The purpose of this presentation is to accomplish the three “Ds”: (1) Define failed arthroscopic subacromial decompression (ASD), (2) Determine the cause, and (3) “Do the right thing.” The success rate with arthroscopic subacromial decompression has historically ranged from 80%-90%. Therefore, a “failure” rate of 10%-20% must be accounted for. In those circumstances where a failure has occurred, frustration on the part of both patient and physician can interfere with a systematic approach to surgical failure. It is essential that the surgeon be meticulous and systematic in the assessment of the failed ASD.

Although the time interval before deciding that an acromioplasty has failed is debatable, patients with persistent symptomatology who are six months status post decompression should be considered potential failures. The definition of a failed arthroscopic subacromial decompression would be any or a combination of the following: persistent pain, weakness, motion loss, or an inability to return to sports, recreation, or the workplace.

Once a failed ASD has been determined, the following questions need to be answered: (1) Was the initial diagnosis correct? (2) Was the appropriate procedure chosen? (3) Was the procedure performed correctly? (4) Was associated pathology recognized and adequately treated? And (5) Was the rehabilitation timely and appropriate? Once these questions have been answered, there are three possible explanations: (1) Incorrect diagnosis, (2) Technical or treatment error, (3) Complication of treatment.

Incorrect Diagnosis

Diagnostic error can account for failure following arthroscopic subacromial decompression. Several investigators have attributed nearly 50% of acromioplasty failures to an incorrect or missed diagnosis. The most common possibilities include unrecognized primary shoulder instability with secondary rotator cuff symptoms, internal rotation contracture, neurologic dysfunction such as a brachial plexus neuritis, thoracic outlet syndrome or cervical radiculopathy, glenohumeral arthritis, acromioclavicular arthritis, and symptomatic os acromiale. The following highlights several of these diagnostic dilemmas.

Instability

Unrecognized shoulder instability can be found in the younger overhand athletic population. Internal impingement as a possible source of pain should be considered, whereas primary external impingement due to outlet impingement is uncommon. Arthroscopic subacromial decompression under these circumstances serves only to destabilize an already compromised shoulder, and in many cases, leads to a worsening of symptoms. Persistent posterior shoulder pain in the young overhead athlete should lead to a high index of suspicion for internal rather than external impingement in this patient population. An arthroscopic subacromial decompression would only be appropriate in conjunction with primary treatment such as capsular plication and/or SLAP repair, and if secondary changes are present in the subacromial space.
Os Acromiale

For those patients with a symptomatic os acromiale, a decompression alone may not be sufficient if the mobile fragment is the source of pain.13 Oftentimes the mobile fragment alone may be the source of ongoing discomfort and may occur absent any impingement phenomenon. Once the diagnosis of a symptomatic os acromiale has been made with the use of a careful history and physical examination, bone scan, contralateral comparison x-rays, and motion at the non-union site has been confirmed at arthroscopy, open reduction and internal fixation with parallel cannulated screws supplemented with local bone graft from the greater tuberosity is the recommended treatment.14,15 One exception would be the pre-acromion, a small fragment which can be excised in its entirety with minimal morbidity. For the meso-, meta-, or basi-acromion, internal fixation would be recommended. Although others16,17 have described successful arthroscopic excision or near total excision of the meso-acromion without residual sequelae, should a subsequent weakness or pain pattern develop due to loss of the deltoid fulcrum, no adequate solution exists.

Posterior Capsular Contracture

Limitations in internal rotation, often noted in the overhand athlete, can shift the center of humeral head rotation into a posterior-superior direction, effectively creating a secondary impingement as the humeral head and rotator cuff are forced into the subacromial space. In a reported series of nine isolated internal rotation contractures,18 three occurring after an arthroscopic subacromial decompression, eight of nine patients who underwent an isolated posterior capsular release were relieved of pain. Internal rotation in the abducted position increased an average of nearly 40 degrees.

An awareness of this unusual source of impingement type pain, and a careful examination evaluating internal rotation in the abducted position can help prevent missing this diagnosis which will not respond to decompression alone.

Technical Error

Technical errors can and do occur and usually take the form of variable bone resection. The most common error is under-resection with a residual spur in an external impingement patient leading to persistent symptoms. Under-resection has been cited as a cause of failure2,3,19,20 in up to 50% of failed cases. Over-resection can lead to intraoperative or delayed fracture,29 as well as possible deltoid detachment.1,22 Fractures following an acromioplasty have historically been associated with a poor prognosis.

