspect of the acromial undersurface will serve as a cutting block to guide the resection of the anterior acromion bone



Fig. 41.6 Cutting block technique [29]

wedge. The burr sheath is firmly applied to the undersurface of the acromion so that mediallateral sweeping of the burr tip creates a shallow groove just at the predetermined point. The burr is then slowly advanced anteriorly while maintaining the medial-lateral sweeping motion. The resection is completed when the anterior edge of the acromion is removed. Finally, the arthroscope may be switched to the posterior portal to better evaluate the most lateral edge of the acromion as this edge is often too close to the arthroscope lens, to allow safe burring when viewing from the lateral portal. Similar probe and burr technique may be used through the lateral portal if modification is necessary [41].

In 1995, Copeland [42] presented a different way of performing this procedure (Fig. 41.7).

The scope is kept through the posterior portal, while the resector works from the lateral portal throughout all the procedure. The starting point is the anterolateral edge of the acromion where the coracoacromial ligament is attached. After the ligament is resected, the exact bony margins of the acromion are visualized. By shaving medially,



**Fig. 41.7** Arthroscopic subacromial decompression [42]

 Table 41.5
 Levy-Copeland arthroscopic classification:

 grading of mechanical effects of impingement [31]

Acromial side
A0: normal – smooth surface
A1: minor scuffing, hemorrhage, or local injection and inflammation
A2: marked scuffing/damage of the undersurface of the acromion and CA ligament
A3: bare bone areas
Bursal side
B0: normal – smooth surface
B1: minor scuffing, hemorrhage, or local injection and inflammation
B2: major scuffing of cuff, partial-thickness tear
B3: full-thickness tear
B4: massive cuff tear

the surgeon can visualize the acromioclavicular joint. All bone lying anterior to acromioclavicular joint is excised down to its full depth. This is best done starting laterally and then moving medially. After removal of the whole thickness of the anterior acromion, the anterior edge of the acromion is then shaped and angled posteriorly to leave the undersurface of the acromion as a straight surface, removing the bony hook. To assess the adequacy of bone removal, the thickness of the shaver can be used (4 mm). The width of the resection can be verified by exactly delineating the origin of the coracoacromial ligament by working from lateral to medial. Once the acromioclavicular joint is reached, the full width of the acromion has been reshaped. The inferior margin of the clavicle needs to be inspected to detach osteophytosis that might cause impingement. At the end of the procedure, the bursa should be irrigated to remove all possible traces of small bone dust [42].

The direct visualization of the kissing lesions (inferior surface on the acromion and bursal side of the cuff) through an arthroscopic procedure led to the development of a new classification (Table 41.5).

Concerning the classification of the rotator cuff lesions, Snyder's Classification needs to be mentioned (Table 41.6).

During revision of ASD procedures, some authors observed the regeneration of the Table 41.6 Snyder classification of RCT

(A) Articular surface

A0. Normal

A1. Minimal superficial bursal or synovial irritation or slight capsular fraying over a small area

A2. Fraying and failure of some rotator cuff fibers in addition to synovial bursal or capsular injury. More severe rotator cuff injury fraying and fragmentation of tendon fibers often involving the whole of a cuff tendon, usually <3 cm

A4. Very severe partial rotator cuff tear that contains a sizeable flap tear and more than one tendon

(B) Bursal surface

B0. Normal

B1. Minimal superficial bursal or synovial irritation or slight capsular fraying over a small area

B2. Fraying and failure of some rotator cuff fibers in addition to synovial bursal or capsular injury. More severe rotator cuff injury fraying and fragmentation of tendon fibers often involving the whole of a cuff tendon, usually <3 cm

B4. Very severe partial rotator cuff tear that contains a sizeable flap tear and more than one tendon

(C) Complete tear

C1. Small complete tear, pinhole sized

C2. Moderate tear <2 cm of only one tendon without retraction

C3. Large complete tear with an entire tendon with minimal retraction usually 3–4 cm

