The evaluation of the failed shoulder arthroplasty

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As the incidence of shoulder arthroplasty continues to rise, the orthopedic shoulder surgeon will be increasingly faced with the difficult problem of evaluating a failed shoulder arthroplasty. The patient is usually dissatisfied with the outcome of the previous arthroplasty as a result of pain, but may complain of poor function due to limited range of motion or instability. A thorough and systematic approach is necessary so that the most appropriate treatment pathway can be initiated. A comprehensive history and physical examination are the first steps in the evaluation. Diagnostic studies are numerous and include laboratory values, plain radiography, computed tomography, ultrasound imaging, joint aspiration, nuclear scans, and electromyography. Common causes of early pain after shoulder arthroplasty include technical issues related to the surgery, such as malposition or improper sizing of the prosthesis, periprosthetic infection, neurologic injury, and complex regional pain syndrome. Pain presenting after a symptom-free interval may be related to chronic periprosthetic infection, component wear and loosening, glenoid erosion, rotator cuff degeneration, and fracture. Poor range of motion may result from inadequate postoperative rehabilitation, implant-related factors, and heterotopic ossification. Instability is generally caused by rotator cuff deficiency and implant-related factors. Unfortunately, determining the cause of a failed shoulder arthroplasty can be difficult, and in many situations, the source of pain and disability is multifactorial.

Level of evidence: Review article.

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As shoulder arthroplasty indications expand and the incidence rises, the shoulder specialist will be increasingly faced with the difficult task of managing arthroplasty failures. A recent study reported a 22.6% overall complication rate for anatomic total shoulder arthroplasty, with 11.2% of cases requiring revision. The time from the index or revision arthroplasty to clinical presentation is highly variable, with 1 study reporting an average of 48.7 months (range, 0-336 months), and 29% presenting within the first year.

On presentation, patients often complain of a history of constant or activity-related pain, poor range of motion, or instability. Many times, patients state that the shoulder arthroplasty never achieved a satisfactory outcome. Alternatively, the patient may report a symptom-free interval (SFI) ranging from weeks to months after the arthroplasty during which pain was relieved and function was restored, only to be followed by a period of worsening shoulder pain and difficulty performing activities of daily living. Frequently, the cause of pain or disability is never firmly established, and in these situations, there may actually be a multifactorial etiology. A thorough and systematic approach and knowledge of...
the common causes of arthroplasty failure are necessary in the workup of these patients so that the most appropriate treatment algorithm can be instituted.

**Patient evaluation**

**History and physical examination**

The initial patient encounter includes a detailed patient history and physical examination. Obtaining an in-depth knowledge of the patient’s general health is critical. With respect to the shoulder, important aspects of the patient’s history include the nature, severity, precipitating factors, and duration of the symptoms. Any history of fever, chills, night sweats, or other constitutional symptoms should be noted. The patient is questioned regarding any history of superficial or deep wound infection or antibiotic therapy. Whether a history of narcotic dependence exists must be established.

The patient is then questioned regarding the presence or absence of a SFI after the index or most recent revision procedure. Another important consideration is whether the pain is persistent or activity-related. The details of all prior surgical procedures, including the indications, should be obtained. Attempts should be made to acquire prior operative reports and imaging. Identification of the implant and its manufacturer is critical if the possibility of revision surgery exists.

On examination, the skin and surgical incision are carefully inspected. The deltoid and rotator cuff muscles are assessed for evidence of contractility or atrophy. The shoulder is palpated for evidence of warmth or joint effusion. A point of maximal tenderness should be identified, and in the setting of reverse total shoulder arthroplasty (RTSA), particular attention should be paid to tenderness or swelling over the acromion and scapular spine. Active and passive range of motion are measured in forward elevation, external rotation in 0° and 90° of abduction, and internal rotation. If a lack of range of motion exists, whether it is secondary to soft tissue tightness, a mechanical block, weakness, or poor effort, must be determined. If the patient has reasonable active range of motion, the deltoid and rotator cuff are evaluated for evidence of weakness; however, an overall weakness relative to the contralateral shoulder may be a normal finding with TSA. A complete neurologic examination of the upper extremity, including cervical nerve root motor function and reflexes, is necessary to rule out cervical spine pathology as a contributing cause of the shoulder pain. In the setting of RTSA, the overall length of the extremity should be compared with the contralateral side.