Obtaining quality, preoperative radiographs and in particular, the outlet view, allows the surgeon to determine the amount of bone resection, as well as the size and shape of the acromion. The cutting block technique23 can be used for the actual bone resection, as well as for measuring the amount of bone excised. If a lateral portal technique for bone resection is used, a blunt trocar passed from the posterior portal can be used to assess acromial morphology and to determine if adequate bone has been resected. If the acromion is thin and curved, the cutting block technique must be modified such that a “contoured” acromioplasty is performed and thinning avoided.

Treatment Failures

Rotator Cuff Insufficiency: There is a role for arthroscopic subacromial decompression in the rotator cuff deficient patient. In a limited goals operation, an arthroscopic subacromial decompression can yield significant pain relief even though the rotator cuff is not repaired. Careful selection of the proper candidate for an isolated decompression in the face of a rotator cuff which is not repairable, or in a patient who elects not to undergo a formal repair, is imperative. Any evidence of superior migration of the humeral head on the AP radiographs is a contraindication to an isolated decompression. As a guideline, patients with an intact subscapularis and at least one-half of their infraspinatus and teres minor intact, usually maintain satisfactory force couples allowing for a decompression without decompensating the shoulder.24,25 Individuals without balanced force couples may require an intact coracoacromial arch acting as a fulcrum in order to elevate their arm. In these instances, removing a portion of the acromion and releasing the CA ligament puts these patients at risk for loss of superior containment of the humeral head with subsequent loss of the arch, a predicament for which there is no good solution.

Partial Rotator Cuff Tear: There has been considerable discussion regarding the appropriate treatment of high-grade partial rotator cuff tears. A recent study26 indicated that for partial rotator cuff tears of less than 50% treated by decompression only, the long-term success rate was equal to that of patients with minimal rotator cuff pathology treated with ASD alone. Others have cited their experience with poor
outcomes following isolated decompression in individuals in whom a high-grade partial tear is present, high-grade being defined as 50% or greater.27-30 In the young active population, especially in a dominant extremity, at the time of a proposed decompression, if significant rotator cuff pathology is noted in the form of a high-grade partial tear, current recommendations include not only a decompression but repair of the partial rotator cuff tear. This could be achieved with an arthroscopic trans-tendon repair with anchors placed in the footprint of the rotator cuff or with completion of the tear from the bursal side of the cuff followed by a routine arthroscopic rotator cuff repair. I believe it is critical for the surgeon to discuss this possibility with all patients undergoing a routine arthroscopic subacromial decompression. Although MRI scans or other preoperative testing may yield what appears to be sufficient information regarding the status of the rotator cuff, there is enough uncertainty with diagnostic testing such that discussion of all possible pathologies and the surgical algorithm must be discussed in advance with the patient so that a complete solution can be accomplished at the time of surgery.

Workers’ Compensation

Results in the Workers’ Compensation category suggest a glaring difference in the success rate between patients treated with a decompression and those injured on an industrial basis treated with a decompression.1,3,20,24 Although there may be a mechanical reason for failure, the issue of “secondary gain” can place the Workers’ Compensation group into the treatment failure category. At least one author has cited a failure rate of 18% despite a correct diagnosis and correct treatment being rendered.3 Despite this potential selection bias, it is important for the surgeon to discuss in advance with the patient and possibly the case manager, the expected course which usually involves more postoperative pain, a slower rehabilitation, and a slower return to duties. Despite the documented poor outcomes in the Workers’ Compensation group, it is important for the surgeon to carefully evaluate each of these patients for a potential cause other than their worker’s status.

Complications of Treatment

Following surgery in the subacromial space, complications can occur, including infection, neurovascular injury, arthrofibrosis, reflex sympathetic dystrophy, heterotropic ossification, late acromioclavicular joint pain, as well as recurrent spur formation. The most common reported complication following arthroscopic subacromial decompression is scarring.35

The Captured Shoulder

The captured shoulder was described in 1996 and attributed to subdeltoid adhesions following subacromial space surgery. This in turn led to compensatory mechanics and asynchronous shoulder girdle motion. Although the arthroscopic subacromial decompression is usually performed with little concern for scarring, the possibility of scarring and “capture” of the subacromial space secondary to bleeding and raw surfaces must be considered. A well-conceived, supervised rehabilitation program can help minimize this risk. In particular, posterior capsular stretching to prevent the possibility of an internal rotation contracture postoperatively can help minimize the risk of continuing impingement forces. Furthermore, a full functional range of motion should be achieved before strength work is initiated, and particular attention should be paid to the scapula as it is often overlooked. For a complete recovery, scapular strengthening and normal scapulothoracic and glenohumeral synchrony must be reestablished safely.