C4. Massive rotator cuff tear involving 2 or more rotator cuff tendons with associated retraction and scarring of the remaining tendon

Comprehensive classification including the size position and quality of tendon [89]

coracoacromial ligament (CAL). This finding, therefore, started to be studied to aid the understanding of the successes and failures of ASD. In 2000 and 2001, Henderson et al. (Melbourne, AUS) and Levy et al. (Reading, UK) reported that coracoacromial ligament has an ability to reform or reattach, whether primarily resected or released, and that this might account for recurrent symptoms [43]. Electron micrograph studies (J. P. Henderson) [43] and histology (O. Levy) [44] revealed appearances indistinguishable from normal ligament, which was in continuity with the reformed periosteum of the acromion. In a following study, Levy et al. showed the results of mechanical testing on eight regenerated coracoacromial ligaments, which appeared to have the



Fig. 41.8 Coracoacromial arch

ability to reform relatively quickly but took time to regain strength (3 years) [45]. Similar results were seen in a study performed on ACL of the knee. Well-organized parallel bundles of collagen fibers on hematoxylin-eosin and Van Gieson preparations were observed in the proximal one third of injured ACLs. Findings were consistent in all patients, and no scar or disorganized fibrous tissue was found. These characteristics are typical for a spontaneous healing process like it is known to happen in the medial collateral ligament (MCL) [46].

This regenerative process fails, instead, when a constant and repetitive strain is applied to the area of insertion of the CAL to the acromion. This happens in patients with a large or massive rotator cuff tear, where a consequent proximal migration of the humeral head is observed. This is a clinical condition in which subacromial interruption of the coracoacromial arch should be avoided, as proximal subluxation with anterosuperior escape of the humerus can occur as well described in previous reports (Fig. 41.8) [2, 13, 15–90].

# 41.1.5 Complications

Complications can be divided into general complications (generic to all shoulder procedures) and complications specific to the type of procedure performed. Complications related to general anesthesia (GA) and nerve blocks and general complications, such as infections, bleeding, and clots, continue to show low incidences. Shoulder arthroscopy presents increased risk of complications over knee arthroscopy in regard to vascular and neurologic injury, fluid extravasation, stiffness, iatrogenic tendon injury, and equipment failure. However, in all recent review studies, the rate of complications is still low (5.8–9.5%) [92].

One of the most dreaded complications, after acromioplasty or arthroscopic decompression, is the fracture of the acromion process. In 1994 a study reported six cases of post ASD fracture of the acromion. Treatment of this complication ranged from total acromionectomy to conservative measures. Indifferently from the treatment, most results were poor. Risk factors include osteopenia and overzealous bone resection. An appropriate preoperative planning and meticulous surgical technique to minimize bony resection may decrease the risk of this complication and its resultant disability [47]. Postoperative heterotopic ossifications (HE) were observed in some patients after ASD or open acromioplasty. The authors reported that bone formation could not be correlated with the method of bone resection and that revision surgeries and HE prophylaxis for recurrence of symptoms are sometimes required [48]. Another rare complication is the recurrence of acromion spur. A case report showed as an anterolateral subacromial spur and clinical impingement signs can recur years after ASD [49].

## 41.1.6 Result Literature Review

Neer's initial results, as well as other follow-up studies, showed excellent outcomes, with success rates from 80 to 95%. Ellman reported satisfactory results for ASD in 88% of patients at 1-3 years of follow-up [39]. Many different techniques have been reported with similar results [50]. Since the initial reports, many authors [51–54] reported their results after ASD: 73-88% good to excellent, approaching the results of the previously reported open subacromial decompressions. Other studies showed even better results. In a recent study, 45 female underwent ASD volleyball players for SAIS. Excellent results were reported in 62.3%,

good in 30.7%, satisfactory in 4.6%, and bad in 2.4%. The final results were 91% included as good and excellent. Other authors had similar results: Paulos (85%), Garstman (90%), Godinho (90.8%), and Altchech (92%) [55].

