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INSTRUCTIONAL COURSE LECTURES

VOLUME 74 2025

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American Academy of Orthopaedic Surgeons

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PREFACE

The Instructional Course Lectures (ICLs) and Symposia that were presented at the 2024 Annual Meeting of the American Academy of Orthopaedic Surgeons (AAOS) in San Francisco, California, continued the tradition of providing innovative information related to orthopaedic education and the diagnosis and management of musculoskeletal disorders. *Instructional Course Lectures*, *Volume 74*, represents concepts curated from those 2024 lectures, written by some of the most experienced and respected subspecialty experts in the field of orthopaedic surgery.

I would like to thank all of the authors of the various chapters for their timely diligence in writing helpful educational information. I would also like to express my gratitude to the chairs and members of the specialty Instructional Course Committees who served as section editors for this volume. I also appreciate the great efforts of the course operations and editorial staff at AAOS and Wolters Kluwer, without whom this work could not be accomplished.

I would like to thank Xinning Li, MD, FAAOS, FAOA, assistant editor, for his hard work in performing so many tasks during this process and reviewing many of the chapters with me. I hope you enjoy *Instructional Course Lectures, Volume 74*, which is intended to be an innovative resource you will frequently return to as you continue to practice orthopaedic surgery.

Carolyn M. Hettrich, MD, MPH, FAAOS, FAOA
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CONTENTS

Section 3: Hand and Wrist

Section 4: Foot and Ankle

Section 5: Adult Reconstruction: Hip

Section 6: Adult Reconstruction: Knee

VIDEO ABSTRACTS

Chapter 5 Ulnar Collateral Ligament Repair

Video 5.1: Ulnar Collateral Ligament Repair Augmented With Synthetic Tape

This video reviews the case presentation of a Division I male javelin athlete who sustained an acute ulnar collateral ligament tear during competition and underwent primary repair via synthetic tape augmentation.

Chapter 6 Medial Ulnar Collateral Ligament Tears: Reconstruction

Video 6.1: Ulnar Collateral Ligament Reconstruction of the Elbow: The Docking Technique

This video demonstrates reconstruction of the ulnar collateral ligament via the docking technique. If precise surgical steps are followed and postoperative rehabilitation is appropriately progressed, the procedure reliably returns athletes to competitive throwing. In addition, the video describes key takeaway points and clinical outcomes for the procedure.

Chapter 15 Tendinitis and Tendinopathies About the Elbow and Forearm

Video 15.1: Radial Tunnel Injection

An ultrasonographic cross section of the forearm for radial tunnel injections is shown. The patient is positioned with the arm supinated and the probe placed directly over the volar surface of the forearm. This can serve both diagnostic and therapeutic purposes, and can be performed during any clinic visit without the need for anesthesia.

Video 15.2: Ultrasonographically Guided First Dorsal Compartment Corticosteroid Injection

This is performed with the probe over the radial aspect of the wrist with arm rotation in neutral. The tendons of the compartment are easily visible, and the procedure takes little time to become proficient.

Chapter 22 Preventing Complications in Complex Repeat Revision Total Knee Arthroplasty: Advanced Exposure, Implant Removal, and Handling the Extensor Mechanism

Video 22.1: Revision Total Knee Arthroplasty: Exposure and Component Removal

This video demonstrates the key steps for exposure and removal of well-fixed components in a knee in which surgery has been previously performed. The video begins by reviewing the vascular supply to the anterior knee and how to select a surgical incision. The video then discusses the approach to systematically expose the knee in a stepwise manner, which includes clearing the medial and lateral gutters, performing anteromedial tibial release, and mobilizing the extensor mechanism. The video also describes advanced techniques to mobilize the patella, which include inside-out lateral release and the quadriceps snip. Removal of various polyethylene inserts is shown to demonstrate the variability of insert locking mechanisms.

The second portion of the video demonstrates removal of well-fixed femoral and tibial components. The video compares the various steps required to remove cemented and noncemented components. The final portion of the video discusses tips and tricks for removal of a fully cemented, stemmed tibial component. Via a first-person intraoperative perspective, the video demonstrates how to carefully manipulate high-speed burrs and trephines to remove implants while minimizing bone loss.

Chapter 28 Nutritional Intervention to Improve Outcomes for Orthopaedic Surgery

Video 28.1: Nutrition Interventions to Improve Outcomes for Orthopaedic Surgery

This video highlights the critical role of perioperative nutritional optimization in improving postoperative outcomes for patients undergoing orthopaedic surgery. It examines the hypermetabolic demands triggered by the surgical stress response.

Implementing targeted nutritional interventions, such as carbohydrate loading and amino acid supplementation, can reduce complications, support muscle recovery, and enhance wound healing. Addressing the increased nutritional demands of surgical patients is essential to decreasing complications and improving overall outcomes.

Chapter 31 Management Principles of Civilian Ballistic Orthopaedic Trauma

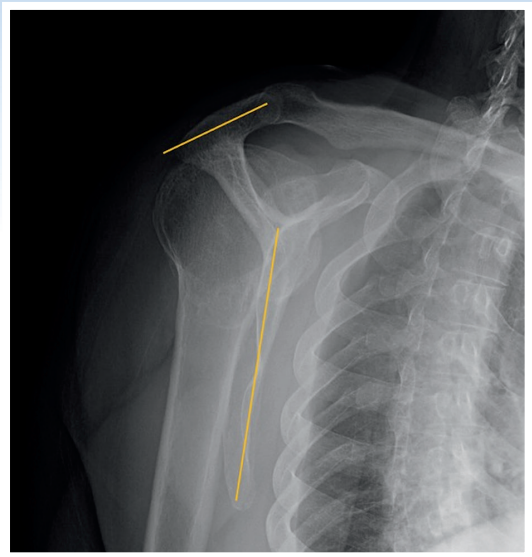
Video 31.1: Achieving Limb-Length Equality in Femoral Shaft Fractures

This video describes multiple techniques for assessing and restoring limb length in the setting of femoral shaft fractures.

SECTION

1

Shoulder and Elbow



CHAPTER

1

Complications in Shoulder Arthroplasty: Why Do They Happen and How to Manage Them

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ABSTRACT

Complications occur with total shoulder arthroplasty (TSA), and they can be daunting to diagnose and treat. It is important to review common TSA-related complications and to summarize risk factors along with causes of these complications and how to avoid them. The orthopaedic surgeon should be knowledgeable about how to successfully manage complications to achieve good patient outcomes and the etiologies and management of the painful and stiff shoulder arthroplasty, subscapularis failure after anatomic TSA, instability after reverse shoulder arthroplasty, and acromion stress fractures in the setting of reverse TSA.

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Introduction

The number of total shoulder arthroplasties (TSAs) performed annually in the United States is rapidly increasing, with a predicted increase of 349% overall between the years 2011 and 2025, with a dramatic increase of 546% for reverse TSA versus a smaller 66% increase in anatomic TSA. This increase is multifactorial, with key factors including the expanded indications for reverse TSA, significant progression in implant options, and subspecialty training, as well as an aging population. In general, implant survivorship is improving, and overall outcomes are excellent, with substantial improvement in patient quality of life. However, with an increasing number of arthroplasties being performed, there is also an unfortunate corresponding increase in the number of patients with pain, limited function, or substantial complications. In a review of patients older than 70 years, complications occurred in 10.2% of patients who underwent anatomic TSA and 9.9% of patients who underwent reverse TSA, with revision required in 2.3% and 2.2% of patients, respectively. With anatomic TSA, the most

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common complications were rotator cuff tear (3.7%) and glenoid loosening (2.9%). By contrast, with reverse TSA, the most common complications were acromial stress fractures (6.1%) and nerve injury (3.1%). Other less common complications are shared between anatomic TSA and reverse TSA, including fracture, instability, hematoma, and infection. It is important to review the several common complications unique to both anatomic TSA and reverse TSA.