Coplaning and Late Pain

Several authors have reported late acromioclavicular (A-C) pain following arthroscopic subacromial decompression combined with co-planing of the distal clavicle.32,33 Interruption of the inferior capsule destabilizes the A-C joint, which may in turn cause premature acromioclavicular joint degeneration. Despite early reports of late A-C pain and the destabilizing effect of co-planing, several recent clinical reviews have failed to identify an increase in late A-C joint disease following co-planing.34,35 Current recommendations emphasize a thorough preoperative evaluation of the A-C joint, and a complete resection in conjunction with an ASD if true A-C pathology is diagnosed. Unless there is evidence that distal clavicular spurring is a source of symptoms, the A-C joint should be spared.

Conclusions

When confronted with a failed ASD, the treating physician must consider the following: diagnostic error, technical or treatment failures, and complications of treatment. The treating physician must methodically identify the reason for failure and then proceed with corrective action if indicated. It is imperative to
remember that up to 15 percent of failures may not have a discernible cause with the correct diagnosis and correct operation having been performed. One must be patient and meticulous and should remember that any problem can be made worse with an indiscriminate intervention.

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CORACOACROMIAL ARCH INSUFFICIENCY

Stephen S. Burkhart, M.D., and Peter M. Parten, M.D.

Coracoacromial arch insufficiency can cause a tremendous amount of dysfunction in the shoulder, and is extremely difficult to treat surgically. Unfortunately, it is not well understood and this lack of understanding has caused treatment options to develop in several disparate directions. In exploring this problem, we will attempt to define the problem by defining the underlying cause, the anatomic deficit, the mechanical problem, and the dysfunction. Then we will look at the various means of treatment, as well as some of the unanswered questions.
Defining the Problem

Patients with coracoacromial arch insufficiency have generally had previous shoulder surgery in which an acromioplasty has been done along with excision of the coracoacromial ligament, in the face of proximal migration of the humerus. This allows the humerus to migrate beneath the anterior deltoid, the last remaining constraint of the coracoacromial arch. Frequently, in patients that have deltoid detachment, or in patients with very small muscle mass, the humeral head will appear to be directly under the skin (Fig 1).

The anatomic deficit in a massive rotator cuff tear can assume one of two patterns. One pattern is somewhat symmetric, wherein a small amount of cuff remains intact both anteriorly and posteriorly. Typically with this pattern, less than half of the subscapularis is intact in the anterior aspect of the shoulder and the teres minor (with or without a small portion of the infraspinatus) is the only intact cuff tendon posteriorly. The second pattern is one in which the subscapularis is intact, but the entire superior and posterior cuff has been torn. Both of these patterns allow proximal migration of the humerus under the acromion1 (Fig 2).

**Figure 1.** Patient demonstrates inability to elevate the left shoulder secondary to coracoacromial arch insufficiency and proximal migration of the humerus.

**Figure 2.** Symmetric pattern of massive rotator cuff tear with a captured fulcrum under the acromion, (A) anterior, (B) posterior.
The mechanical deficit relates to a loss of balanced force couples in the shoulder. The force couple in the transverse plane is lost (Fig 3). In the coronal plane, the inferior portion of the cuff is not adequate to counterbalance the deltoid so that proximal migration occurs\textsuperscript{1-4} (Fig 4).

The dysfunction involves pain and loss of overhead motion. On attempted elevation of the shoulder, patients typically have only a “shoulder shrug” of motion (see Fig 5). Patients with posterosuperior tear patterns may have an external rotation lag, in which they are unable to actively externally rotate the shoul-

\textbf{Figure 3.} Asymmetric posterosuperior pattern of rotator cuff tear, (A) anterior, (B) posterior.

\textbf{Figure 4.} Force couple in the transverse plane between the anterior cuff (subscapularis) and the posterior cuff (infraspinatus and supraspinatus).

\[ \Sigma M_{O} = O = I \times R - S \times r \]
\[ \therefore I \times R = S \times r \]

\textbf{Figure 5.} Coronal plane force couple between the deltoid and the inferior portion of the rotator cuff.
der to neutral (Fig 6). Those that have loss of sub-
scapularis function will exhibit a positive Napoleon
test5 (Fig 7).

