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The risk of nerve injury during anatomical and reverse total shoulder arthroplasty: an intraoperative neuromonitoring study

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Background: This study compared the incidence and pattern of potential nerve injuries between reverse shoulder (RSA) and total shoulder arthroplasty (TSA) using intraoperative neuromonitoring. Our hypothesis was that RSA has a greater risk of nerve injury than TSA due to arm lengthening.
Methods: We reviewed 36 consecutive patients who underwent RSA (n = 12) or TSA (n = 24) with intraoperative neuromonitoring. The number of nerve alerts was recorded for each stage of surgery. Neurologic function was assessed preoperatively and postoperatively at routine follow-up visits. Predictive factors for increased intraoperative nerve alerts and clinically detectable neurologic deficits were determined.
Results: There were nearly 5 times as many postreduction nerve alerts per patient in the RSA cohort compared with the TSA cohort (2.17 vs. 0.46). There were 17 unresolved nerve alerts postoperatively, with only 2 clinically detectable nerve injuries, which fully resolved by 6 months postoperatively. A preoperative decrease in active forward flexion and the diagnosis of rotator cuff arthropathy were independent predictors of intraoperative nerve alerts.
Conclusion: RSA has a higher incidence of intraoperative nerve alerts than TSA during the postreduction stage due to arm lengthening. Decreased preoperative active forward flexion and the diagnosis of rotator cuff arthropathy are predictors of more nerve alerts. The clinical utility of routine intraoperative nerve monitoring remains in question given the high level of nerve alerts and lack of persistent postoperative neurologic deficits.
Level of evidence: Level II; Prognosis Study

Keywords: Arthroplasty; Anatomic; Reverse; Nerve injury; Nerve monitoring; Motor evoked potential; Somatosensory evoked potential

Neurologic injury is a known complication of TSA, with reported incidences ranging from 1% to 4%.[1,12] These injuries are most often the result of brachial plexus stretch injuries that occur during positioning of the arm at the extremes of motion.[8,12] RSA may have a greater incidence of neurologic injury than TSA, possibly due to the resultant arm lengthening

The New England Baptist Hospital Institutional Review Board approved this study (approval protocol # 548025).

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and the need for increased glenoid exposure. However, data on the patterns and etiology of neurologic injuries during TSA or RSA are limited.[3,9,12-14,16-18]

Intraoperative nerve monitoring has been shown to be a useful tool used by orthopedic surgeons to avoid neurologic injury in spine surgery[6-8] and shoulder surgery, including TSA,[12,15,16] Latarjet for instability,[4] arthroscopy,[5] and fracture fixation.[20] Despite the previously reported utility of intraoperative nerve monitoring in orthopedics, and particularly during TSA,[12,16] no investigation has assessed the utility of intraoperative nerve monitoring during RSA or compared such monitoring between TSA and RSA.

The purpose of the present study was to compare the incidence and patterns of intraoperative nerve alerts between anatomic TSA and Grammont-design RSA, as detected by continuous intraoperative nerve monitoring, and to identify predictive factors for intraoperative nerve alerts during RSA and TSA.

Materials and methods

Patient selection

We retrospectively reviewed prospectively collected data of 36 consecutive patients who underwent RSA (n = 12) or TSA (n = 24) by a fellowship-trained surgeon (R.L.P.) at a single institution between March and September of 2013.

Inclusion and exclusion criteria

No patients were excluded. To be included in the study, all patients underwent an RSA or anatomic TSA procedure and provided a reliable postoperative clinical neurologic examination for the attending surgeon in the postanesthesia care unit immediately after surgery. All patients were included regardless of comorbid profile, history of rheumatoid arthritis, cervical spine disease, previous rotator cuff surgery, fracture sequelae, previous failed arthroplasty, and current or prior prescribed steroid use.

Surgical technique

Patients underwent RSA or TSA using a deltopectoral approach in the beach chair position with a pneumatic arm holder. The subscapularis was taken down with a lesser tuberosity osteotomy for the TSA and with a subscapularis peel (if present) for the RSA. The coracoacromial ligament was preserved in both procedures. A Zimmer (Warsaw, IN, USA) Anatomical prosthesis was used for all TSAs and a combination of a Zimmer Anatomical stem and a Zimmer Bigliani-Flatow baseplate and glenosphere were used in a Grammont-design RSA.