**Diagnostic evaluation**

**Imaging**

**Plain radiographs**

Plain radiographs are the initial imaging modality. The necessary views include an anteroposterior (AP) and lateral of humerus, a true AP of the shoulder (Grashey view), a scapular Y, and an axillary lateral. The most recent plain radiographs are compared with the earlier postoperative films. First, the overall alignment of the joint and prosthesis is assessed. The prosthesis is scrutinized for evidence of device failure (eg, component dissociation, screw breakage, etc.). The humeral component is inspected for subsidence, change in alignment (eg, falling into varus), osteolysis, and radiolucent lines (RLLs). A humeral stem is considered loose if there are circumferential RLLs or if a shift in stem position has occurred on successive radiographs. A stem is considered to be “at risk” of loosening if a RLL of 2 mm or more is present in 3 or more Gruen zones. The Gruen zones, as adapted to the humeral stem, are shown in Figure 1. The diaphyseal humerus is also examined for evidence of a periprosthetic fracture. The tuberosities are assessed for evidence of fracture, bony resorption, or nonunion.

Next, the glenoid component is evaluated for evidence of migration or the presence of RLLs. Radiolucencies around a keeled polyethylene (PE) component are assessed on the Grashey view and are graded according to the method of Franklin et al (Table I and Fig. 2). Lucencies of pegged PE glenoid components are graded according to the system of Lazarus et al (Table II and Fig. 3). Although the classifications Franklin et al and Lazarus et al are often referenced, the interobserver and intraobserver reliability of these classifications has been shown to be poor. Implant seating is assessed on the Grashey and axillary radiographs. In RTSA, scapular notching and lucencies around the baseplate are assessed on the Grashey view and graded using the Sirveaux et al classification (Table III).

**Computed tomography scan**

The computed tomography (CT) scan is an important imaging modality for evaluating a failed shoulder arthroplasty. CT is useful for evaluating bony abnormalities not seen on conventional radiographs. The glenoid should be carefully assessed for loosening and component migration, abnormal positioning of the component, and perforation of the glenoid vault. The remaining bone stock must be carefully evaluated if planning future reconstructive surgery. An arthrogram can be added to the study when a rotator tear or component loosening is suspected. When the RTSA patient’s history or examination is suggestive of scapular spine or acromial insufficiency fracture, a CT of the entire scapula is obtained.

**Magnetic resonance imaging**

Magnetic resonance imaging (MRI) is not widely used in the setting of shoulder arthroplasty given the amount of metallic artifact produced. However, with certain pulse-sequence parameter modifications, in some centers, MRI is potentially useful in evaluating soft tissue integrity in the setting of shoulder arthroplasty. In a series of 22 patients who subsequently underwent revision surgery, MRI with these pulse-sequence parameter modifications correctly predicted the absence of rotator cuff tears in 8 of 10
shoulders, the presence of rotator cuff tears in 10 of 11 shoulders, and the presence of glenoid cartilage wear in 8 of 9 shoulders.\textsuperscript{98} However, given the cost and the variable results with standard sequences, MRI has a limited role in evaluating a failed shoulder arthroplasty.

**Ultrasound**

Ultrasound (US) is not affected by metallic artifact and is very useful for evaluation of rotator cuff tendon integrity, muscle atrophy, biceps tendon abnormalities, and subacromial or subdeltoid bursitis after shoulder arthroplasty.\textsuperscript{91} On US, the prosthesis appears hyperechoic, bone surfaces demonstrate shadowing, and tendon tears are evidenced by a well-defined defect filled with anechoic fluid.\textsuperscript{47} US has limited utility in the examination of a stiff shoulder due to the dynamic nature of the study. US is not useful for evaluation of osseous structures and should be used in conjunction with other imaging modalities when bony abnormalities are suspected.