Radiographically, the AP x-rays show proximal hu-
meral migration (Fig 8). The MRI scan shows a mas-
sive rotator cuff tear (Fig 9). Since we have found the
MRI to be less reliable for subscapularis tears than for
the rest of the rotator cuff,5 the subscapularis may or
may not appear to be intact on MRI.

The Awning Effect

The senior author (S.S.B.) did a kinematic study
several years ago in which he observed an awning
effect in patients with proximal humeral migration. While viewing them fluoroscopically, he observed that those with “long awnings” seemed to have their humeral head abut against the awning and could not elevate forward past the edge of it, whereas those with “short awnings” seemed to hit the front of the awning over the humeral head and then be able to elevate the arm upward. In addition, this fluoroscopic study revealed that patients who were able to use the arm overhead despite the fact that they had proximal migration were patients that initiated scapular abduction very early. Those that were unable to get the arm overhead did not have much abduction of the scapula at all.

Arthroscopic Findings

Arthroscopically these massive tears demonstrate what we call a “Grand Canyon view,” particularly when viewed through a lateral viewing portal (Fig 9). These massive tears typically have an apex located above or medial to the glenoid.

Partial tears of the subscapularis are best viewed through a posterior viewing portal with the arm internally rotated about 20 degrees. This will show a bare subscapularis “footprint” if there has been partial disruption in the superior part of that tendon.

Chronic tears of the subscapularis can be more difficult to visualize. In their retracted position, it may be impossible to tell which part of the tissue represents subscapularis. One must look for a “comma sign,” a distinctive configuration of the pathoanatomy created by the attachment of the superior glenohumeral ligament to the superolateral aspect of the subscapularis. When both of these structures tear from their contiguous bone attachments on the lesser tuberosity, they remain linked together in the shape of a “comma” (Fig 10), giving rise to the arthroscopically unique “comma sign.” The junction of these two structures causes the “comma” appearance in these chronically retracted tears.

The adhesed subscapularis represents an even more difficult problem. Frequently a chronically retracted subscapularis tendon will be adhesed up against the deltoid and such a shoulder can appear to have an absent subscapularis. One must look carefully for the “comma sign” and use a probe to detect thickening of the tendinous portion of the subscapularis. Once it is located, one can use a pencil-tip electrocautery to dissect the adhesed subscapularis from the deltoid.

One must also dissect the adhesed posterior cuff off the deltoid. It is best to locate the interval between the acromion and the superior rotator cuff, which typically lies just above the glenoid in the area of the AC joint. The surgeon then should follow this plane posteriorly with his shaver and continue in that plane, which generally will contain a fibro-fatty layer of tissue between the deltoid and the rotator cuff. The power shaver can be used to further define the plane between the rotator cuff and the deltoid.

Treatment of Coracoacromial Arch Insufficiency

Rotator cuff repair to restore the force couples, and to repair the subscapularis tendon in particular, can restore overhead function and in some cases can provide reversal of proximal migration of the humerus. We have followed 10 patients with repair of combined tears of the subscapularis, supraspinatus, and infraspi-
natus tendons who had proximal humeral migration pre-operatively (average tear size 5 × 8 cm). After repair, eight of these ten had reversal of their proximal humeral migration. At average follow-up of 11 months, these eight shoulders improved their forward flexion from a pre-op average of 50.8 degrees to a post-op average of 135.2 degrees. The subscapularis is particularly important and appears to have a function as a static constraint even if the muscular function is in doubt as represented by a fatty infiltration on MRI. We have found that medialization of the subscapularis up to one centimeter seems to be acceptable and subscapularis repair in that position can still provide significant improvement in function. Therefore we think that, if at all possible, a torn subscapularis should be repaired. It should be repaired even if the tear is chronic and even if the tendon has to be medialized from its anatomic attachment.

A number of other treatments have been recommended. These are open reconstructive treatments. One of these is coracoacromial ligament reconstruction using fascia lata as reported by Flatow and associates. Two of their six patients were satisfied, but there was not any significant increase in motion. Wiley recommended a similar idea, although he described bone grafting from the acromion to the coracoid to provide a bony constraint, rather than a fascial constraint. He reported on the technique of the procedure, but no long term results have been reported. Latissimus dorsi transfer has been recommended for dysfunction related to proximal migration of the humerus with coracoacromial arch insufficiency.
Miniaci and associates reported on 17 patients. They stated that their patients’ average flexion improved from 42 degrees to 101 degrees and that their UCLA scores improved from 6.8 to 16.4. Hemmigan et al. reported on 14 patients in which the ASES score improved from 39.5 to 65.9. They postulated a tenodesis effect.

