In addition to neurologic injuries such as peripheral nerve palsy, axillary vessel injury should be recognized as a possible complication of reverse total shoulder arthroplasty. Limb lengthening associated with Grammont-type reverse total shoulder arthroplasty places tension across the brachial plexus and axillary vessels and may contribute to observed injuries. The Grammont-type reverse total shoulder arthroplasty prosthesis reverses the shoulder ball and socket, shifts the shoulder center of rotation distal and medial, and lengthens the arm. This alteration of native anatomy converts shearing to compressive glenohumeral joint forces while augmenting and tensioning the deltoid lever arm. Joint stability is enhanced; shoulder elevation is enabled in the rotator cuff–deficient shoulder. Arm lengthening associated with reverse total shoulder arthroplasty places a longitudinal strain on the brachial plexus and axillary vessels. Peripheral nerve palsies and other neurologic complications of reverse total shoulder arthroplasty have been documented.

The authors describe a patient with rotator cuff tear arthropathy and a history of radioulnar synostosis who underwent reverse total shoulder arthroplasty complicated by intraoperative injury to the axillary artery and postoperative radial, ulnar, and musculocutaneous nerve palsies. Following a seemingly unremarkable placement of reverse shoulder components, brisk arterial bleeding was encountered while approximating the incised subscapularis tendon in preparation for wound closure. Further exploration revealed an avulsive-type injury of the axillary artery. After an unsuccessful attempt at primary repair, a synthetic arterial bypass graft was placed. Reperfusion of the right upper extremity was achieved and has been maintained to date. Postoperative clinical examination and electromyographic studies confirmed ongoing radial, ulnar, and musculocutaneous neuropathies.
By design, the Grammont-type reverse total shoulder arthroplasty reverses the shoulder ball and socket, medializes and distalizes the shoulder center of rotation, and lengthens the arm by an average of 2 to 3 cm.¹ ³ This nonanatomic modification of joint architecture serves multiple important mechanical functions, including tensioning and increasing the lever arm to the deltoid and converting a shear to a compressive joint stabilizing force.¹ ⁴

Arm lengthening associated with reverse total shoulder arthroplasty places soft tissues, including axillary vessels and the brachial plexus, under some degree of longitudinal strain. In a cadaveric study using 3-dimensional computer modeling, direct reverse total shoulder arthroplasty-induced strain was calculated to be as high as 19.3% for certain elements of the brachial plexus.⁵ Lädermann et al⁶ found a statistically significant increase in subclinical postoperative nerve injury as determined by electromyographic changes in patients undergoing reverse total shoulder arthroplasty over standard total shoulder arthroplasty.

Neurologic complications following reverse total shoulder arthroplasty are relatively frequent, although typically mild and transient.² ⁴ They include reflex sympathetic dystrophy⁶ and radial,⁶ ⁷ ulnar,⁸ median,⁸ musculocutaneous,⁷ and axillary nerve palsies.⁸ ¹⁰ Described vascular complications of reverse total shoulder arthroplasty include postoperative phlebitis¹¹ and wound hematoma.⁹ ¹¹ Axillary vessel injuries have not been previously reported. The current authors describe a patient who underwent reverse total shoulder arthroplasty complicated by brachial plexus injury and axillary artery avulsion. This case suggests neurovascular injuries were causally associated with reverse total shoulder arthroplasty–induced limb lengthening in a patient with predisposition for abnormal anatomy.

CASE REPORT

A 78-year-old woman presented with a 6-month worsening of chronic right shoulder pain. She reported no antecedent trauma. Pain was indicated in a chevron distribution and was exacerbated with attempted overhead activity. Both pain and function had become an impediment to perceived quality of life and performance of activities of daily living. Her medical history was notable for osteoporosis and bilateral proximal radioulnar synostosis. Surgical history included right deltoid split rotator cuff repair, right open rotator cuff repair, and right total elbow arthroplasty. She underwent split rotator cuff repair, right open rotator cuff repair, and right total elbow arthroplasty for rotator cuff tear arthropathy and scheduled for elective reverse total shoulder arthroplasty.

With the patient in the beach-chair position, a standard deltopectoral approach was used. During exposure, the remaining subscapularis tendon was found to be thin and atrophic. The subscapularis tendon was released, and tag sutures were placed. A routine sequence of bone preparation and prosthesis placement ensued. Due to proximal total elbow arthroplasty cement bolus, the offset-type Comprehensive Reverse Shoulder System (Biomet, Warsaw, Indiana) with microstem humeral component was used (Figure 2). Final components showed acceptable range of motion, good stability, and no toggle under distal traction.