In both procedures, a humeral cut was made at the anatomic neck at the native version. In the RSA, according to the standard technique, there was some additional reaming of the metaphysis for placement of the onlay prosthesis, which has a 155° neck angle. The glenosphere baseplate was placed as low as possible on the glenoid without compromising fixation and with slight (approximately 5°-10°) inclination. The baseplate creates 2 mm of lateral offset.

Trial reduction was used to determine the optimal tension of the prosthesis. The aim was to obtain tension in which the prosthesis could just be manually reduced. The final trial was not actually reduced because of potential difficulty in redislocation. Therefore, the final prosthesis tension was determined by feel of the senior author (A.J.) with the goal of being "tight" to prevent instability. With nerve monitoring, the patient had no muscle relaxation. Stability was tested at the extreme of external rotation and adduction to assure stability after final reduction. The subscapularis peel or lesser tuberosity osteotomy were repaired with #2 nonabsorbable sutures passed through drill holes in the bicipital groove and wrapped around the prosthesis.

Peripheral nerve blocks were performed after the procedure in the postanesthesia care unit after a thorough neurologic examination by the attending surgeon. All patients underwent a further thorough neurologic examination on all postoperative days by the attending surgeon. No patients were discharged before the nerve block wore off.

Nerve monitoring

All patients underwent a standard anesthesia protocol with propofol for intubation and no muscle relaxation during the procedure. Continuous intraoperative nerve monitoring was recorded using transcranial electrical motor evoked potentials (MEPs), somatosensory evoked potentials (SSEPs), and free electromyogram (EMG), as previously described.[13] Stimulating leads were placed in the scalp, and recording leads were placed in the operative arm after sterile preparation and draping and in the nonoperative arm for reference. MEPs and free EMGs were recorded from deltoid, triceps, biceps, extensor carpi radialis longus, abductor pollicis brevis, and abductor digiti minimi muscles. SSEPs were recorded from the myotomes on the basis of major innervation patterns. Nerve alerts were defined as greater than 80% amplitude attenuation of MEPs or SSEPs, or both, compared with the contralateral arm. Each procedure was divided into 4 stages (surgical approach, humeral preparation, glenoid preparation, and postreduction), with the number of nerve alerts recorded per stage.

Study variables and protocols

At the initial preoperative evaluation, each patient was assessed for active range of motion as well as neurologic function, with no patient demonstrating any preoperative neurologic issues. In the immediate postoperative period, all patients were managed using the same standard shoulder arthroplasty protocol, which included a standard sling for 6 weeks with active assisted and passive range of motion, with external rotation limited to 20°. Each patient had a standard neurologic examination by the senior author (A.J.) in the recovery room each postoperative day and at each postoperative visit assessing the motor and sensory function of the axillary, musculocutaneous, radial, median, and ulnar nerves, respectively. Each patient was evaluated at 2 weeks postoperatively by the senior surgeon (A.J.), and if there was no clinically evident sensory or motor dysfunction (consistent with previous examinations), the patient was no longer monitored as part of this study protocol. If clinically evident neurologic dysfunction was seen, the patients were to be monitored until their symptoms completely resolved or there was no further improvement.

Table I Nerve event breakdown

Affected nerves	TSA	RSA	Total
	No. (%)	No. (%)	No. (%)
Axillary	36 (34.0)	18 (18.6)	55 (27.1)
Radial	19 (17.9)	22 (21.6)	41 (20.2)
Median	17 (16.0)	18 (18.6)	35 (17.2)
Ulnar	9 (8.5)	14 (14.4)	23 (11.3)
Musculocutaneous	10 (9.4)	7 (7.2)	17 (8.4)
Combined	15 (14.2)	18 (18.6)	33 (16.3)
Total events	106	97	203

RSA, reverse shoulder arthroplasty; *TSA*, total shoulder arthroplasty.

Table II Comparison of the number and timing of nerve alerts per procedure

Timing	TSA	RSA	*P* value
Exposure	0.8	1.3	.51
Humerus	1.9	1.8	.64
Glenoid	1.3	2.8	.14
Postreduction	0.4	2.2	.002*
Total events	4.4	8.1	.14

RSA, reverse shoulder arthroplasty; *TSA*, total shoulder arthroplasty.
* Statistically significant ($P < .05$).