Wirth and Rockwood described a pectoralis major transfer that was superficial to the conjoined tendon. Concerns about the direction of pull of the transferred tendon resulted in subcoracoid placement of the pectoralis major transfer as described by Resch and associates. Galatz et al. also have reported using this transfer. They favor it because they feel that the direction of pull helps to limit proximal migration and that the coracoid base acts as a pulley, optimizing the direction of muscle contraction. In their 17 patients, flexion improved from 37 degrees to 72 degrees. Williams and Iannotti described iliotibial band graft reconstruction in the front of the shoulder for subscapularis insufficiency. They felt that there was a beneficial tenodesis effect, even though they did not restore any muscular function. Forward flexion improved from 42 degrees to 105 degrees. Hemiarthroplasty has been recommended for treatment of a painful shoulder with proximal humeral migration and coracoacromial arch insufficiency. Cofield et al. reported pain relief in 75 percent, but stated that motion was not improved in these patients.

Finally, as a last resort, one might consider glenohumeral arthrodesis as a solution to this very difficult problem.

Conclusions

1. Coracoacromial arch insufficiency is a very difficult problem to treat, and prevention is the best means of treatment. This requires meticulous rotator cuff repair in patients that do not have proximal humeral migration.

2. In patients with proximal humeral migration, repair of chronic subscapularis tears in association with repair of the rest of the cuff can sometimes result in reversal proximal humeral migration.

3. Static tenodesis (for example with ITB graft) has reported results with better function than pectoralis major transfer, and approximately equivalent to latissimus dorsi transfer.

4. Hemi-arthroplasty should be done for pain relief only.

5. Arthrodesis should be considered as a last resort.

6. The tenodesis effect in place of subscapularis muscle function may be a mechanism of improving function, by restoring the force couple in the transverse plane. The big unknown is the role of the awning effect in enhancing or diminishing overhead function.

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GLENOHUMERAL INSTABILITY AND ROTATOR CUFF DYSFUNCTION

R. Michael Gross, M.D.

Glenohumeral instability and rotator cuff tears appear to be unrelated, but that is often not the case to joint destruction. In fact, instability and rotator cuff disorders are acutely interrelated with one frequently directly contributing to and/or causing the other to develop. It is important to understand the mechanics of the normal anatomy and how damage of either the ligaments and/or the rotator cuff can lead to pathology within related structures.

The Vacuum Phenomena

Peter Helmi et al. demonstrated the loss of shoulder stability in the unloaded state simply by puncturing the capsule thus releasing the vacuum. This study noted that with the loss of the vacuum, the humerus exhibited marked increased laxity on both the AP as well as the superior inferior plane. Habermeyer et al. noted that traction on a normal shoulder leads to an increased negative pressure while this increase was not noted in a shoulder with a Bankart lesion. One can only assume that the same effect would be seen with a full thickness rotator cuff tear.

Conformity Compression Principle

Stephen Lippitt et al. have introduced the conformity compression principle. It states that the conformity of the G/H joint coupled with the compressive load from the rotator cuff stabilizes the shoulder. With the labrum intact, the humeral head resists a tangential force of up to 60 percent of the compression load across the joint. Resection of the glenoid labrum (as with a Bankart injury) drops the resistance force by 40 percent.

Capsular Structures

The capsular structures are the main passive stabilizers of the glenohumeral joint. The superior glenohumeral ligament stabilizes the shoulder from inferior subluxation while the arm is at 0 degrees of abduction, and from posterior subluxation while it is flex 90 degrees and internally rotated. O’Brien et al. studied the inferior glenohumeral ligament and described a three-part structure: an anterior and posterior band with the axillary pouch being supported between these two bands. In abduction and external rotation, the anterior band prevents anterior translation of the humeral head while the posterior band rotates inferior to the humeral head, and supports the abducted arm from inferior glide. The opposite happens while the shoulder is in abduction and internal rotation.

Stephen Howell et al. studied humeral motion on the transverse plane. Howell’s group found a consistent 4-mm posterior translation of the abducted humeral head as it reached full external rotation. This phenomenon is lost in an unstable shoulder. Using selective nerve blocks Howell isolated the cause of this “roll back” phenomenon to the capsular structures.