Evaluation and Management of the Painful and Stiff Shoulder Arthroplasty

The outcomes of anatomic TSA are historically good; however, many of the long-term outcome studies are based on implants that are no longer used. In general, outcomes depend on many factors, such as patient age, with several studies showing worse outcomes in younger patients.[1] Glenoid morphology is an important factor, with worse outcomes in patients with more advanced glenoid deformities such as the B2 glenoid.[2] The preoperative diagnosis affects outcomes such that fracture sequelae and dislocation arthropathy lead to worse outcomes. Glenoid and humeral design and surgeon factors are crucial in outcomes. The overall complication rate is approximately 10%, with infection, component loosening (primarily glenoid), instability, rotator cuff failure, and neurologic injury as the primary issues.[3]

In some cases, complications and reasons for poor outcomes of shoulder arthroplasty are obvious. However, at times, the evaluation of the painful and stiff shoulder replacement is complex, and arriving at the proper diagnosis can be a challenge. This chapter will focus on diagnosis and management of the painful shoulder arthroplasty, with emphasis on infection, generalized stiffness, stiffness caused by nonanatomic components, superior rotator cuff failure, aseptic loosening of components, and neurologic causes (**Table 1**).

Infection

Cutibacterium acnes is the most common bacterium causing periprosthetic joint infection (PJI) in the shoulder (38.9% of shoulder PJI cases); however, unlike the more obvious *Staphylococcus* infections, *C acnes* infections typically present later with subtle findings such as pain and stiffness, and thus can present a diagnostic challenge.[4] Patients undergoing revision shoulder arthroplasty with unexpected positive cultures for *C acnes* commonly presented with both pain and stiffness. Risk factors for *C acnes* include male sex, younger age, and revision surgery. *C acnes* is not associated with the risk factors typically associated with PJI caused by more virulent organisms such as *Staphylococcus*, such as smoking history, higher body mass index, diabetes, or immunocompromised conditions. Radiographically concerning findings

TABLE 1
Causes and Workup of Painful and Stiff Anatomic Total Shoulder Arthroplasty

Etiology	Diagnostic Testing	Treatment
Infection (*Cutibacterium acnes*)	Labs (ESR/CRP/CBC) often normal	One-stage versus two-stage revision, antibiotics
	Synovial fluid (IL-6, alpha defensin, culture hold for 14 d, cell count)	Can consider arthroscopic biopsy and diagnostic arthroscopy
	Radiography, CT (humeral lucency)	—
Idiopathic (noninfection) stiffness	Rule out infection	Nonsurgical treatment with physical therapy
	Evaluation of preoperative radiographs and range of motion, surgical notes/technical factors	Arthroscopic release and biopsy
	Radiography and CT for humeral or glenoid overstuffing	Revision arthroplasty (often to reverse shoulder arthroplasty)
Superior rotator cuff failure	Radiography	Nonsurgical treatment with physical therapy
	CT arthrography or MSK ultrasonography	Revision to reverse shoulder arthroplasty
Neurologic injury	Physical examination	Observation
	EMG/NCV	Consideration of nerve releases or transfers in select cases

CBC = complete blood count, CRP = C-reactive protein, EMG = electromyography, ESR = erythrocyte sedimentation rate, IL = interleukin, MSK = musculoskeletal, NCV = nerve conduction velocity

for PJI include radiolucent lines, osteolysis, bone erosion, endosteal scalloping, new periosteal bone formation, and component shifting. However, the most common findings consistent with *C acnes* have been humeral loosening and humeral osteolysis. It has been shown that routine serum laboratory tests such as the traditional serum white blood cell count, erythrocyte sedimentation rate, and C-reactive protein are not very helpful in identifying low-virulence organisms such as *C acnes* because these values are typically normal.[5] Synovial fluid cytokine analysis has been shown to be more sensitive in identifying *C acnes* compared with other tests, in particular synovial interleukin 6 and alpha defensin.[6] Although obtaining synovial fluid can be challenging, it is critical to attempt aspiration via either fluoroscopy or ultrasonography for the best accuracy, and it should be noted that even if the aspiration is dry or the fluid obtained yields negative cultures, this analysis does not rule out infection. If adequate fluid is obtained for culture, in general, the faster *C acnes* grows the more likely the result is truly positive versus a probable contaminant if growth is seen after 11 days or longer. Because shoulder PJI thus often cannot be excluded based on the aforementioned testing, obtaining synovial tissue for biopsy has been proposed by the International Consensus Meeting on Orthopaedic Infections as possibly a more reliable way of identifying the more fastidious organisms such as *C acnes*.[4] One of the benefits of an arthroscopic biopsy is that it has been shown to be curative, up to 46% in some cases, and allows recognition of other pathology that may be present but has eluded detection, such as rotator cuff tears and component loosening. Studies have shown that arthroscopic biopsies have good diagnostic accuracy with high sensitivity and specificity, although additional studies are needed to better define the role of arthroscopy and standardize the biopsy protocols. When infection has been confidently ruled out as the cause of persistent pain and stiffness, several other diagnoses should be explored.

Stiffness That Is Not Caused By Infection

Idiopathic stiffness is uncommon after anatomic TSA and is most often caused by a difficult-to-diagnose infection. It is important to understand what the patient's preoperative range of motion (ROM) was, because this is often a reflection of postoperative mobility, and patients with significant preoperative stiffness may experience improved ROM but not achieve that of more mobile patients, but this is usually not a painful problem for patients. If the loss of ROM is more a loss of external rotation, it is important to reflect on the way the subscapularis was repaired because it is possible to overtension the repair when performing a peel or tenotomy if the arm is in internal rotation. Restoration of anatomy is critical for success after anatomic TSA, and several studies have highlighted the potential for inaccurate humeral head cuts leading to shifting of the humeral center of rotation (COR) or oversizing of the humeral components, both of which can essentially overstuff the joint and lead to pain and decreased mobility (**Figure 1**). Several studies have shown that short stem and stemless implants are more prone to varus positioning, and more likely to cause significant deviations from the COR (leading to inaccurate restoration of proximal humeral anatomy).[7,8] The level of the humeral head cut was found to be the reason for 80% of the cases of overstuffing. However, other studies have shown that the use of stemless implants allows a surgeon to better respect the native humeral anatomy, leading to a more routine approximation of the COR. Either way, given the known risk of poor positioning of the humeral component, careful preoperative templating, intraoperative attention, and potential consideration of the use of an intramedullary guide have been suggested as ways to lower this risk. Although occurring less frequently, it is also possible to overstuff on the glenoid side since the advent of augmented glenoid components. Studies have shown that leaving the glenoid in less than 10° to 15° of retroversion leads to good outcomes, and thus the surgeon should not attempt to correct to 0° of retroversion, especially in the significant B2 glenoids. Preoperative and postoperative images should be analyzed for overlateralization of the joint line (as a result of too large of a glenoid component) as a potential cause of pain and stiffness along with the more common humeral overstuffing.