Comparing arthroscopic ASD to open acromioplasty, literature confirms that open surgery doesn't allow better results. In a recent study in the group of patients that fail to benefit from the arthroscopic decompression, only a marginal improvement was noted after revision with open decompression [56].

However, despite the good results reported, literature is still divided. According to a Cochrane review, there is little evidence to support or refute the efficacy of common interventions for shoulder pain [57]. Moreover, evidence supporting the superiority of subacromial decompression relative to physiotherapy with training has been unconvincing [58, 59].

In a recent article in Orthopedics Today [60], doubts have been raised over the effectiveness of ASD. Health economists in Denmark have reported low and delayed return to work for patients treated for SAIS with ASD. Their argument is that there are no financial benefits for the government due to the poor rate of return to work. Surgeons argue that patients achieve good pain relief and a high standard of activities of daily living (ADLs) after ASD. A detailed review of the literature suggests that there is no clear benefit of surgery over conservative treatment.

Randomized controlled trials by Brox et al. [59] and Haahr et al. [61] comparing exercises with ASD found that, although individually they are successful treatments, ASD was not superior to specialized exercise programs. Also, systematic reviews by Dorrestijn et al. [34] and Gebremariam et al. [62] show similar results.

A long-term follow-up randomized study was performed in the Kanta-Häme Central Hospital, Hämeenlinna, Finland. The authors suggested [63] that treatment with ASD combined with structured exercise treatment did not provide better results at 5 years compared with structured exercise alone, when assessed by self-reported pain. The same pattern was seen in the secondary outcome measures of disability, pain at night, number of painful days, and the proportion of pain-free patients.

To confirm that the debate is still open, the findings of another study published in 2014 [2] have demonstrated significant improvement in outcome for patients with SAIS undergoing ASD, who have had previous failed conservative treatment with standard physiotherapy and at least one subacromial injection. The median Oxford Shoulder Score improved significantly at 6 months after ASD. This implies that patients have reported benefits in their activities of daily living. Furthermore, the improvements in the individual components of the constant score highlight excellent pain relief, objective benefits in ROM, and also increase in shoulder strength. The study showed that 75% of patients achieved a minimum of 5-point benefit in OSS and 77 % of patients achieved a minimum of 10-point improvement in CS. This is a significant result and highlights the success of ASD for patients with SAIS [2].

Different impingements were documented more recently. Internal impingement refers to overhead athletes that experience shoulder pain during throwing sports. It can present as a constellation of pathological processes, including partialor full-thickness rotator cuff tears, anterior or posterior capsular injury, labral tears, glenoid chondral erosion, chondromalacia of the posterosuperior humeral head, and biceps lesions. Moreover, the absence of these lesions does not exclude a diagnosis of internal impingement. Muscle fatigue can affect the mechanism of throwing leading to a humeral hyperextension in the late-cocking phase of throwing [69]. This can lead to a damage of the posterior capsulolabral structures. This condition is further permitted by the development of anterior capsular microinstability [70, 71] and posterior capsular stiffness (GIRD – glenohumeral internal rotation deficit), which lead to the translation of the humeral head (peel-back mechanism), which may "peel off" from the glenoid the labral biceps insertion (type II SLAP lesions) [72–93]. Stretching and physiotherapy give good results in patients with a mild symptomatology and no intrinsic lesions. When a lesion of the RC is found (Walch et al. 1991) [68],



**Fig. 41.9** (a) An arthroscopic view of the right shoulder shows an articular partial tear of the insertion of the supraspinatus tendon (*arrow*) with a tendinous flap, just poste-

rior to the biceps. (b) An arthroscopic view shows posterior labrum delamination (*arrow*) [73]

however, a debridement of the tendon and of the labrum [68], followed by the shaving of the bone spur on the posterior edge of the glenoid [73], is recommended. Walch described this condition as the posterosuperior glenoid impingement (PSGI), in which a lesion occurs between the deep side of the supraspinatus tendon and the posterosuperior edge of the glenoid cavity (Fig. 41.9a, b).