The Rotator Cuff

Hsu et al. at the Mayo Clinic studied the effect of damage to the rotator cuff on the stability of the glenohumeral joint. Hsu et al. found that tears in the critical zone had a greater effect on the inferior stability of the joint, while those located in the rotator interval had a greater affect on the anterior stability of the joint. The size of the tear had a greater effect when it was located in the rotator interval than in the critical zone.

From this understanding of the static and dynamic stabilizers of the joint, one can separate 4 pathological conditions or groups that occur when the static, the dynamic, or both components are deficient: Groups I and II, external and internal impingement in the overhead athlete; group III, Primary anterior dislocation in the older patient is frequently; and group IV, Rotator cuff tear arthropathy.

EXTERNAL IMPINGEMENT AND THE OVERHEAD ATHLETE

In the mid-’80s, Tibone and Jobe discussed the frustration of treating impingement pain in the young overhead athlete with a decompression. While Tibone and Jobe reported excellent pain relief in this population, unfortunately only 46 percent were able to return to their pre-entry-level of activity, and only 25 percent with capsular laxity or labral pathology did so. Tibone and Jobe’s work identified the concept of mild instability leading to and presenting as an impingement problem.

Clinical Presentation

Pain is the presenting complaint with all of these patients. Few if any have a sense of instability. The classic impingement sign of Neer and Hawkins is almost always positive. The apprehension sign is pain-
ful but not apprehensive. The Jobe relocation test is often positive and an asymmetrical positive sulcus sign is occasionally present.

**Treatment**

Treatment always begins with rest, anti-inflammatory medications, and oftentimes a cortical steroid injection into the subacromial bursa. The goal of this initial treatment is to break the cycle of pain/inflammation with the bursa and rotator cuff tendons to allow the patient an opportunity to rehabilitate effectively. The entire shoulder musculature, including the scapula stabilizers, must be rehabilitated. Tight posterior capsular structures need to be stretched. If surgery is necessary, it begins with an examination under anesthesia and arthroscopy. Evidence of instability can be subtle. The arthroscopic examination must include the bursa. If the bursal tissue is hypertrophied, it must be cleaned out. The bursal side of the rotator cuff and the coracoacromial ligament must be evaluated. If no damage is found, decompression is not justified. Partial tears of the rotator cuff, bursal or articular, in a young individual usually require repair. The instability problem needs to be dealt with in a fashion that will return the athlete to full strength, full motion, and normal function. As Tibone and his associates discovered, pain relief and mobility alone are not adequate measures of recovery. Bankart lesions must be repaired, and stretched ligaments should be plicated. Jobe has emphasized that the plane of plication should be superior to inferior without disruption of the subscapularis muscle. His results were excellent: 92% good or excellent. Jobe’s goal was full motion by 3 months and full sporting activities at 6 months. The temptation is to try to reproduce these results arthroscopically with a Bankart repair to restore the anatomy and plication of the capsule or more commonly thermal energy to achieve the same goal. Although this is possible, failure rates with this approach are worrisome.

**INTERNAL IMPINGEMENT**

This syndrome presents as a painful shoulder in an overhead athlete. Initially the symptoms are vague and present only during sports, but as the problem progresses pain can become constant and even disturb sleep. The clinical examination was spelled out clearly in a paper by Kevin Paley et al. Fifteen percent A/C joint pain, 26 percent positive Neer or Hawkins sign, 62 percent evidence of subtle glenohumeral instability as indicated by a positive relocation test, and 63 percent a painful apprehension test. Morgan and Burkhardt specifically noted an average of 25 degrees decrease in internal rotation on the affected side.

**Anatomy**

Both arthroscopic findings as well as laboratory dissections have indicated that contact between the posterior superior labrum and the rotator cuff is both common and physiological. Helbrecht used a gadolinium enhanced MRI in a study comparing throwing and non-throwing shoulders. He noted that abnormalities of rotator cuff and superior labrum could be seen in completely asymptomatic throwing shoulders. His results led him to caution against treating the young overhead athlete on MRI findings alone.

**Arthroscopic Findings**

The arthroscopic findings in internal impingement can include any of the following: 1) damage to the posterior superior labrum, 2) damage to the rotator cuff, 3) damage to the posterior humeral head in the area of the insertion of the supraspinatus tendon, and 4) damage to the anterior glenoid labrum and/or inferior glenohumeral ligament. In addition, Jobe would add a normal or near normal subacromial bursa.