Statistical and power analysis

Demographic variables and nerve alerts per stage were compared between RSA and TSA groups using *t* tests and Fisher exact or χ^2 tests, as appropriate. Predisposing patient factors for intraoperative nerve alerts were determined by way of the Wilcoxon rank signed test and the Pearson correlation coefficient test, as appropriate. Post hoc power analysis was performed using the Wilcoxon rank sum test for total number of nerve alerts and subgrouped by alerts per stage of procedure (surgical approach, humeral preparation, glenoid preparation, and postreduction). All analysis was performed by an experienced statistician.

Results

The RSA group consisted of 4 men and 8 women with a mean age of 69.3 years (range, 50-87 years), and the TSA group consisted of 12 men and 12 women ($P = .3$) with a mean age of 63.0 years (range, 37-82 years; $P = .10$). Thirty-four patients did not exhibit a clinically evident postoperative neurologic injury (sensory or motor) and were only monitored to the standard 2-week postoperative evaluation, whereas 2 patients did demonstrate neurologic injury and were subsequently monitored to the 6-month visit, when complete resolution of symptoms was observed.

MEP and SSEP monitoring recorded at least 1 nerve alert in 35 of the 36 patients. A total of 203 alerts were recorded for the combined TSA and RSA cohorts; the TSA and RSA cohorts had 106 and 97 nerve alerts, respectively. The average nerve alerts per procedure for the combined cohorts was 5.6, with 89% being MEP attenuation.

Total shoulder arthroplasty

Of the 106 nerve alerts demonstrated during TSA, 36 were of the axillary nerve, 19 radial, 17 median, 9 ulnar, 10 musculocutaneous, and 15 were combined nerve alerts (Table I). Eighteen nerve alerts occurred during the surgical approach, 31 during glenoid preparation, 46 during humeral preparation, and 11 during the postreduction stage (Table II).

Reverse shoulder arthroplasty

Of the 97 RSA nerve alerts, 18 were of the axillary nerve, 22 radial, 18 median, 14 ulnar, 7 musculocutaneous, and 18 were combined nerve alerts (Table I). Sixteen nerve alerts occurred during the surgical approach, 33 during glenoid preparation, 22 during humeral preparation, and 26 occurring postreduction (Table II).

Total and reverse shoulder arthroplasty

Overall, there were more nerve alerts per RSA procedure (8.08 vs. 4.42), but this finding was not statistically significant ($P = .17$). However, there were nearly 5 times as many postreduction nerve alerts per patient in the RSA cohort (2.17) than in the TSA cohort (0.46; $P = .002$). Post hoc analysis revealed 83% power for the postreduction stage of the procedure, but overall, the study was underpowered to determine a difference in the number of nerve alerts between RSA and TSA.

Of the 203 alerts, 17 nerve alerts (8%) were unresolved postoperatively. However, only 2 patients demonstrated clinically detectable nerve symptoms in the postanesthesia care unit and at the initial clinical follow-up with the operative surgeon. One patient in the TSA group had partial motor (abductor pollicis brevis weakness) and sensory median nerve symptoms, and 1 patient in the RSA group presented partial ulnar sensory nerve symptoms. The total number of intraoperative nerve alerts demonstrated by each of these 2 patients was consistent with the remaining cohort, with both of these clinically detectable nerve deficits reaching full resolution by 6 months postoperatively. No false-negative alerts were observed.

The number of nerve alerts correlated with decreased preoperative active forward flexion ($P = .04$) along with the preoperative diagnosis of rotator cuff arthropathy ($P = .04$). Previous open shoulder surgery was not predictive ($P = .11$; Tables III and IV).

Discussion

Partially consistent with our hypothesis, RSA demonstrated a higher incidence of intraoperative nerve alerts than

Table III Demographics and range of motion correlation with nerve events

Variable	Mean	Pearson correlation	*P* value
Demographics			
Age	65.1	−0.14	.41
Body mass index	29.8	0.21	.23
Range of motion			
External rotation	24.3	0.01	.94
Forward flexion	94.0	−0.39	.04*

* Statistically significant ($P < .05$).

Table IV Predisposing factors by character variables

Variables	Yes	No	*P* value
Predisposing Factors			
Gender			.68
Male	16	20	
Female	16	20	
Diabetes	8	28	.54
Osteoarthritis	29	7	.95
Rheumatoid arthritis	2	34	.35
Cervical spine disease	2	34	.65
Methotrexate use	1	35	.96
Steroid use	3	33	.37
Prior surgery or pathology			
Rotator cuff repair	6	30	.75
Open shoulder surgery	8	28	.11
Rotator cuff arthropathy	7	29	.04*
Fracture Sequelae	5	31	.07

* Statistically significant ($P < .05$).

anatomic TSA during the postreduction stage of the procedure due to subsequent arm lengthening inherent in the Grammont-design RSA. Overall, there was a trend toward more nerve alerts in the RSA cohort, but the difference was not statistically different in this underpowered study. There were 17 unresolved nerve alerts (8%) postoperatively, with only 2 patients demonstrating clinical deficits at initial follow-up. Both reached full resolution by 6 months. There were no false-negative nerve alerts.