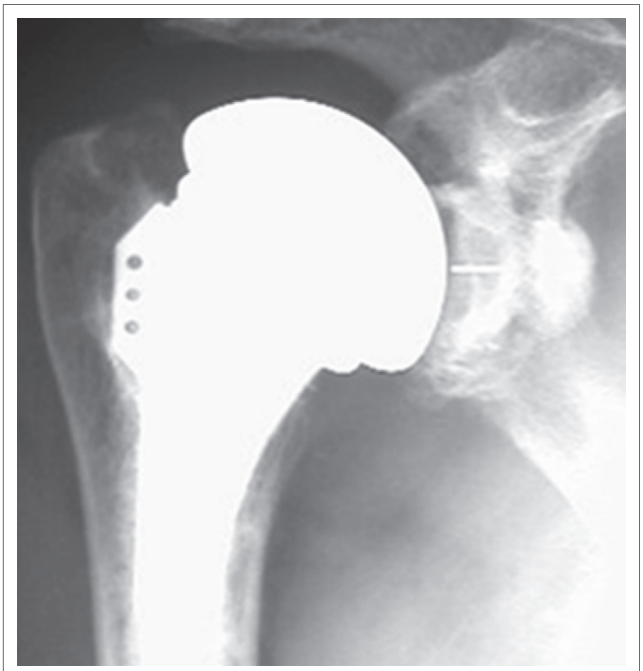

FIGURE 1 AP radiograph showing an anatomic total shoulder arthroplasty, with clear evidence of humeral overstuffing and hence a nonanatomic restoration of the humeral center of rotation.

Superior Rotator Cuff Failure as a Source of Pain and Stiffness and Lack of Function

The rate of superior cuff failure after anatomic TSA is likely higher than reported and can range from 16.8% up to 30% based on the study and the length of follow-up.[9] Certainly, to some degree, with an aging population, failure of the superior cuff may be unavoidable, but efforts to effectively restore the humeral head COR and avoid joint overstuffing will minimize the strain on the rotator cuff. The consequences of superior cuff failure, in addition to pain and loss of function, include glenoid component loosening caused by the rocking horse phenomenon, as well as possible joint instability.

The presentation can sometimes be a traumatic event that leads to superior cuff failure and loss of function, but more often physical examination reveals progressive pain, weakness, and loss of active ROM, which eventually can lead to loss of passive ROM as well. In chronic cases, radiographs will often show superior migration of the humeral head, but this is often not the case in more acute events. In these cases, CT arthrography or musculoskeletal ultrasonography can be diagnostic. Treatment typically starts with physical therapy and symptomatic management. However, when these modalities fail, often the most reliable surgical intervention is conversion to reverse TSA.

Neurologic Injury as a Source of Pain and Stiffness

The risk of neurologic injury after anatomic TSA is much lower than that for reverse TSA; in one study looking at 19,262 shoulder arthroplasties, the risk was more than twice as common in reverse TSA as anatomic TSA, with an overall rate of 0.4%.[10] In anatomic TSA, the most common scenario when a neurologic injury would occur is due to stretch-induced neurapraxia during glenoid exposure or extremes of external rotation during humeral preparation. Careful retractor placement and intermittent relaxation of retraction has been shown to be helpful in minimizing this potential complication. Although an overt injury is typically obvious, with a careful physical examination indicating numbness, tingling, and muscle atrophy, more subtle neurapraxias may not be as obvious and require a level of clinical suspicion, especially if the patient's pain levels and function are not improving as anticipated. Electromyography and nerve conduction studies can be performed to confirm the diagnosis and also provide valuable information regarding recovery potential and treatment options.

Aseptic Loosening of Components

The rate of aseptic humeral component loosening with standard-length stems has been historically very low, such that it has been considered as almost pathognomonic for underlying infection, hence the weight given to defining shoulder PJI by the International Consensus Meeting on Orthopaedic Infections in 2018.[4] However, with the advent of shorter stems and stemless components, recent studies have shown that the rate of aseptic humeral loosening is higher than once thought, reaching 10.9% in one study.[11] Therefore, the patient presenting with pain and decreased mobility with radiographic signs of humeral component loosening, which once may have been attributed only to infection, should be carefully considered for potential humeral loosening caused by stress shielding and stem design. This appears to have been more prevalent in the first-generation short stems, because studies show that this condition is diminishing in the more recent second-generation stems. Aseptic glenoid component loosening is much more common; there are similar rates of loosening between keeled and pegged implants, with some studies showing rates up to 43.8% based on the length of follow-up. Glenoid loosening has been shown to be among the most common reasons for revision of anatomic TSA, and initial bone stock, glenoid morphology, and initial component positioning play a large role.[12] Patients with humeral or glenoid loosening may present with pain and stiffness, most often occurring years after the arthroplasty. Infection, such as with *C acnes*, is a potential cause and should be evaluated. Advanced loosening is typically obvious on radiographic evaluation with progressive lucencies around the glenoid component; however, for more subtle loosening, advanced imaging studies such as CT or bone scan may be indicated (**Figure 2**). CT arthrography can show the dye under the glenoid and around the pegs or keel, indicative of loosening. The surgeon should also recognize that polyethylene wear, with or without an overtly loose glenoid, can lead to pain and loss of function for a patient.

Anatomic TSA: Subscapularis Failure—Why Did This Happen and What Can Be Done?

Subscapularis failure is the second most common reason for failure of anatomic TSA following glenoid component failure.[13] The subscapularis is the most common rotator cuff tendon to fail after TSA. Early revision within 1 year after the initial surgery is most commonly due to instability, followed by rotator cuff failure and component loosening.[14] The subscapularis can fail acutely both traumatically or atraumatically or it can fail chronically in patients with persistent pain and dysfunction who initially recovered uneventfully from their shoulder arthroplasty procedure. Overall, subscapularis failure is common after anatomic TSA and is a potentially preventable complication. Early recognition and diagnosis are crucial to minimizing complications and morbidity of failures.

Subscapularis Management

There are four major steps in the management of the subscapularis tendon in anatomic TSA: takedown, mobilization,

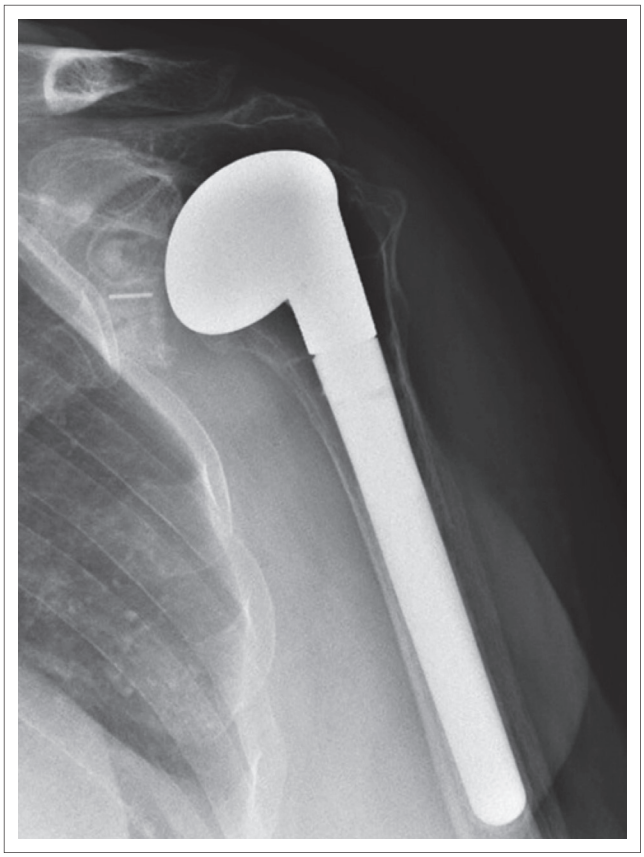

FIGURE 2 AP radiograph showing an anatomic total shoulder arthroplasty 10 years after placement, with clearly visualized radiolucent lines surrounding the glenoid pegs. At the time of revision, the glenoid was grossly loose with a significant cavitary defect.

repair, and rehabilitation. These four variables all are modifiable and potentially influence healing and complication rates. The three main options for subscapularis takedown are tenotomy, lesser tuberosity osteotomy (LTO), and peel-off. In addition, there are many described technical variations in both takedown and repair strategies. The reported maximum internal rotation force of the subscapularis muscle is 250 N.[15] Many biomechanical studies have been conducted to evaluate the initial load to failure of various repair techniques. LTO strength has been reported from 447 to 738 N,[16-20] tenotomy from 252 to 487 N, and peel-off from 431 to 683 N. It is clear from these large, overlapping ranges that there is considerable heterogeneity in these data, and there is no clear consensus on which repair type is the strongest. One purported benefit of LTO is the ability to monitor healing radiographically during the postoperative period. Although this is beneficial in some cases, it is not always visible or useful. There have been multiple randomized controlled trials aimed at identifying the ideal subscapularis repair technique. Overall, no difference has been shown in strength, American Shoulder and Elbow Surgeons or Western Ontario Osteoarthritis of the Shoulder Index scores, healing, or fatty infiltration. Currently, there is no gold standard technique for subscapularis repair in TSA.[21] It is possible that further study and technique development will lead to a preferred method in the future.