**Nuclear scans**

Nuclear scans are not limited by the presence of orthopedic implants and are useful for identifying inflammation in

\begin{table}[h]
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\begin{tabular}{|c|p{0.8\textwidth}|}
\hline
Grade & Description \\
\hline
0 & No radiolucency \\
1 & Radiolucency at the superior and/or inferior flange \\
2 & Incomplete radiolucency at keel \\
3 & Complete radiolucency ($\leq 2$ mm wide) around the keel \\
4 & Complete radiolucency ($> 2$ mm wide) around the keel \\
5 & Gross loosening \\
\hline
\end{tabular}
\caption{Grading of radiolucency around a keeled glenoid according to the system of Franklin et al.\textsuperscript{33}}
\end{table}

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{image.png}
\caption{Gruen zones of the humeral stem.}
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\begin{figure}[h]
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\includegraphics[width=\textwidth]{image.png}
\caption{The grading system of Franklin et al.\textsuperscript{33} is used to assess radiolucencies around a keeled glenoid. Redrawn with permission from The Journal of Bone and Joint Surgery America.}
\end{figure}
periprosthetic tissue. Studies of the efficacy of nuclear scans in detecting prosthetic joint infection (PJI) are limited to total hip and total knee arthroplasty. A technetium bone scan can identify occult fractures and is sensitive for detecting subclinical loosening or infection; however, it lacks specificity for infection, and the result may be abnormal for up to 1 year after surgery due to remodeling of the periprosthetic bone. Studies have shown indium-111-labeled autologous leukocyte scans has poor sensitivity in diagnosing PJI, and this is thought to result from a lack of neutrophil presence in a chronic infection. The specificity of combined leukocyte and bone scans may be improved over a leukocyte or bone scan alone. Many investigators have found that combined leukocyte and bone marrow scans have very high sensitivity and specificity and may be the nuclear imaging modality of choice in diagnosing PJI.

### Laboratory values

The surgeon must have a high clinical suspicion of chronic PJI in the setting of shoulder arthroplasty failure. Inflammatory markers commonly drawn include white blood cell count (WBC), C-reactive protein, and erythrocyte sedimentation rate. Unfortunately, the clinical sensitivity of inflammatory markers in ruling out chronic shoulder PJI may be limited. In a series of 75 shoulders without overt signs of infection that had positive cultures at the time of revision surgery, Topolski et al reported an abnormal WBC in only 7%, an abnormal erythrocyte sedimentation rate in 14%, and an abnormal C-reactive protein in 25%. Pottinger et al, in a series of revision surgeries, similarly found no association with positive cultures and elevated preoperative laboratory markers. Interleukin-6 has been shown to have high diagnostic accuracy in the setting of infected total knee and hip arthroplasty; however, its utility in diagnosing chronic shoulder PJI has not been established.

### Arthrocentesis

If the clinical suspicion of PJI exists, arthrocentesis is a logical next step in the diagnostic algorithm. If the patient is being treated for a suspected infection, all antibiotic therapy should be stopped at least 14 days before the procedure. The joint aspiration can be performed with or without fluoroscopic guidance. Fluid that is successfully aspirated is sent for gram stain, cell count, and aerobic and anaerobic culture. Although the threshold synovial WBC count predictive of periprosthetic shoulder infection has not been established, in total knee arthroplasty, it is thought to be >1700 cells/µL or >65% polymorphonuclear neutrophils and is >4,200 cells/µL or >80% polymorphonuclear neutrophils in total hip arthroplasty. Joint aspiration is likely a specific but not a sensitive means of diagnosing PJI.