During wound closure, specifically when approximating suture-pretagged medial and lateral subscapularis tendon edges, a brisk rush of arterial bleeding was encountered. The ipsilateral radial pulse was lost, and capillary fill became markedly delayed. The wound was packed and pressure held with dry gauze sponges. Anesthesia was alerted. Vascular surgery was consulted emergently. Stat laboratories and blood products were obtained.

Following arrival of the vascular surgery team, proximal and distal control was established. The source of bleeding was meticulously dissected and definitively identified as the axillary artery. The axillary artery within the brachial plexus ran just 1 cm medial to the glenoid head (glenosphere). Close scrutiny of the injured vessel segment revealed no sharp

Figure 1: Preoperative anteroposterior radiograph of the shoulder showing previous postoperative changes.

Figure 2: Postoperative anteroposterior radiograph of the shoulder showing the Comprehensive Reverse Shoulder System (Biomet, Warsaw, Indiana). Final components include a standard glenoid baseplate, 36+6 glenoid head (glenosphere), size 10 press-fit microhumeral stem, and standard 36 socket.
laceration or puncture consistent with either a needlestick-type or sharp incisional mechanism of injury. Rather, the vessel demonstrated a large, longitudinally oriented tearing with redundant stretched vessel wall edges consistent with tractional or avulsive trauma. Just 2 mm of posterior wall remained intact. No brachial plexus lesion was observed.

A primary repair with pericardial patch was initially attempted by the vascular surgeon. However, after preparing the vessel ends and beginning anastomosis, unacceptably high tension across the repair was encountered, and this method of treatment was aborted. A second tack, that of bypass grafting, was begun. Hemashield knitted graft (Maquet, San Jose, California) was selected and anastomosed proximally and distally using 5-0 Prolene suture (Ethicon, Inc, Somerville, New Jersey) in standard end-to-end fashion. A graft-spanned vessel gap of 2.5 cm eliminated longitudinal tension. Doppler flow, strong distal pulses, and adequate capillary refill were reestablished.

In the postoperative care unit, arm vascularity was lost. An emergent trip to the angiography suite revealed an occluded right axillary graft (Figure 3A). A mechanical thrombectomy with AngioJet (Medrad, Inc, Warrendale, Pennsylvania) and percutaneous balloon angioplasty was performed. Perfusion to the right upper extremity was again restored (Figure 3B).

Over the course of the day of the operation, hemoglobin fell from a preoperative baseline of 14.5 to a low point of 7.0. Four units packed red blood cells were provided after vessel injury; an additional 2 units were dispensed on postoperative day 3. The patient’s hospital course was notable for development of complex brachial plexus palsy, stable hemodynamic status, and discharge on postoperative day 4. Outpatient electromyogram 2 weeks postoperatively revealed severe radial, ulnar, and musculocutaneous mononeuropathies and no evidence of axonal continuity with any respective muscles. Suprascapular neuropathy and a chronic median mononeuropathy were also seen. At most recent follow-up 6 months postoperatively, complete motor and sensory dysfunction of radial, ulnar, and musculocutaneous nerves persisted. Partial return of suprascapular and median nerve function was suspected, and active forward flexion of the shoulder and opposition of the thumb were noted.

**DISCUSSION**

Since the introduction of reverse total shoulder arthroplasty by Grammont et al12 in 1987, indications for surgery and procedure popularity have expanded considerably. Approved by the United States Food and Drug Administration for use in patients with rotator cuff tear arthropathy in 2004, many today undergo reverse total shoulder arthroplasty for massive humerus fracture, neurologic repercussions may be attributable to posttraumatic status of the shoulder. Reported injuries may also be related to intraoperative traction, manipulation of the arm, or injudicious retractor placement. In exposing the glenoid during a standard deltopectoral approach, the humerus is placed in external rotation, abduction, and posterior-directed retraction. Such a position has been reported to significantly accentuate traction across the brachial plexus.16

Reverse total shoulder arthroplasty may pose an inherent potential risk to the brachial plexus.2 The Grammont-type prosthesis, along with reversing the shoulder ball and socket, also medializes and distalizes the shoulder center of rotation. The advantages of this configuration are well known. In medializing the center of rotation, the deltoïd muscle lever arm is nearly doubled, improving effective strength recruitment for shoulder abduction.1 Distalization tensions the deltoïd muscle and opens space for unrestricted proximal humerus range of motion.3