**Pathology**

The difficulty in dealing with internal impingement comes in the strong differences of opinion as to the pathomechanics of the problem. Morgan and Burkhart are adamant that the problem starts and ends posteriorly. They described the pathology as starting with a tight posterior capsule leading to a posterior superior shift of the G/H rotation point, which results in a “peeling off” of the posterior superior glenoid labrum which results in what they term as “pseudo-laxity.” If conservative measures fail, their logical treatment is first a course of posterior capsular stretching followed by arthroscopy and repair of the posterior type II SLAP. Many physicians, notably Walch, have not found instability (pseudo or otherwise) to be associated with this disorder and often find damage but not avulsion or peeling off of the posterior labrum.

C. Jobe described the problem of internal impingement as an excess of external rotation leading to posterior impingement and overload of the posterior labrum and rotator cuff. Jobe recommended that physical therapy should include exercises that strengthen the rotator cuff muscles and the scapula stabilizers as
well as coordination exercises. He also suggested that if conservative measures fail, surgery should consist of an examination of anesthesia, an arthroscopic examination, debridement or repair of the rotator cuff as indicated, and anterior capsular labral stabilization if necessary.

Conclusion

This topic was featured in Arthroscopy “Point Counterpoint.” The discussion was presented as if there were one answer for this disorder. That may not be the case. There may be different pathological problems, which present with similar symptoms and arthroscopic findings. It seems as if the most predictable approach is for the surgeon to understand that the symptoms that present as internal impingement can have any of a number of disorders, including a type II SLAP, anterior capsular labral abnormalities, and rotator cuff damage, as the major disorders. Each must be dealt with as the situation presents.

PRIMARY ANTERIOR SHOULDER DISLOCATION IN THE OLDER PATIENT

The third circumstance that emphasizes the relationship between glenohumeral instability and the rotator cuff is found in the patient who is ≥40 years of age when he/she suffers a primary anterior shoulder dislocation. In their review of 500 primary anterior shoulder dislocations, Rowe and Sakellarider pointed out that the frequency of a primary dislocation is as common for those over the age of 45 years as it is for individuals under 45. In this study, they said that while the recurrent rate was much greater in the younger patient, complications were both more frequent and more severe in the older patient. The 3 complications that seemed to increase with age are: 1) fracture of the greater tuberosity or glenoid, 2) axillary nerve injury, and 3) rotator cuff tear. The third complication is the most pertinent to this discussion. Many have recognized the association of rotator cuff tear with a primary dislocation in an older patient. Frequency of 30-90% has been reported in this age group.

Robert and Tom Neviaser reported on 12 patients with recurrent instability following a dislocation that occurred after the age of 40. In the 11 patients who were suffering from anterior recurrent instability, the surgical findings revealed an isolated subscapularis tear associated with an avulsion of the capsular ligaments from humerus (HAGL). In a separate study, Robert, Tom, and Jules Neviaser reported on 31 older patients who were unable to abduct the involved arm after a primary dislocation. All were found to have a rotator cuff tear. In each case, the patient was presumed to have an axillary nerve injury, yet only 4 of the 20 EMGs done to document the injury confirmed that the axillary nerve was injured. Berbig et al. offer the same caution: “if the patient is unable to elevate the affected arm > 90 degrees in plane of the scapula two weeks after a dislocation there should be a high suspicion of rotator cuff tear.”

The conclusions that one can draw from these are:

1. The combination of a rotator cuff tear with a primary dislocation in a patient over 40 is common.
2. Patients who do not recover quickly (within 3 weeks), especially in terms of active abduction, should be evaluated for a rotator cuff tear.
3. Subscapularis tears and capsular avulsion from the lesser tuberosity seem to be disproportionately common in terms of surgical findings.
4. In a patient suffering from the combination of recurrent instability along with a torn rotator cuff, satisfactory surgical results can only be obtained if both problems are dealt with.

ROTATOR CUFF TEAR ARTHROPATHY (RCTA)

This disorder was described 140 years ago by Smith and Adams. It wasn’t mentioned again until Codman described it in his 1934 monograph on shoulder disorders. He looked at the problem as the end result of untreated rotator cuff disease. RCTA was not mentioned again until the 1960s. Then over the next 20 years it was rediscovered and renamed on several occasions. Finally in 1983 Dr. Neer et al. gave it the name that has stuck: rotator cuff tear arthropathy.