Subscapularis Failure

It is important to note that failure of the subscapularis after TSA is not always readily identifiable and can occur in different ways. The tendon may never heal, or tear after healing. It may also attenuate, denervate, or atrophy because of fatty infiltration of the muscle belly. This process is often unrecognized. Failure is common both traumatically and atraumatically. Failure can also result from implant malposition, leading to tension on the rotator cuff or altered kinematics.

Diagnosis of subscapularis failure begins with a thorough history and physical examination. History of a pop or sudden pain in the anterior shoulder with or without trauma is a red flag for possible subscapularis failure. Physical examination should focus on typical subscapularis muscle evaluation, including the belly-press, bear hugger, and lift-off tests. There may also be increased passive external rotation and possible anterior laxity with positive apprehension. If history and physical examination findings are consistent with possible subscapularis repair failure, the diagnosis should be confirmed with imaging. The tendon can be evaluated with radiography identifying subluxation or LTO displacement, ultrasonography, or CT (**Figure 3**). MRI is frequently of lesser value because of metal artifact. These modalities all vary greatly in sensitivity, specificity, interreader reliability, and cost. The imaging modality of choice will depend on a multitude of factors including physician preference and system availability.

Treatment of Subscapularis Failure

Treatment for subscapularis tendon failure typically falls into one of five categories: nonsurgical, primary repair, tendon reconstruction or augmentation, tendon transfer, or revision to reverse TSA. Some patients may do well with nonsurgical care, especially if they are lower demand and centering of the glenohumeral joint can be maintained.

Primary repair can be challenging and frequently is hampered by poor tendon quality. Often, there is type 2 failure at the musculotendinous junction or midtendon, which is also less likely to heal with primary re-repair. Repair can be considered in young patients with an acute tear addressed urgently, but it is not reliable. Risk factors that lead to tendon failure in the first place, such as poor tendon quality, patient comorbidities such as smoking and diabetes, or patient noncompliance are likely to hamper this attempt at

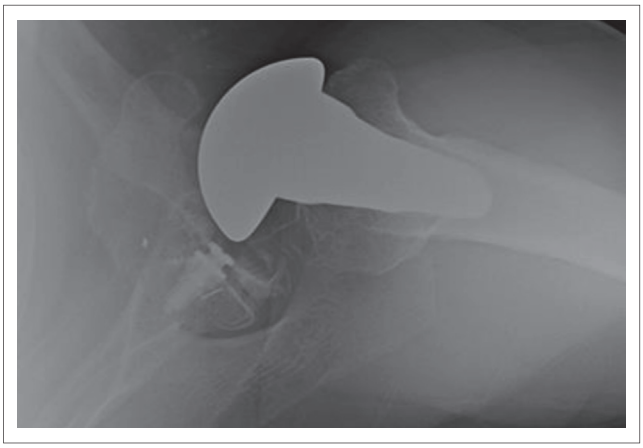

FIGURE 3 Axillary radiograph from a patient with subscapularis rupture and anterior subluxation of the humerus after total shoulder arthroplasty.

repair as well. Overall, an attempt at primary repair after subscapularis failure may work in young, active patients, but has a high rate of recurrent failure.

A second surgical option for subscapularis failure after TSA is tendon reconstruction or augmentation. Commonly used grafts include Achilles tendon or dermal allografts. It is challenging to adequately secure and fix the graft to both the retracted tendon and the humerus and to obtain appropriate tension. This can provide more robust fixation than primary repair and can also be considered in young, active patients with an acute rupture. This can be made as an intraoperative decision if the surgeon is planning for primary repair and is faced with inadequate tendon quality or mobilization. An alternative to augmentation is to use a graft as a capsular reconstruction attaching the medial graft to the glenoid.

Tendon transfer of the pectoralis major or latissimus dorsi is an additional option for subscapularis-deficient shoulders. Of the two tendons, the pectoralis major tendon has traditionally been used more, but latissimus dorsi tendon transfer is becoming more prevalent. It is noted that this treatment also can fail and is more likely to lead to complications if there is preoperative anterior subluxation of the humerus. The pectoralis major tendon transfer after TSA has been less successful than when used for subscapularis insufficiency alone.

The most predictable, definitive option for subscapularis failure after TSA is revision to reverse TSA.[22] Reverse TSA provides good, reliable functional outcomes in subscapularis-deficient shoulders. There may be challenges when revising well-fixed implants. This burden can be relieved somewhat with the use of convertible implants for TSA. Implant systems are available that in some cases can be converted to a reverse shoulder arthroplasty (RSA) without complete removal. This allows a glenosphere to be placed on a metal-backed glenoid component, and the humeral head component can be removed and a reverse humeral component can be placed with stem removal. Stemless, short stem, and convertible implants have reduced the burden of revision TSA to RSA.

Dislocation After Reverse TSA

Instability after reverse TSA is a common and devastating complication, affecting approximately 7% of all instances of reverse TSA. Initially, it was the most common complication usually within the first year after surgery, but in recent years it has been surpassed by PJI.[23] With improvements in reverse TSA design and technique, the incidence of early dislocation after reverse TSA is decreasing. Nevertheless, reverse TSA instability remains a difficult complication to correct because of high recurrent instability rates with nonsurgical treatment and high complication rates with revision surgery. There has been a 50% complication rate noted after revision surgery for reverse TSA instability, often including further instability.[24]

Who Is at Risk?

There are multiple risk factors for early reverse TSA dislocation (**Table 2**). The most significant risk factor is prior arthroplasty surgery, as well as prior proximal humerus fractures managed nonsurgically or with open reduction and internal fixation.[25] Other risk factors include young patients, subscapularis and posterior rotator cuff deficiency, bone deficiency, and elevated body mass index. The surgical indication may affect instability rates, and technical factors may serve as risk factors, including soft-tissue tension, diameter of the glenosphere, constraint on the humeral side, lateralization, and mechanical impingement. Finally, technical errors such as bony impingement, errors in version, and poor component placement such as a high baseplate position or superior inclination may predispose to instability. Axillary nerve dysfunction or acromion insufficiency or fracture may also contribute to reverse TSA instability. Understanding which potential risk factors contribute to instability for each patient will help with diagnosis and appropriate treatment individualized for each patient.