Nerve injury is a serious but uncommon complication of shoulder arthroplasty.[3] The patient factors that contribute to these complications were elucidated by Nagda et al[16] in their prospective neuromonitoring study of conventional TSA. They found that limited preoperative external rotation and extreme external rotation during humeral and glenoid preparation were risk factors for nerve injury. More recently, some authors have shown a greater risk for nerve injury after RSA than after conventional TSA.[3,7] These studies suggest that with the Grammont-design prosthesis, the brachial plexus is stretched with arm lengthening after the final reduction of the humeral tray to the glenosphere, which may predispose the patient to neurologic injuries during surgery.[16] In addition, anatomic cadaveric analysis has demonstrated that the main anterior branch of the axillary nerve may be as close as 2 mm from the humeral implants, potentially increasing the risk of injury.[13] The need for increased glenoid exposure and prolonged retraction for the larger glenosphere implant may also increase the risk of nerve injury.[1,2]

Using the same protocol as Nagda et al,[16] we used transcranial electrical MEPs and SSEPs for 36 consecutive RSAs and conventional TSAs with intraoperative nerve alerts analyzed at well-defined stages of the surgical procedure. Although the difference between the total number of nerve alerts per procedure for TSA vs RSA was not significant, there was a striking increase in the number of alerts during the postreduction stage for the RSA group compared with the TSA group with adequate power of 83%. This finding is consistent with other reports, which suggest that the effect of arm lengthening may result in tensioning of the brachial plexus and predispose patients to nerve injuries. Unfortunately, our study is underpowered to evaluate the overall number of nerve alerts for TSA vs. RSA, although the trend of increased alerts with RSA is clear.

The exact amount of strain on the brachial plexus during RSA is unknown. However, Wall et al[19] reported in a nerve conduction study using a rabbit tibial nerve model that a 6% strain decreased action potential amplitudes by 70% at 1 hour, with complete recovery within hours. A 12% strain showed complete loss of action potentials, with minimal recovery thereafter. Using guinea pig sciatic nerves, Rickett et al[17] demonstrated no effect on conduction testing with 5% strain; however, further elongation decreased the amplitude linearly related to the amount of strain.

Without significantly increased alerts between the TSA and RSA groups seen during glenoid preparation, the need for increased glenoid exposure for RSA does not appear to be related to increased nerve alerts in our study. However, most of the nerve alerts for both groups in this study occurred during humeral (43% for TSA vs. 23% for RSA) and glenoid preparation (29% for TSA vs. 34% for RSA) while the arm is in external rotation. This accounted for 65% of all nerve alerts in both groups.

We theorize that when the arm is in external rotation during surgery, increased tension and strain is placed on the brachial plexus, resulting in nerve alerts. Delaney et al[4] also reported 79.4% of all nerve alerts during the Latarjet procedure were during arm external rotation. Warrender et al[20] reported 58% of all their intraoperative nerve alerts during proximal humeral fixation occurred with arm external rotation and abduction. Thus, the surgeon must be cognizant during the glenoid or humeral preparation stage of the TSA or RSA procedure of the amount and the time the arm is externally rotated. If prolonged time is spent in this arm position due to difficulties encountered in surgery, we recommend putting the arm in a neutral position intermittently to rest and reduce the strain on the plexus to avoid nerve injury.

Our findings support the direct relationship between arm lengthening after reduction and neurologic injury. This relationship was initially suggested by Van Hoof et al[18] with

their computerized 3-dimensional model of the brachial plexus showing that a Grammont-design RSA led to significant strain on the plexus that could lead to neurologic injury. Lädermann et al[12] also supported this theory in their prospective study comparing preoperative and postoperative EMG evaluations and arm lengths of patients undergoing conventional TSA and RSA. They found more subclinical changes on EMG performed 3.6 weeks after the RSA (47%) vs. TSA (0.4%) and also a mean arm lengthening of 2.7 cm in the RSA group. Most of the EMG changes in the RSA group involved the axillary nerve, although the injury was completely resolved in 8 of 9 patients at the 6-month follow-up visit. The authors reported 10.9-times higher risk of acute postoperative nerve injury in the RSA group, but most of these nerve injuries are transient. They were unable to create a threshold for the amount of arm lengthening that led to EMG changes but speculated a direct correlation between the two.