### Nerve conduction studies and electromyography

Nerve conduction studies (NCS) and electromyography (EMG) should be considered if there is any suspicion that failure of the arthroplasty is related to a neuropathy.
Neuropathy may result from an acute injury or chronic nerve compression. NCS and EMG are useful for providing information about the localization and severity of the nerve injury. Acute injuries after surgery or a traumatic event may involve axonal loss but are more commonly neurapraxies. The general recommendation in this situation is to wait approximately 10 to 21 days before obtaining the first study, because before this, NCS and EMG are unable to distinguish between lesions with axonal loss vs those with demyelination. If the lesion does not have a significant axonal loss component, a follow-up study is performed at 3 to 6 months to document reinnervation. NCS and EMG are also useful for characterizing chronic nerve injuries, particularly of the axillary nerve, and should be obtained if revision surgery is planned.

**Common complaints and the common causes**

**Pain**

Shoulder pain is the most frequent presenting complaint of a failed shoulder arthroplasty.34 The presence or absence of a SFI after surgery is an important clue in determining the cause of a painful shoulder arthroplasty. Causes of shoulder pain after arthroplasty without a SFI include technical errors during the operation, early periprosthetic infection, neurologic injury, and complex regional pain syndrome (CRPS). Causes of shoulder pain after arthroplasty with a SFI include delayed or chronic periprosthetic infection, component wear and loosening, glenoid erosion, rotator cuff degeneration, and fracture.

**Technical errors**

Surgeon technical errors are a very common cause of pain after shoulder arthroplasty presenting without a SFI. Examples of these are numerous, including performing a humeral head replacement (HHR; hemiarthroplasty or humeral resurfacing) in the setting of advanced glenoid arthrosis, iatrogenic periprosthetic fracture, a malpositioned or a poorly sized implant leading to overtensioning of the soft tissue or mechanical impingement, and implanting a prosthesis that never achieves adequate fixation.

**Prosthetic joint infection**

PJI is an important consideration in a painful shoulder arthroplasty, with an overall incidence reported to be between 0.4% and 2.9% in anatomic shoulder arthroplasty24,32,39,89,97,100 and between 1% and 10% in RTSA.18,27,44,112,116,122 PJI may present with a constant baseline pain with or without a SFI. The pain of a PJI may also be associated with shoulder stiffness or prosthetic instability. Constitutional systems and examination findings suggestive of an infection may not necessarily be present. PJIs are classified temporally as early (<3 months), delayed (3 to 24 months) or late (>24 months)121 and can be further subclassified as acute (<6 weeks) or chronic (>6 weeks) by the duration of symptoms before presentation. Acute PJIs are often caused by virulent organisms, such as coagulase-positive *Staphylococcus*, anaerobes, or gram-negative bacilli, and are thought to occur by direct wound contamination or by hematogenous seeding of the joint with enteric organisms.

Acute PJIs commonly present with all the classic hallmarks of septic arthritis, including loss of range of motion, erythema, warmth, swelling, wound drainage, elevated laboratory markers, and positive joint and blood cultures. Chronic shoulder PJI is increasingly recognized as a cause of undiagnosed pain. Pain may be the only clinical finding in a chronic, indolent shoulder PJI. Associated radiographic findings include component malposition or loosening, periprosthetic fracture, or instability.9

Causative organisms are often low virulence and include *Propionibacterium acnes* and coagulase-negative *Staphylococcus*. *P acnes* is an anaerobic, gram-positive rod found in the sebaceous follicles of the axilla,77 and until recently, was generally considered a culture contaminant rather than a true orthopedic pathogen. Frequently, a *P acnes* infection presents without overt signs of infection. In a series of 193 revision shoulder arthroplasty procedures performed for stiffness, pain, or loosening, Pottinger et al17 reported that 56% had intraoperative positive cultures, of which 70% were positive for *P acnes*. In that study, male sex, humeral osteolysis, cloudy synovial fluid, humeral loosening, glenoid wear, and membrane formation were positively associated with obtaining a positive, intraoperative *P acnes* culture.79 Intraoperative histology is not sensitive for diagnosing *P acnes* PJI. In a series of 27 patients with confirmed *P acnes* shoulder PJIs, only 40% had evidence of acute inflammation (>5 neutrophils per high-power field) on intraoperative histology.17 *P acnes* is a slow-growing bacterium, and it is recommended that both anaerobic and aerobic culture media be incubated for at least 13 days to optimize recovery of the organism. The average time to positive *P acnes* culture is reported to be 9 to 13.3 days.29,61