One unintended consequence of this nonanatomic joint architecture is that greater tension may be placed across the brachial plexus. Reverse total shoulder...
arthroplasty can be expected to lengthen the arm (as measured from tip of the acromion to the elbow) by 2 to 3 cm.\textsuperscript{1}–\textsuperscript{3} Van Hoof et al\textsuperscript{2} used 3-dimensional computer modeling to attempt to quantify the degree of strain placed on elements of the brachial plexus. Their model calculated direct prosthesis-related strains of up to 15.3\% and 19.3\% for the median nerve lateral and medial roots, respectively. The authors did not directly investigate or comment on stresses placed on major vascular structures of the upper extremity. In a recent prospective investigation, Lädermann et al\textsuperscript{2} found a 10.9-times higher risk of acute postoperative nerve injury in patients undergoing reverse total shoulder arthroplasty as opposed to standard total shoulder arthroplasty. The study also confirmed an average limb shortening of 2.7±1.8 cm associated with reverse total shoulder arthroplasty.

In the current patient, the authors estimated a limb shortening of 2.2 cm and an increase in humeral shaft offset of 0.8 cm following reverse total shoulder arthroplasty. Without pre- and postoperative full-length humerus radiographs, the authors were unable to estimate limb shortening in exactly the same manner as a previously standardized and validated protocol.\textsuperscript{3} Lädermann et al\textsuperscript{2} used the distance along the humeral diaphyseal axis from the acromion to the epicondylar axis for pre- and postoperative limb length estimates. Instead of the epicondylar axis of the elbow, the current authors used the proximal extent of the total elbow arthroplasty cement mantle, which could be seen distinctly on pre- and postoperative radiographs, as a fixed distal humerus reference point. Their calculated limb shortening is within range of what might be expected during placement of a Grammont-type reverse total shoulder arthroplasty.\textsuperscript{1}–\textsuperscript{3} The authors acknowledge inherent limitations in their methods of calculation and encourage interpretation with appropriate caution.

During this case, the authors encountered abrupt-onset, voluminous arterial hemorrhaging from the medial aspect of the surgical bed. After an uneventful procedure, observing a new and significant vascular injury should signal to the treating surgeon a shift in treatment priorities. Procedural goals typical of routine arthroplasty should be supplanted by principles of damage control surgery. The authors’ first step was firm packing with multiple dry gauze sponges to halt further blood loss. The anesthesia team reassessed the patient, called for additional staffing, hung additional fluids, collected laboratory studies, ordered blood products, and obtained additional intravenous access. An on-call vascular surgeon arrived accompanied by vascular surgery instrument trays and a dedicated vascular surgery scrub technician.

With appropriate resources in place, the next surgical task was to establish proximal and distal vessel control. This was achieved by extending the skin incision, adjusting retractors, further releasing local scar tissue, and performing further dissection directed specifically at the source of bleeding. In this manner, first the proximal and then the distal sources of bleeding were clamped with vessel clamps. Although unnecessary in the current case, additional options for gaining proximal vessel control include coarctation osteotomy, excision of the clavicle, and release of the strap muscles to better access the axillary vessels. For difficulty obtaining distal vessel control, release of the pectoralis major insertion is also an option.

Given the ubiquity of the anterior approach to the shoulder and the close proximity of the brachial plexus and axillary vessels in this exposure, a sound understanding of neurovascular anatomy is prerequisite. The brachial plexus and axillary artery arise from the posterior triangle of the neck, bound by the sternocleidomastoid muscle, trapezius, and clavicle. Traversing superficial to the first rib and deep to the clavicle and subclavian muscle, the brachial plexus and axillary vessels enter the axilla behind the pectoralis minor, medial to its coracoid insertion. At the level of the glenohumeral joint, the brachial plexus and axillary vessels run medial to the anterior glenoid rim before coursing into the brachium.

In a dedicated cadaveric investigation, McFarland et al\textsuperscript{17} sought to better define the relationship of the brachial plexus to the glenoid in the context of a deltopectoral surgical approach to the shoulder. The authors measured the distance from the anterior glenoid rim to the musculocutaneous nerve, axillary artery, posterior cord, and medial cord at 0\textdegree, 60\textdegree, and 90\textdegree shoulder abduction. They found the brachial plexus and axillary artery to lie within 2 cm of the glenoid rim and as near as 5 mm in certain instances. A medial retractor, whether placed superficial to the subscapularis muscle or stationed on the scapular neck within the subscapularis tendon split itself, was found to make direct contact with the brachial plexus in all arm positions. Changing the position of the arm led to no statistically significant difference in distance from the glenoid to the brachial plexus and axillary artery.\textsuperscript{17} This finding contrasts with other references, such as Surgical Exposures in Orthopaedics,\textsuperscript{18} which warns against abduction of the arm during an anterior approach to the shoulder joint. Hoppenfeld et al\textsuperscript{19} posit that arm abduction causes the brachial plexus to become tight and come into closer proximity of the coracoid tip. It is suggested that the arm remain adducted during any work about the coracoid process.