Jobe [70] and Andrews [93], however, consider the anterior instability as the principal cause of intrinsic impingement and recommend treating anterior capsule laxity, in order to achieve good results.

The coracoid process can be another area of impingement, even if this is a less common cause of shoulder pain. Symptoms occur when the sub-scapularis tendon impinges between the coracoid and lesser tuberosity of the humerus. Coracoid impingement should be included in the differential diagnosis when evaluating a patient with activity-related anterior shoulder pain [74–76].

Palpation often elicits tenderness of the soft tissues around the coracoid process and between the coracoid process and the lesser tuberosity [77–79]. The coracoid impingement test is performed in a manner similar to that used to perform the Kennedy-Hawkins impingement sign, except that the patient's shoulder is placed in a

position of cross-arm adduction, forward elevation, and internal rotation to bring the lesser tuberosity in contact with the coracoid [80]. Pain is elicited more consistently in the midrange of forward elevation than in the full elevation that is used to detect subacromial impingement [81, 82]. A lidocaine injection in the subcoracoid region may also be of utility in establishing a diagnosis [83]. MRI or CT examinations appear to be more precise in establishing the diagnosis than simple X-rays [84]. In most cases, axial sequences are used to measure the coracoid-humeral distance (CHD), defined as shortest distance between the humeral head and the coracoid process [85]. In addition to this, the coracoid index, defined as the lateral projection of the coracoid beyond the glenoid joint line in axial CT or MR images, is theorized to have an influence on developing coracoid impingement.

The first line of treatment for coracoid impingement should be a program of activity modification, with avoidance of the provocative positions of forward flexion and medial rotation, and physical therapy to strengthen rotator cuff muscles and scapular stabilizer musculature. Surgical decompression of the subcoracoid space may be undertaken if the above conservative measures fail (Fig. 41.10a, b) [77]. The options



**Fig. 41.10** (a) Preparation of coracoid process (*dotted black line*) with electrocautery through the open joint capsule and (b) completed coracoplasty [95]

include open or arthroscopic coracoplasty, a combination of coracoacromial ligament resection and acromioplasty, or anterior shoulder stabilization [81, 86].

Partial-thickness tears of the subscapularis muscle are usually found in these patients. Most of them are undersurface tears, but it is possible to find linear longitudinal subscapular tears, which indicate a tensile undersurface fiber failure (TUFF lesion). The "roller-wringer effect" was described by Burkhart to explain the pathomechanism of TUFF lesion (increased tensile forces on undersurface of subscapularis insertion) and is common in this kind of impingement [93].

## Conclusion

When we look at shoulder impingement, we can't consider only subacromial impingement. In the last years, the authors described different typologies of impingement. The shoulder is a complex joint that needs to be balanced to work properly. Mechanical forces have to be counteracted by muscle action and capsular ligament structures.

When at least one of these anatomical structures loses its function (due to injuries, overload, age, dyskinesia, etc.), movements of the humeral head are not controlled, anymore. Therefore, during the range of motion of the joint, humeral head loses the natural rapports with the socket and hits the surrounding structures (bone structures, labrum, and tendons) leading to an inflammatory process and, eventually, to a structural damage.

Arthroscopic decompression is a quick and safe procedure that gives good results with a low rate of complication and failures. However, it is very important that the surgeon has a clear idea of the pathogenic mechanism that leads to the symptoms, before surgical procedure is offered to the patient.

Failure to improve patient symptoms, therefore, can be expected if the procedure is proposed with an incorrect indication. The shoulder is a high-demanding joint that hardly forgives diagnostic errors.

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