**Complex regional pain syndrome**

Another consideration of a persistently painful shoulder arthroplasty is CRPS, formerly known as reflex sympathetic dystrophy. The overall incidence of CRPS is estimated to be 1% after anatomic shoulder arthroplasty.39 The diagnosis of CRPS requires that regional pain after a noxious event be present. Clinical presentation may mimic infection and usually includes swelling, warmth, and trophic changes of the overlying skin. The etiology of CRPS is poorly understood but likely involves neurogenic inflammation,173 central nervous system dysfunction,3,63,78,80 and a genetic predisposition.64

**Neurologic injury**

Another cause of persistent pain after shoulder arthroplasty is an unrecognized neurologic injury. The incidence of neurologic injury after anatomic shoulder arthroplasty is
reported to be 0.6% to 4.3%, and RTSA is reported to carry a risk of 2% to 12%. In both cases, the actual incidence is likely much higher than the reported incidence. In a series of 30 patients who underwent neuromonitoring during shoulder replacement surgery, 56% had nerve alerts during the course of the surgical procedure. Of the intraoperative nerve alerts, 46% involved multiple nerves at the level of the trunk or cord of the brachial plexus, with the upper trunk being most frequently involved (42.9%), followed by the posterior cord (21.4%). Isolated nerve alerts most frequently involved the musculocutaneous nerve (20%) and the axillary nerve (16.7%). The authors of this study identified the positioning of the humerus as the part of the procedure that places the brachial plexus at highest risk.

Another potential cause of neurologic injury in shoulder arthroplasty is iatrogenic brachial plexus injury during an interscalene block. Most interscalene block-related neurologic injuries are temporary, with permanent plexus nerve injury reported to be <1% using modern ultrasound-guided techniques. Other reported causes of neurologic injury during shoulder arthroplasty include cement extrusion causing injury to the radial nerve and excessive humeral lengthening in RTSA causing brachial plexus stretch and prominent peripheral baseplate screws in RTSA causing injury to the suprascapular nerve.

Component loosening
Glenoid loosening is a frequent cause of activity-related pain and declining patient satisfaction and usually presents after a SFI. The overall incidence is reported to be 14% in anatomic shoulder arthroplasty. Radiographic loosening of the glenoid likely precedes symptomatic loosening by several years. A recent multicenter study reported the survivorship of a cemented, keeled glenoid component was 98.3% at 10 years with the end point of revision surgery for glenoid loosening and 51.5% at 10 years with the end point of radiographic loosening of the glenoid. Survivorship of the glenoid component depends on several factors, including implant design, surgical technique, and integrity of the rotator cuff. With respect to implant design, cemented all-PE components have been shown to have a better clinical result than metal-backed implants, with multiple reports of implant failure in metal-backed implants. This may be related to the favorable mechanical properties of an all-PE implant that allows the humeral head to translate without imparting excessive stress at the bone–implant interface.

The conformity of the glenoid and humeral implant likely plays a role in implant survival; however, the optimal conformity is debatable. A conformed glenoid with little or no radial mismatch theoretically distributes the load to the bony glenoid evenly, but this comes at the expense of translation that can only occur with aricular separation and eccentric loading. Walch et al. in a study of 319 TSAs, found the least glenoid radiolucency and highest Constant scores are achieved with a radial mismatch of 6 to 7 mm. However, in a biomechanical study using a cementless, metal-backed implant, a radial mismatch of >6 mm increased interface micromotion when compared with a fully conforming implant.