The vessel injury observed in the current case occurred within the third zone of the axillary artery, lateral to the pectoralis minor. During an anterior approach to the shoulder, this vessel segment is nearest to planes of dissection and most accessible to direct iatrogenic injury. The artery was not encased in an abundance of scar tissue or otherwise visibly tethered. It is unclear why the vessel injury occurred in this specific region.
At the outset of this case, a standard subscapularis tendon release was performed, leaving a cuff of insertional tissue for later repair. No further subscapularis releases were performed. Until vessel injury, retractors were placed deep to the incised subscapularis tendon, just medial to the anterior glenoid rim and lateral to the brachial plexus and axillary artery. The surgical dissection following arterial injury was taken to an extent necessary for control of bleeding and vascular repair, not for the purpose of complete exploration of the brachial plexus. During this dissection, elements of the brachial plexus, including cords and branches, were encountered but not individually identified. No evidence of nerve injury was seen. It should be noted that iatrogenic stretch during this dissection could have contributed to nerve injuries sustained during this case.

Multiple factors may have accounted for the observed injuries in this case. It has been suggested that removal of a well-fixed humeral stem, surgical dissection in altered anatomy, and the presence of scar tissue may be associated with a heightened risk of neurologic injury during total shoulder arthroplasty. The current patient’s surgical history included ipsilateral open rotator cuff repair and treatment of radioulnar synostosis. The authors believe that this surgical history was likely not a significant factor in the development of the patient’s complication. Surgeon factors such as retractor placement, arm position, implant selection, and surgical exposure also represent important potential etiologies of iatrogenic neurovascular injury. The authors believe these factors were not pertinent in this case. They do not attribute the development of the observed injuries to any specific aspect of the instrumentation.

The authors believe that this patient’s vascular and neurologic injuries were causally related to limb lengthening in intrinsic to reverse total shoulder arthroplasty. This hypothesis is supported by the gross appearance of the injured vessel consistent with avulsion, concurrent additional nerve palsies, and failure of the primary repair with the need for arterial bypass graft spanning a 2.5-cm vessel gap to relieve vessel tension. The patient showed excellent hemostasis through initial exposure, preparation of the proximal humerus and glenoid, placement and resection of the prostheses, and even final irrigation of the wound. It was not until, and precisely at the point of, approximation of the incised subscapularis tendon via pull on pretagged sutures that abrupt bleeding was encountered.

Although the authors are uncertain of the etiology of vascular and neurologic injuries in their patient, they suspect that reduction of the prosthesis in a lengthened arm placed undue tension on the axillary artery. Either through physiologic adhesion of the subcapsularis to the adjacent brachial plexus and axillary artery or because of prior scar tissue, they surmise that reapproximation of the suscapsularis applied further tension across the axillary artery, now sufficient to cause vessel rupture.

Although scattered reports and biomechanical studies have commented on neurologic complications of reverse total shoulder arthroplasty, few reports on associated vascular injuries can be found in the published literature. Considering the close proximity of axillary vessels to the anterior glenoid rim, the limb-lengthening characteristics of the Grammont-type reverse total shoulder prosthesis, and the numerous reported brachial plexus injuries, it is surprising that axillary artery injury has not previously been reported as a complication of reverse total shoulder arthroplasty.

**CONCLUSION**

This case highlights principles of damage control surgery and expands on the details of reverse total shoulder arthroplasty pertinent to the shoulder and elbow specialist. Swift application of pressure, prompt communication with anesthesi colleagues, and early involvement of vascular surgery specialists helped optimize outcome in this limb- and lifethreatening operative event. In addition to nerve palsy, axillary artery injury should be recognized as a potential complication of limb lengthening associated with Grammont-type reverse total shoulder arthroplasty. In a patient with multiple risk factors for neurovascular injury, including multiple prior ipsilateral limb surgeries, a well-fixed shoulder prostheses, or variant anatomy, the authors will proceed with increased caution and consider preoperative vascular imaging.

**REFERENCES**