The 17 unresolved nerve alerts in our study reinforce the fact that there may be subclinical nerve changes after both RSA and TSA. Nerve injury may present without obvious sensory or motor dysfunction but as pain. In addition, nerve injury most likely occurs during external rotation for humeral and glenoid preparation stage and in the reduction/arm-lengthening stage of RSA.

Our observations also raise the question of whether a non-Grammont design that lateralizes the glenosphere (or the humerus) has less brachial stretch and fewer nerve injuries. Lädermann et al[10] reported decreased acromiohumeral distance with a lateralizing design but did not address the correlation with subclinical or clinical nerve injury. In addition, the optimal deltoid lengthening required for the RSA to drive the prosthesis while minimizing brachial plexus strain is not fully elucidated.

This concern has been partially addressed in the literature with a recent systematic review emphasizing the importance of deltoid tensioning and suggests maximal humeral lengthening <2 cm to avoid postoperative clinical neurologic impairment.[11] An additional study found that anterior active elevation is directly related to deltoid tensioning with significantly greater anterior active elevation (difference of 23°) demonstrated with arm lengthening during RSA.[14] Additional biomechanical and clinical studies are needed to help provide more complete answers to the above questions with further correlation with clinical outcomes.

We found that the diagnosis of rotator cuff arthropathy and limited forward flexion correlated with increased nerve alerts. The reasons for these relationships are not clear; however, one theory is that proximal humeral head migration is observed in patients with rotator cuff arthropathy. In this subset of patients that develop proximal humeral head migration, more distalization of the center of rotation is needed to facilitate reduction of the humeral tray to the glenosphere, thus resulting in more arm lengthening and brachial plexus tensioning or strain. Importantly, a larger study may have shown a difference in the nerve alerts between RSA and TSA because we found nearly twice as many alerts in the RSA group, but the difference was not statistically significant. A post hoc power analysis showed this particular comparison to be underpowered.

Lastly, our study brings into question the routine clinical utility of intraoperative nerve monitoring for shoulder arthroplasty. There were no false-negative results (postoperative nerve findings with no nerve alerts at the end of the procedure), but there were 15 false-positive nerve alerts. Although an EMG may show some intraoperative changes, as Nagda et al[16] and Lädermann et al[12] demonstrated, the clinical examination was unaffected. Furthermore, Warrender et al[20] reported only 3 patients had transient nerve palsies after proximal humeral fixation (26 intraoperative nerve alerts), and all fully resolved within 3 weeks of surgery.

Intraoperative neuromonitoring for shoulder arthroplasty will significantly increase the cost and length of surgery, thus we critically question the true benefit or value. Garces et al[7] reported no added benefit of intraoperative neuromonitoring for 1 or 2 level lumbar fusion after analysis of 73 patients. The hospital length of stay and pedicle revision rate were similar compared with 39 patients who had the same operation but with no nerve monitoring. However, the cost of surgery and the length of surgery were significantly higher. Given the costs, routine use of neuromonitoring may be hard to justify, whereas patients at higher risk for nerve injury may be better served with its use. Importantly, though, nerve injury may present as pain and not clinically obvious nerve dysfunction.

This investigation is valuable because it evaluates consecutive patients of a single surgeon with the same approach and technique. Further, the findings are valuable because they have implications for prosthetic design and the use of intraoperative nerve monitoring.

Our study is limited, however, by its small numbers and the inherent bias associated with being a retrospective review. In addition, the neurologic examinations were performed by the operative orthopedic surgeon rather than by an independent observer. However, the senior surgeon (A.J.) performed every neurologic examination at every visit with great detail to both sensory and motor dysfunction for all peripheral nerves.

Conclusions

RSA has a higher incidence of intraoperative nerve alerts in the postreduction stage compared with TSA due to the resultant arm lengthening. In addition, rotator cuff arthropathy and a preoperative decrease in active forward flexion are independent predictors of intraoperative nerve alerts. However, the clinical utility of routine intraoperative nerve monitoring remains in question given the high incidence of false-positive nerve alerts and the lack of clinical correlation with persistent postoperative neurologic deficits.

Disclaimer

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