Proper surgical technique is also integral to glenoid survivorship. Farron et al. in a 3-dimensional finite-element model, found that a glenoid placed in retroversion increases the stress within the cement mantle by 326% and micromotion at the cement–bone interface by 706% compared with a neutrally implanted glenoid. A recent clinical series showed that glenoid implant retroversion >15° was associated with increased odds of osteolysis of the glenoid. Walch et al. in a clinical series with >5 years of follow-up, showed that eventual glenoid component migration was associated with implantation in a low position, superior tilt on the immediate postoperative films, and the use of excessive reaming at the time of surgery. Fully seating the implant on a congruent and adequately prepared bone surface is critical because it minimizes implant wobble (movement of the implant) and warp (deformation of the implant) when loaded eccentrically. Any movement of the implant relative to the bone not only produces mechanical loosening but also likely generates PE wear debris.

Glenoid survival is also related to cementation technique. Cement pressurization leads to better cement penetration and fewer RLLs compared with glenoids implanted without pressurization.

Lastly, an intact rotator cuff plays an essential role in prevention of glenoid loosening. Long-term cuff dysfunction with humeral head superior migration may lead to eccentric loading of the glenoid and initiation of the “rocking horse phenomenon” that may ultimately produce glenoid loosening.

Glenoid-sided loosening is also a reported complication after RTSA and may be related to PE wear debris generated by scapular notching. Scapular notching occurs from mechanical impingement of the reverse humeral prostheses against the inferior scapular border as a result of the medialized center of rotation and nonanatomic neck-shaft angle found in many commercially available Grammont-style implants. Glenosphere placement, high on the glenoid, is also known to contribute to scapular notching. For example, scapular notching developed in an 82-year-old woman who was treated with RTSA for a proximal humeral fracture, in which the glenosphere was placed superiorly (Fig. 4). Scapular notching generates PE wear debris and may lead to baseplate loosening. The end-stage of baseplate loosening is implant migration, and the reported incidence was 3.4% in a series with follow-up of at least 8 years.

Humeral loosening is another potential cause of pain with a SFI after shoulder arthroplasty. Humeral loosening is less common than glenoid loosening, with a reported incidence of 0% to 7% in TSA and 3.6% to 8.8% in RTSA. Cil et al. found that failure or loosening of the humeral component in TSA most frequently
occurs in the setting of glenoid component issues. In a series of 38 revision TSAs for aseptic humeral loosening, the glenoid component was revised in all but 2 shoulders. A radiograph of a loose humeral stem with circumferential RLLs is shown in Figure 5.

The risk of humeral loosening may be higher for RTSA than it is for TSA and is probably related to the semi-constrained nature of the RTSA articulation imparting a greater shear stress at the stem–bone interface.

Glenoid arthrosis
Glenoid arthrosis is a well-recognized sequela of HHR, resulting in activity-related shoulder pain. In fact, glenoid erosion is an expected outcome when HHR is performed in young, active patients, with rates approaching 100%. The estimated survival of HHR in patients aged <50 years old is 91% at 5 years, 82% at 10 years, and 75% at 20 years. An AP and axillary radiograph of a 55-year-old man who developed symptomatic glenoid arthrosis 10 years after HHR is shown in Figure 6.

Rotator cuff degeneration
Rotator cuff degeneration may initially present as a painful TSA after a SFI. Partial-thickness tears and small full-thickness tears may be well tolerated for a period of time; however, if tear progression ensues, the shoulder may become increasingly unstable, resulting in loss of function and implant loosening.

Fracture
A periprosthetic fracture is another potential cause of a painful shoulder arthroplasty after a SFI. A 1% incidence of postoperative periprosthetic fracture was reported in a series of 4019 shoulder arthroplasties, and medical co-morbidity, as assessed with the Deyo-Charlson index, was a significant risk factor. If a patient has a known history of a periprosthetic fracture and persistent pain, fracture nonunion or infected nonunion is a possible etiology (Fig. 7).

In RTSA, scapular insufficiency fractures are a commonly reported complication. In a series of 400 RTSAs, Crosby et al reported the overall incidence of scapular fracture was 5.5%. A common clinical scenario involves an osteoporotic woman who presents several weeks to months after RTSA with an acute onset of posterosuperior shoulder pain and limited elevation. Reported fracture patterns include acromial avulsion fractures, fractures of the acromion posterior to the acromioclavicular joint, and fractures of the scapular spine.
Poor range of motion

Poor range of motion is a second common patient complaint when presenting for an unsatisfactory shoulder arthroplasty and may result from stiffness, loss of muscle function, or a mechanical block. The underlying causes generally include inadequate postoperative rehabilitation, implant-related factors, and heterotopic ossification (HO).