Pathology

While the index injury for this disorder seems to be a rotator cuff tear, only a small percent (4%) of a group of patients whom Dr. Neer followed over an 8-year period developed rotator cuff arthropathy. The common denominator does not seem to be the size of the tear so much as the loss of balance in the transverse and coronal plane of the force couples, generated by the rotator cuff. Dr Neer postulated that only the tears that progressed to the loss of the primary and secondary stabilizers of the shoulder joint would develop RCTA.

This is an odd disorder that progresses in an oppo-
site fashion as rheumatoid arthritis. It begins as a degenerative arthritis and ends as an inflammatory arthritis. The inflammation is the result of a reaction to calcium phosphate crystals in the synovial and synovial fluid. These crystals cause a low grade inflammatory reaction by the synovial tissue, which induces the synovium to synthesize proteolytic enzymes that are responsible for the degradation of collagen material, causing damage to both the articular cartilage and the rotator cuff.

**Treatment**

Although the discussion of rotator cuff arthropathy is beyond the scope of this presentation, it is important to recognize the shoulder in which the force couples have been "unlinked," or as Dr. Burkhart refers to it "biomechanically unstable." If the shoulder is biomechanically unstable, poor treatment decisions will only accelerate the process. Decompression without restoring biomechanical stability to the rotator cuff frequently makes matters worse. Repair or replacement without decompression are far safer routes to take. The key to successful treatment is the recognition that the primary and secondary stabilizers of the shoulder joint along with the rotator cuff need attention.

**ANNOTATED REFERENCES**

   
   This is one of the first papers to identify that impingement pain in an overhead athlete may be a more complex problem than in an older nonathletic individual. They report an 89 percent improvement in the symptoms of pain but only 43 percent of their patients were able to return to their preoperative athletic level. Within that group of athletes, no swimmers recovered to their pre-injury level and only 22 percent of the baseball pitchers and throwers recovered.

   
   Jobe et al. relate that most instability operations are more predictable in terms of restoring stability than in terms of maintaining full external rotation. This becomes pertinent with the overhead athlete. The goal of the capsular labral reconstruction is to offer a safe, predictable, reproducible surgical procedure that offers stability as well as maintains a full range of motion. His surgical procedure does not violate the subscapularis insertion and tightens the capsular structures primarily on a superior to inferior direction. The results of 76 patients at an average of 39 months postoperative were: 96 percent satisfied with procedure, 93 percent return to preoperative athletic level. The average loss of external rotation at 90 degrees of abduction was 2 degrees while the loss of abduction was 1 degree.

   
   This article focuses on the pathomechanics of internal impingement. Two differing views are featured. Chris and Frank Jobe both favor anterior laxity or increased external rotation as the primary abnormality that leads to internal impingement. Burkhart and Morgan favor a tight posterior capsule which prevents normal external rotation and shifts the fulcrum of rotation superioposterior to the insertion of the biceps tendon causing a "peel back" injury of the biceps insertion into the posterior labrum. They indicate that the instability is "pseudolaxity" and will disappear with repair of the SLAP injury. Since both authors have been successful in treating this problem, yet with differing approaches, it leaves room for the possibility that the single problem may have more than one pathomechanical pathway.

   
   This is a report of 12 patients with recurrent instability following their index dislocation, which occurred after the age of 40 years. Eleven of the patients with anterior instability all had a rupture of the subscapularis muscle as well as a humeral avulsion of the glenohumeral ligaments. The main message from this article is that the pathology for recurrent dislocations in this age group is frequently not the routine Bankart injury as seen with younger individuals.

   
   The authors describe a condition of the shoulder in which a tear of the rotator cuff progresses in size and in location to the point where the force couples are disrupted and secondary instability ensues. The degenerative process, which results from this situation, creates basic calcium phosphate crystals, which cause an inflammatory condition that frequently results in severe bony deformity. This is a thorough overview of rotator cuff tear arthropathy and would be an excellent article for any individual interested in a deeper understanding of this disorder.

   
   Burkhart emphasizes the importance of looking at the rotator cuff as a three-dimensional structure. He emphasizes that the length of a tear is not nearly as important as is its effect on "force couples." The loss or the maintenance of force couples on the frontal (deltoid balanced by the intact portion of the rotator cuff), and the transverse plane (the subscapularis balanced by the infraspinatus and teres minor), will allow the surgeon a better biomechanical view of the effect of a tear on the shoulder. This paper offers a logical approach to assess and plan treatment for a rotator cuff tear.