Boileau examined a series of patients with atraumatic instability after reverse TSA and found the mean time to dislocation was 3.4 weeks. In that demographic, 64% had prior surgery. Eighty-two percent of patients with instability were male. Eighty-two percent of patients who sustained dislocations were overweight. A total of 64% of the patients who experienced early dislocation had not undergone subscapularis repair.[24] Similarly, Cheung et al[26] examined a group of patients and showed that 9.2% with a reverse TSA had instability in the early postoperative period that occurred an average of 8 weeks after surgery. There was a higher risk in male patients who had prior surgery, and those with a history of fractures. The absence of a subscapularis repair was also an independent risk factor for dislocation after reverse

TABLE 2
Risk Factors for Instability After Reverse Total Shoulder Arthroplasty

Prior shoulder arthroplasty surgery
No repair or absence of the subscapularis tendon
Male sex
Elevated body mass index
History of proximal humerus fracture
Elevated superior inclination angle
Deltoid dysfunction
Humeral bone loss/deficiency
Acromion and scapular stress fractures
Bony or soft-tissue impingement
Upper extremity ambulation

TSA. Forty-five percent of those patients with an initial reverse TSA dislocation sustained a second dislocation that required additional surgery. In this cohort, initial treatment was closed reduction in nine patients. One patient underwent open prosthetic reduction, and another underwent open reduction using a thicker polyethylene insert. Closed reduction and polyethylene exchange were inadequate in 45% of patients requiring another revision with exchange to a larger glenosphere and thicker humeral inserts.[26]

Recognition of the Problem

Lack of Soft-Tissue Tension and Compression

Appropriate soft-tissue tension of the deltoid-acromion complex is critical for stability of reverse TSA, with biomechanical contributions including deltoid and acromion function, glenosphere size, a variety of implant designs that distalize or lateralize the humerus, and various degrees of medialization of the COR. Melbourne et al[27] reported that this is the most common cause of reverse TSA instability, with subcategories including undersized implants, loss of deltoid contour, humeral height loss, subscapularis deficiency, acromion fracture, and deltoid or axillary nerve dysfunction. Mismatch of these factors to patient anatomy and pathology may lead to instability because the deltoid and remnant rotator cuff do not adequately compress the humerus against the glenosphere. For instance, smaller glenospheres or lack of lateralization and distalization in larger male patients can create a construct that fails to appropriately tension the deltoid and contributes to reverse TSA instability. Revision surgery with larger, more lateralized or eccentric-inferior glenospheres, as well as increased humeral lateralization or distalization with thicker trays and polyethylene, can help to address soft-tissue undertensioning of the deltoid and achieve stability of the reverse TSA.

Subscapularis deficiency can also create dead space and lack of anterior constraint to dislocation. The research regarding the need to repair the subscapularis has been mixed, and there are some advantages to not fixing it (time in the operating room and early motion for the patient). Some suggest that a more lateralized implant is enough to prevent the need for subscapularis repair. However, there are multiple studies demonstrating a higher dislocation rate without subscapularis repair, such that the authors of this chapter recommend trying to repair the subscapularis if there is adequate tissue present.[28] Underlying deltoid dysfunction caused by axillary nerve palsy can also be a source of instability because the deltoid loses the ability to compress across the joint adequately.[29]

Significant bone deficiency on the humeral side, particularly in proximal humeral fractures or revision surgery, can lead to deltoid loss of contour and undertensioning and may require increased humeral height or options to reconstruct the proximal humerus bone with allograft or endoprosthesis.

Acromion and scapular stress fractures can cause gross lack of tension of the deltoid and can lead to dislocation of the prosthesis as a result of loss of the compression force of the deltoid. In the attempt to increase tension and with more lateralized designs, this risk is higher, and care should be taken to assess each patient and avoid overtensioning in those with osteoporotic bone to avoid acromion and scapular fractures (ASFs). In the unstable reverse TSA, open reduction and internal fixation of ASFs can be considered to improve stability, but their management is controversial and must be balanced with a high complication profile from open reduction and internal fixation of scapular and acromion fractures.

Loss of Containment

Loss of containment refers to failure of the glenohumeral articulation as a result of mechanical failure or eccentric polyethylene wear. Catastrophic mechanical failure of the

implant through trauma, aseptic loosening, metal fatigue, and glenosphere or polyethylene dissociation all can cause instability of the prosthesis. Each case is unique, and these signs should be looked for with radiography and CT. Many of these complications are less common with improvements in prosthetic design. Eccentric polyethylene wear can occur with scapular notching or glenoid component malposition superiorly inclined or located, as well as more often with varus neck-shaft Grammont-style designs. This can lead to late dislocation during which the loss of the inferior polyethylene allows the humerus to escape from the normal semiconstrained reverse TSA articulation with the glenosphere.

Impingement

Impingement is unfortunately a common cause of instability after reverse TSA. It can be due to soft-tissue or bony impingement that leads to levering of the glenohumeral articulation with ROM (**Figure 4**). Recognizing and avoiding soft-tissue and bony impingement at the index procedure is important to prevent later dislocation. Taking the arm through a ROM with the patient relaxed is a vital part of the procedure, and often the prosthesis can be seen to be levering out of position if impingement is present. Being careful to take down the bony osteophytes and accounting for malunited tuberosities from fracture sequelae can help prevent this. Awareness of a mismatch of the prosthesis circumference with the patient's native anatomy is also important. At the index procedure, taking time to trim down the extra bone overhanging the prosthesis, particularly posteriorly in large male patients, can help prevent this issue from occurring.

Soft-tissue impingement is at times more difficult to assess, but often in patients with prior instability, inferior and posterior palpation of the tissue is possible. Calcified rotator cuff tissue and scar tissue from prior trauma or surgery are contributing factors. Palpation of the area with a finger while taking the arm through a ROM with the trial implants in place helps the surgeon to identify this problem and will be a guide to indicate where excision is necessary.

Prosthetic malalignment can contribute not only to impingement of the prosthesis but also to dislocation. A glenoid baseplate placed too superior will cause the humeral component and polyethylene to impinge on the lower portion of the glenoid and/or scapular spine and cause direct levering out of the prosthesis. As mentioned previously, a baseplate placed in a superiorly tilted position can make the implant more likely to subluxate or dislocate.[30]

Addressing the Problem

It is important to recognize that not all reverse dislocations are the same and identifying the etiology is important before revision surgery. Familiarity with different reverse TSA designs can help the surgeon understand contributing