Inadequate rehabilitation

Inadequate rehabilitation is often cited as a cause of stiffness in shoulder arthroplasty; however, there is actually little evidence correlating negative outcomes with a lack of involvement in formal, postoperative physical therapy. Most current rehabilitation programs of anatomic shoulder arthroplasty consist of early range of motion and are based on Neer’s original protocol of 3 stages of progression under the supervision of a therapist. Studies by Boardman et al. and Mulieri et al. have challenged the traditional therapist-based approach. Mulieri et al. found that a physician-guided home-based therapy program was as effective as formal physical therapy with respect to final range of motion and other clinical outcomes. Others have shown early range of motion exercises may not be necessary to regain motion. In a series of 40 hemiarthroplasties for 3- or 4-part proximal humeral fractures treated with sling immobilization for 4 weeks, no excessive stiffness was seen as long as the tuberosities united, and the average forward elevation and average external rotation were 130° and 40° in this group. Despite the controversy, lack of involvement in postoperative rehabilitation remains a potential risk factor for postoperative stiffness.

Implant-related factors

In anatomic shoulder arthroplasty, proper surgical technique and component sizing are critical to avoid poor

Figure 6  (Left) Anteroposterior and (Right) axillary radiographs of a 55-year-old man who developed symptomatic glenoid arthrosis 10 years after humeral head replacement.

Figure 7  Anteroposterior radiograph of a 72-year-old woman with persistent pain following treatment of a periprosthetic humeral fracture (A). Closer inspection revealed a periprosthetic fracture nonunion (B).
postoperative range of motion. The shoulder capsule is normally lax throughout the functional range of motion, and if adequate capsular releases are not performed or if the replacement articular surfaces are too large, range of motion will be lost owing to the undue tension on the soft tissues. Harryman et al. found that nearly all directions of motion are decreased when the joint is “overstuffed” with a humeral head prosthesis that is too large. An overstuffed joint can also limit active range of motion by reducing rotator cuff muscle excursion and potentially lead to rotator tendon degeneration. Placing a humeral component too high or into varus effectively overstuffs the joint and can also lead to poor range of motion.

Several surgical factors in RTSA may lead to poor postoperative range of motion. Overtensioning of the soft tissues, particularly the deltoid, may result in diminished elevation. Although some deltoid lengthening is necessary to increase its efficiency as a forward elevator and abductor, if the muscle is lengthened too far beyond its normal resting length, it likely begins to lose excursion as a result of being placed too high on the muscle fiber length-tension curve. Implanting too large a glenosphere or positioning the glenosphere superiorly on the glenoid may also limit elevation due to impingement of the humerus on the acromion. The use of an implant with a medialized and distalized center of rotation may result in a loss of rotation by several postulated mechanisms, including detensioning of the teres minor, impingement of the humerus on the coracoid process or the scapular spine, and a change in the anterior and posterior deltoid force vectors to a more vertical direction resulting in increased recruitment for elevation and abduction at the expense of rotation. Subscapularis failure may result in a loss of internal rotation. Neurologic injury, usually to the axillary nerve, will also severely limit motion.

**Heterotopic ossification**

HO is a less common cause of stiffness after shoulder arthroplasty. HO can present with pain, stiffness, erythema,
and effusion. Neer’s original classification of HO in 1970 was later modified to the widely used classification by Brooker et al in 1973. The overall incidence of HO in the shoulder is much less common than it is in the hip, knee, and elbow, but is reported to be 12% to 54%. Symptomatic shoulder HO with loss of range of motion is even less common, with an incidence of <10%. HO is more common in RTSA, with a reported incidence of 45% to 100%. HO is also likely more common when arthroplasty is performed in the setting of a proximal humeral fracture. A 75-year-old woman who was treated with a RTSA for 4-part proximal humeral fracture that developed abundant HO is shown in Figure 8.

Instability

Instability is a third common patient complaint in the setting of a failed shoulder arthroplasty. The rate of instability after TSA is reported to be approximately 4%, and may be anterior, superior, or posterior in direction. The incidence of instability after RTSA is probably higher and is reported to be 5% to 16%. Common causes of instability after arthroplasty include rotator cuff dysfunction and problems related to surgical technique.

Rotator cuff dysfunction

Rotator cuff dysfunction is a common cause of instability after TSA. The most common pattern of instability in anatomic shoulder arthroplasty is anterior owing to failure of the subscapularis. In fact, some loss of subscapularis function is a common finding after shoulder arthroplasty. A study of 41 TSA patients monitored for 1.9 years found 67.5% had abnormal lift off tests and 66.6% had abnormal belly press examinations. The rate of full-thickness subscapularis tear after TSA may be as high as 50%.

Multiple techniques exist for taking down the subscapularis (ie, tenotomy, peel, osteotomy), but none has proven to be the gold standard given that studies have produced variable results. Posterosuperior rotator cuff tearing may present acutely after a traumatic event or chronically as the result of progressive deterioration. Tears of the supraspinatus and infraspinatus lead to a superior instability pattern and eventual superior migration of the humeral head (Fig. 9). In a clinical series of TSAs with a mean follow-up of 11 years (range, 10-15 years), the radiographic finding of superior migration of the humerus was seen in 69% of shoulders. Survivorship free of secondary cuff dysfunction in a series of 704 TSAs was 100% at 5 years, 84% at 10 years, and 45% at 15 years. Long-term sequelae of this instability pattern include glenoid loosening and, ultimately, anterosuperior escape if the coracohumeral arch is violated. Massive anterosuperior tears (supraspinatus and subscapularis) can lead to an anterosuperior instability pattern, as shown in Figure 10.

Implant-related factors

Errors in surgical technique may also cause instability. In anatomic shoulder arthroplasty, undersizing of components or implanting the humeral component too low can lead to instability, as shown in Figure 11. In TSA, glenoid implant malalignment, usually in the form of uncorrected glenoid retroversion, can result in posterior instability.

There are several risk factors for instability in RTSA, and most involve some form of soft tissue imbalance. Insufficient tension on the deltoid is probably the most frequently implicated cause and may result from placing the glenosphere too high on the glenoid, excessive medi- alization of the glenosphere, cutting the humeral neck too low, or using a PE liner that is too thin. Other less common causes of poor deltoid tension include axillary nerve injury, generalized neurologic conditions (eg, Parkinson disease), or acromial stress fracture. Instability resulting from soft tissue imbalance may also result from incomplete capsular releases or a lack of a repairable subscapularis. Instability may also be attributed to implant-related factors.
Despite proper soft tissue tensioning, these include a baseplate inclined superiorly on the glenoid,\(^\text{36}\) a lack of bony ingrowth resulting in instability at the bone–component interface (eg, stem derotation\(^\text{11}\)), and component failure (eg, PE dissociation or fracture\(^\text{83}\)). In cases in which the cause of instability is not easily detected, infection may be the underlying cause. Lastly, not infrequently, patients may present complaining of “instability” without any objective evidence on the initial workup (plain radiographs and physical examination). In a situation where advanced diagnostic studies remain inconclusive, a fluoroscopic examination under anesthesia may be a useful.

**Figure 11** Radiograph shows anteroinferior instability development in a 60-year-old woman who was treated with a humeral head replacement that was implanted inferiorly in the humerus.

**Conclusions**

The failed shoulder arthroplasty is a complex clinical entity that requires a diligent workup and a thorough knowledge of mechanisms of failure. Although it is logical to sort possible causes of failure according to patient complaints, it is important to note that multiple underlying etiologies often exist—especially in cases that are longstanding and those that are complicated by a history of multiple revisions. Unless there is a clearly identifiable cause of the unsatisfactory result, infection should always be considered.

**Disclaimer**

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**References**