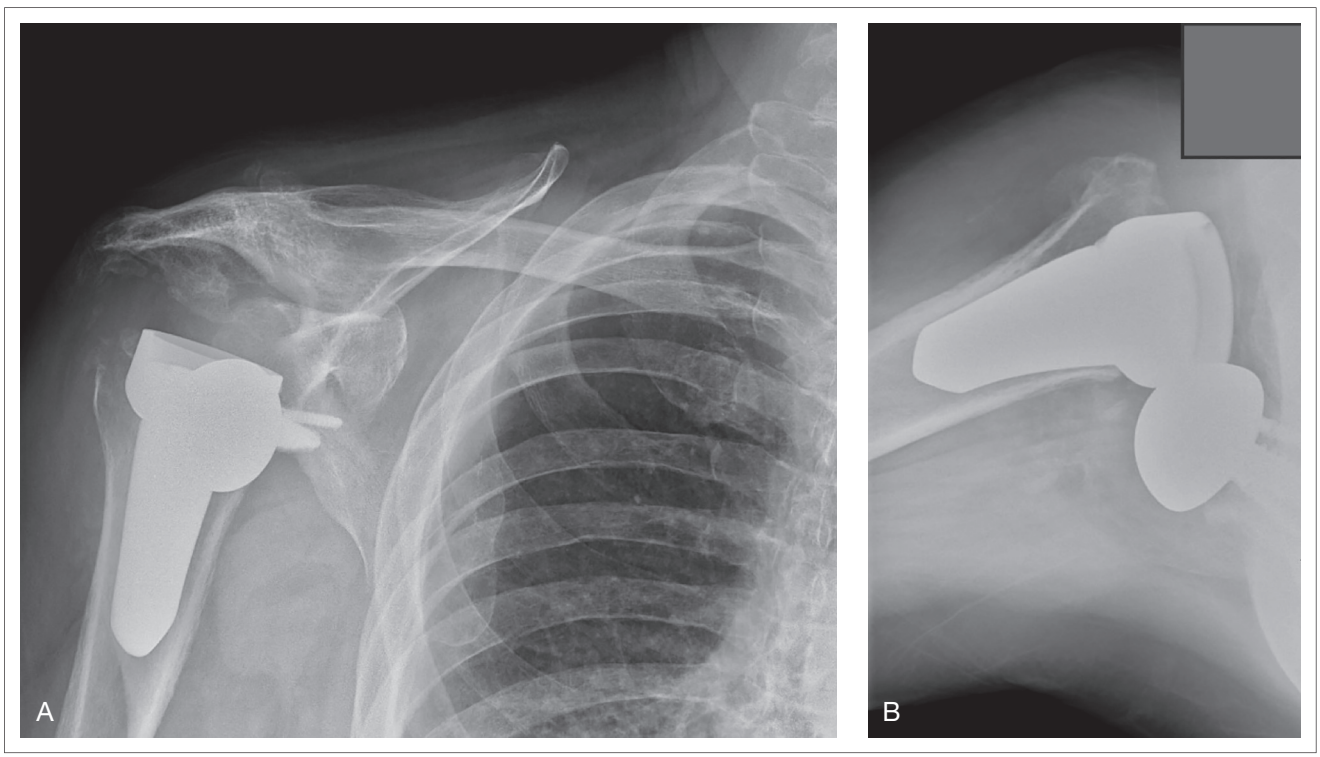

FIGURE 4 **A**, Preoperative AP radiograph showing reverse total shoulder with instability. **B**, Postoperative axillary radiograph obtained after revision with the repositioned lateralized baseplate, larger glenosphere, and constrained polyethylene.

factors for instability as well as solutions to achieve stability with revision of modular components if the baseplate and humeral stem are well positioned. Closed reduction is successful in only 30% to 60% of cases and can be attempted particularly if there was a trauma resulting in instability, but there is a high rate of recurrent instability and revision is often required.

Initial evaluation of a dislocated reverse TSA involves studying the radiographs to determine if there is gross mechanical failure. If so, there has been loss of containment, and it is usually obvious that one or both sides of the prosthesis need to be removed and revised. The surgeon should be prepared to deal with massive bone loss caused by catastrophic failure. Having appropriate bone graft such as a femoral head or cancellous chips is helpful for larger defects, and a custom implant might be necessary to perform the revision in this case. Massive humeral defects caused by trauma or infection might need to be addressed with an allograft or endoprosthesis.

If there is no obvious mechanical failure, identification of positional malalignment is the next step and can be identified on radiographs and often CT scan. If so, impingement is the likely cause, and this can be corrected in several ways.

Soft-tissue impingement can be addressed by excision of the mass or scar tissue causing the impingement. Bony impingement on the humeral side can be removed. In the case of implant malposition, it is possible that the baseplate can be retained and addressed with an eccentric or larger glenosphere placed to clear the inferior glenoid and scapular spine. If malposition is excessive, however, the baseplate or humeral components may need to be revised, and any revision surgery should include preparations for how to remove these with as little trauma as possible.

If there is underlying laxity of the glenohumeral components, this can lead to loss of compression of the entire prosthesis. Upsizing and/or lateralizing the glenosphere or increasing the glenosphere offset are simple techniques to increase tension while keeping an original well-fixed baseplate intact. On the humeral side, increasing the thickness of the polyethylene or adding a spacer to the construct can also increase tension to prevent instability. Rotation of the humeral tray in an onlay prosthesis can help lateralize the implant, providing more tension. For an inlay prosthesis that has a higher neck-shaft angle, rotation of the polyethylene component, if possible, to alter the angle can provide more lateralization of the implant on the humeral side.

Ways to Prevent Dislocation

Ideally, subscapularis restoration is a very simple addition to the surgery that can help prevent dislocation. The subscapularis is considered a protector of anterior dislocation in a medialized reverse TSA design. As reverse designs have evolved to be more lateral, horizontal forces on the deltoid create more compression to stabilize the joint and subscapularis repair may not be required. A recent meta-analysis by Matthewson et al[28] demonstrated that subscapularis repair reduces the rate of dislocation regardless of implant design.

Deltoid tension can be increased by glenoid lateralization. Lateralized baseplate glenoid components and larger glenosphere implants can be used to increase this tension and avoid impingement. Humeral distalization can also be increased by adding a metallic spacer or increasing the width or depth of the polyethylene component. One warning is to avoid excess overtensioning of the component to prevent complications of axillary nerve or brachial plexus neuropathy or stress fractures of the acromion and scapula.

Outcomes After Treatment for Reverse TSA Instability

Instability after a reverse TSA is an unfortunate complication. There are few reports in the literature to describe outcomes. Chalmers et al[31] noted that if the reverse TSA was a primary dislocation, closed reduction was successful in 60% of patients. For patients who had already undergone revision, success of a closed reduction was only 20%. Four of the five patients requiring revision reverse TSA maintained stability with just upsizing their polyethylene component; one patient required glenoid revision for malpositioning; and one patient underwent conversion to a constrained polyethylene liner. Fortunately, there are data suggesting that if the patient needs the polyethylene upsized and changed to a constrained liner, there is no negative effect on ROM in the future.[32]

A more recent series found that closed reduction and simple polyethylene exchange alone often was not enough to achieve reduction. Forty-five percent of those patients ended up needing additional open surgery to upsize and/or lateralize the glenoid component or distalize the humeral components as well.[26] In a surgeon's practice, it is common to increase the glenosphere diameter or lateralization as well as to increase polyethylene thickness to have the best chance of successfully stabilizing the reverse TSA with the initial revision.

Acromion and Scapula Fractures After Reverse TSA

Background

ASF after reverse TSA is an increasingly recognized complication that can be associated with significant patient morbidity including loss of function and pain. The reported incidence of ASF ranges from 1.5% to 12%. These fractures occur most commonly in elderly females with poor bone mineral density[33,34] and poor bone stock and with rotator cuff arthropathy and/or inflammatory arthropathies. On average, this complication occurs approximately 3 to 12 months after surgery, without trauma, and along the acromion or the spine of the scapula. Both surgical and

nonsurgical management have been discussed in the literature, with ASF after reverse TSA associated with a decline in initial postoperative outcomes.

Presentation

Evaluation for acromial or scapular injury after reverse TSA begins with a detailed physical examination and history of present illness. Functional status including active and passive ROM must be assessed. The patient should be queried about the exact location of pain. Gentle pressure should be applied across the acromion and the spine of the scapula to determine if pain is elicited. Duration and change in pain intensity should be documented. At least three radiographic views, including Grashey, axillary, and scapular Y, should be obtained. Radiographs of the scapula may be obtained if patients present with pain along the spine of the scapula.

Patients may present with acromial stress reactions or formal stress fractures. This most commonly occurs without antecedent trauma. In addition, patients may present with or without functional decline. Stress reactions are associated with pain during palpation of the acromion or scapula without any imaging abnormalities. These patients frequently have reasonably maintained function but with significant pain during shoulder movement. In contrast, patients with stress fractures have significant pain on palpation of the acromion or scapula in association with marked functional decline and positive imaging findings. Radiographs are frequently insufficient to diagnose these fractures, and CT is recommended to properly assess for fractures.[35]

Classification

Currently, there are two described classification schemes for ASF after reverse TSA. The Crosby classification[36] categorizes the fracture into three subgroups and is based on the relationship of the fracture to the acromioclavicular joint, with types I, II, and III corresponding to fractures at the tip of the acromion, posterior to the acromioclavicular joint, and at the posterior acromion of scapula spine, respectively.

More commonly used and with higher interobserver agreement, the Levy classification[37] categorizes ASF into three subgroups based on the deltoid origins. In this classification, type I fractures involve the anterior and middle deltoid origin, type II fractures involve the middle deltoid origin, and type III fractures involve the middle and posterior deltoid origins (**Figure 5**).

Etiology

Although there is much debate as to the cause of ASF, it is widely thought to be caused by excessive deltoid tension that is beyond the remodeling threshold, leading to structural failure. The specific effects of global lateralization and/or lengthening in relation to ASFs have been met, with conflicting reports likely a result of the multifactorial number of factors involved.[38] Other potential etiologies include a thin acromion, impingement, and superior screw perforation leading to stress risers about the scapular spine. There are also implant-related and technique-related factors that can predispose to ASF by creating an unfavorable biomechanical construct predisposing the patient to risk of ASF.

Implant-Related Risk Factors

Implant Design

RSA implants may be subgrouped into three categories: Grammont design with a medial glenoid and humerus (MG/MH); lateral glenoid and medial humerus (LG/ML); and medial glenoid and lateralized humerus (MG/LH). All three designs attempt to provide a fulcrum, stabilizing the COR while increasing the mechanical advantage of the deltoid. Because of the significant differences in implant design, the biomechanics of the deltoid are altered differently based on subgroup. Theoretically, regarding the deltoid, the ideal implant will minimize deltoid resting tension, maximize the moment arm and efficiency, and allow for minimal elevation and abduction force. Of note, differences exist within each subgroup, including neck-shaft angle, inlay versus onlay design, and screw diameter. These differences likely play a role in the occurrence of ASFs.

Various studies have attempted to determine whether there are differences based on the type of implants. In addition, results may vary among implants in the same subgroup. For example, Routman et al[33] noted an ASF incidence of 1.8% among 4,125 patients who underwent RSA with an onlay design with a medial and lateral glenosphere and humerus, respectively. This is in contrast to another study by Haidamous et al[39] that reported a 12% ASF rate with another onlay implant. In addition to different study designs, these varied results indicate that implant-based risk factors may be more nuanced than the general subgrouping. Few studies have formally compared the incidence of ASF among different implant subgroups. King et al,[34] in a systematic review involving 9,083 patients from 90 studies, evaluated the incidence of ASF after RSA and noted that the LG/MH implants had the highest incidence of ASF (3.8%), whereas the MG/LH design had the lowest rate of ASF (1.5%).

Despite these results, other studies have presented contrasting results. Haidamous et al,[39] in a retrospective multicenter study, evaluated the incidence of ASF with an onlay/MG/LH (Ascend Flex, Stryker) and inlay/LG/MH (Altivate, DJO and Univers Revers, Arthrex) design. There were 342 and 84 inlay and onlay implants, respectively. The study authors noted that significantly more patients in the onlay group sustained ASF (4.7% versus 12%). They noted significantly higher lengthening (acromiohumeral distance) and COR offset in the onlay system. As noted previously, these results contrast with another study showing ASF rates of 1.7% and 1.5%, using another onlay (MG/LH) implant (Equinoxe, Exactech).[33]

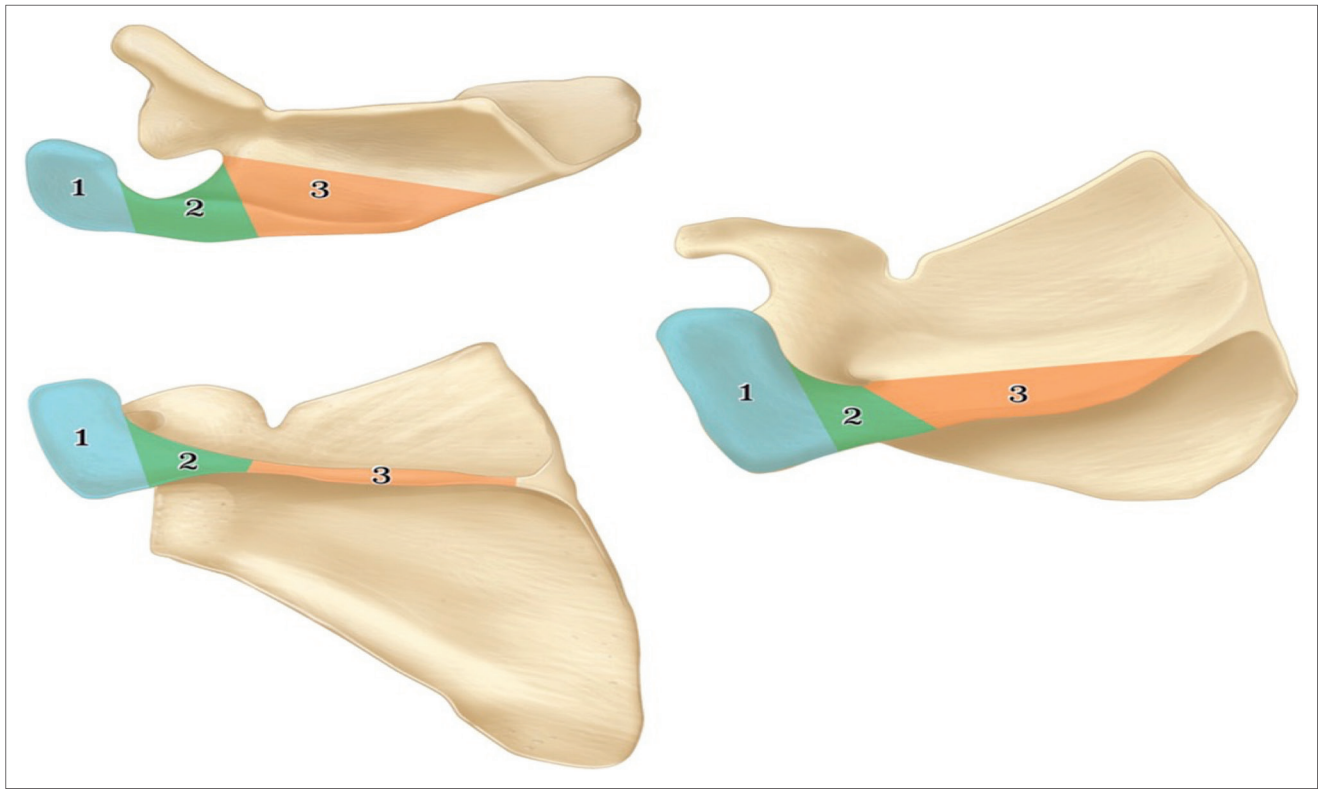

FIGURE 5 Schematic showing the Levy classification based on deltoid head origins. Type 1 represents involvement of a portion of the anterior and middle deltoid origin; type 2, involvement of at least the entire middle deltoid origin; and type 3, involvement of the entire middle and posterior deltoid origin. (Reprinted with permission from Levy JC, Anderson C, Samson A: Classification of postoperative acromial fractures following reverse shoulder arthroplasty. *J Bone Joint Surg Am* 2013;95:e104.)

Baseplate Augmentation

The use of augmented baseplates to account for glenoid deficiencies has increased. In addition to correction of deformity, many of these augmented baseplates provide varying amounts of lateralization in comparison with the nonaugmented baseplates. As a result, these implants may increase the risk of ASF. This was noted to be the case when Kirsch et al[40] reviewed their outcomes with augmented baseplates (Ascend Flex, Stryker) in a series of 44 patients with a minimum follow-up of 1 year. They noted an ASF rate of 11.4% in this patient group. In another study, a subgroup analysis of augmented baseplate cases was performed (Equinoxe) and the study authors did not note a difference in rate of ASF in this group compared with the nonaugmented group.[33]

Baseplate Bicortical Screws

Placement of bicortical baseplate screws has been associated with ASF. Specifically, these factors have been noted to occur along the scapular spine at the tip of the screw. Kennon et al,[41] in a single-surgeon retrospective comparison study involving 318 undergoing reverse TSA, evaluated the risk of ASF based on the presence of a bicortical superior screw. In 206 patients with a superior bicortical screw, they noted a fracture rate of 4.4%. This is in comparison with the other 112 patients without superior bicortical screws in which none of the patients sustained an ASF. Similarly, Otto et al[35] noted that most of their 14 of 16 type 3 ASFs occurred at the tip of a baseplate screw. In a large multicenter study involving 4,125 patients who underwent reverse TSA, Routman et al[33] noted an association between number of baseplate screws and ASF. Most of these studies describe associations as opposed to causality of screws and ASF. Additional controlled studies with appropriate numbers of patients with corresponding screw data are needed to further determine the role of screws and ASF. However, surgeons should consider placement of unicortical screws, when possible, superiorly and posteriorly to avoid this complication.

Treatment and Outcomes

Initial treatment of these fractures should always begin with a nonsurgical trial of at least 8 weeks of sling use and cessation of shoulder activity. Physiotherapy should be paused if indicated. Patients should be followed with sequential imaging studies and physical examinations to

evaluate for healing and clinical improvement. In general, patients with ASF showed worse outcomes than those without ASF. Factors that may determine patient recovery include fracture location and healing status. Levy et al[37] looked at the outcomes of nonsurgical treatment in 18 patients with ASF after reverse TSA. On average, they noted worse ROM and American Shoulder and Elbow Surgeons scores (44) than in typical patients who underwent reverse TSA. They did note that type IIe fractures had the best subjective satisfactory ratings. A more recent study by Boltuch et al[42] looked at 44 patients with ASF who were treated nonsurgically and compared with a matched control group. Fractures were grouped into medial and lateral (lateral to level of the glenoid) subgroups corresponding to location of injury. They found the lateral-based ASF did best, with outcomes similar to those of the control group. A nonunion rate of 61% most frequently occurred among the lateral-based fractures. Although union status was not correlated with outcomes these nonunions can lead to catastrophic failure because of severe scapula notching, instability, and other complications.

In terms of surgical treatment, there is limited evidence detailing outcomes. Generally, concerns with surgical treatment include poor bone quality for fixation and perpetuation of the underlying cause with implant retention. Crosby et al[36] described the best outcomes in the literature, reporting pain-free fixation in four patients with type II fractures and an average American Shoulder and Elbow Surgeons score of 80. Although surgical treatment is associated with increased union rate, healing is not predictable of significantly improved clinical outcomes.[43] More studies are needed to determine when surgical fixation is needed. In addition, it is unknown whether better outcomes are seen if surgical repair is coupled with implant revision to lower deltoid tension. This may be especially useful if the main goal is to provide pain relief (**Figure 6**).

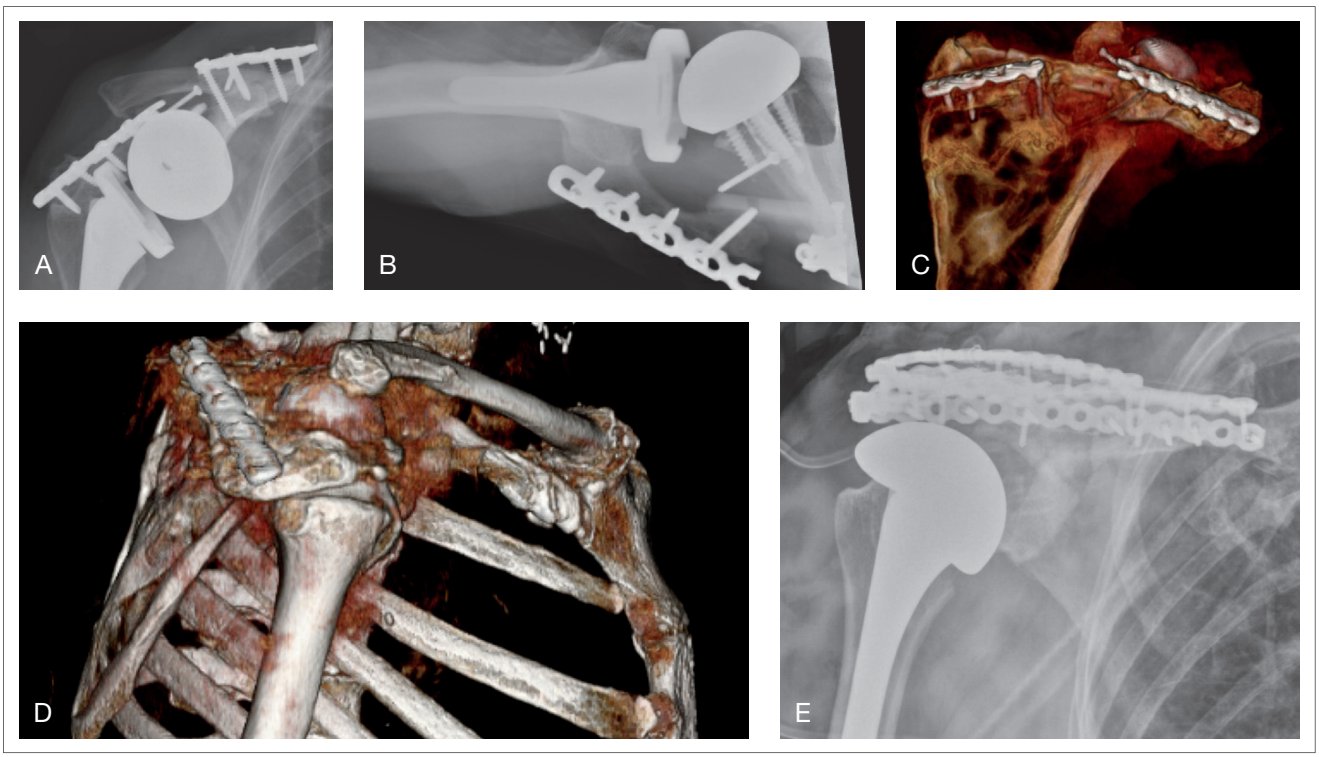

FIGURE 6 **A** and **B**, AP and axillary radiographs, respectively, from a 79-year-old woman who presented with a Levy type III acromion and scapular fracture after reverse shoulder arthroplasty (RSA) that was fixed with a reconstruction plate and fibula strut combination. The fixation failed, with plate breakage and nonunion. Three-dimensional CT reconstruction confirmed significant fracture displacement (**C**) and acromioclavicular (AC) joint diastasis (**D**). Because of severe recalcitrant pain, the patient underwent removal of hardware and revision of the fixation with dual plate fixation and conversion of RSA to a hemiarthroplasty with a cuff tear arthropathy head to decrease deltoid tension and allow for fracture healing. The AC joint was fused to increase the stability of fixation. Ten months after surgery, the patient reports no shoulder pain but has decreased function postfracture (**E**).

Summary

Complications do occur after anatomic and reverse TSA, and although they are infrequent, they can be daunting and difficult to diagnose and treat. Given the anticipated rise in shoulder arthroplasty over the next decade, all surgeons performing shoulder arthroplasty should be familiar with the most common complications, etiologies, and potential treatments. The complications seen most frequently after anatomic TSA are infection, stiffness, rotator cuff failure (superior cuff and subscapularis), and neurologic injury. The most devastating complications after reverse TSA include instability and acromion stress fractures, as well as infection and neurologic injury. A thorough understanding of these unfortunate complications will allow the treating surgeon to hopefully take measures to avoid their occurrence. However, because not all complications are avoidable, it is important for the treating surgeon to expediently recognize and address them when they occur.

KEY TAKEAWAY POINTS

- The painful anatomic TSA is uncommon and indolent infection should be ruled out in every case.
- Subscapularis failure after anatomic TSA is a debilitating complication that is often preventable; if it occurs, the most reliable treatment is conversion to reverse TSA.
- Instability after reverse TSA is not infrequent and is challenging to manage; all patient factors and implant factors should be considered before the conclusion of the index procedure.
- Acromion stress fractures are often a devastating complication and although outcomes do depend on the location of the fracture, in general, there is a functional decline when these occur.

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