JSES Reviews, Reports, and Techniques 2 (2022) 458–463

Contents lists available at ScienceDirect

JSES Reviews, Reports, and Techniques

journal homepage: www.jsesreviewsreportstech.org

Prevalence, management, and outcomes of nerve injury after shoulder arthroplasty: a case-control study and review of the literature

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ARTICLE INFO

Keywords:
Reverse shoulder arthroplasty
Total shoulder arthroplasty
Nerve injury
Neurapraxia
Nerve recovery

Level of evidence: Level III; Prognostic study

Background: Neurologic injury is a rare and potentially devastating complication of shoulder arthroplasty. Patients typically present with a mixed plexopathy or mononeuropathy, most commonly affecting the axillary and radial nerves. Given the paucity of studies available on the topic, our goal was to elucidate the prevalence of nerve injury after shoulder arthroplasty and to describe the treatment course and outcomes of neurologic injuries.

Methods: This is a retrospective case-control study performed at a single, urban, academic institution. Consecutive patients who underwent anatomic total shoulder arthroplasty (TSA) or reverse shoulder arthroplasty (RSA) by a single surgeon from 2014 to 2020 were reviewed, and patients with a documented nerve injury were identified. A control group of patients without nerve injury were selected in a 2:1 ratio controlling for age and procedure type (TSA vs. RSA; primary vs. revision). Data collected included demographics, comorbidities as per the Charlson Comorbidity Index, radiographic evaluations, surgical and implant details, patient-reported outcome measures, and perioperative complications.

Results: Of 923 patients, 33 (3.6%) sustained an iatrogenic nerve injury: 10 (2.1%) after TSA, 23 (5.0%) after RSA, and 3 (7.8%) after revision arthroplasty. Axillary mononeuropathy was most common (42%), followed by brachial plexopathies (18%). There was no significant difference in age, sex, race, body mass index, and preoperative diagnoses between groups. Patients with nerve injury had fewer comorbidities (Charlson Comorbidity Index <3, 33 vs. 65%, $P<.001$). Patients with nerve injury had higher rates of cervical spine pathology (15 vs. 6%; $P = .15$) and increased postoperative lateralization (8.9 mm [7.2] vs. 5.5 mm [7.3]; $P<.06$). The majority (91%) were managed with observation alone. Three (9%) underwent an additional procedure: carpal tunnel release (1, 3%), ulnar nerve decompression (1, 3%), and ulnar nerve transposition (1, 3%) for peripheral compressive neuropathies. At the final follow-up, 19 (57%) nerves fully recovered, and 14 (43%) showed mild residual sensorimotor dysfunction. The mean time to first sign of recovery and ultimate recovery were 11 (7.2) and 36 (23.5) weeks, respectively. At the final follow-up, patients with nerve injury performed worse on patient-reported outcomes, including visual analog score pain (2.2 vs. 1.0, $P<.001$), American Shoulder and Elbow Surgeons score (67.8 vs. 84.8, $P<.001$), and Single Assessment Numeric Evaluation scores (62 vs. 77, $P = .009$).

Discussion: Nerve injury after shoulder arthroplasty is rare, occurring in 3.6% of our patient population. Axillary mononeuropathy and brachial plexopathies are the most common. Most patients can be managed expectantly with observation and will recover at least partial nerve function, although clinical outcomes remain inferior to those without nerve complication.

Shoulder arthroplasty is increasingly becoming used for the treatment of end-stage glenohumeral joint osteoarthritis. Iatrogenic nerve injuries are a known complication with an incidence ranging from 1% to 4%.[1,12,23] Clinically, patients may present with a mixed plexopathy or mononeuropathy, with the most common being the axillary and suprascapular nerves.[1,7,18] Although studies have highlighted the transient nature of this complication, the management of nerve injuries remains challenging to both surgeon and patient. The current literature lacks a comprehensive report on iatrogenic nerve injury with respect to epidemiology and presentation, risk factors, management and recovery, and patient-reported outcomes (PROs).

Mass General Brigham Institutional Review Board approved this study (Protocol #: 2021P000201).

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https://doi.org/10.1016/j.xrrt.2022.04.009

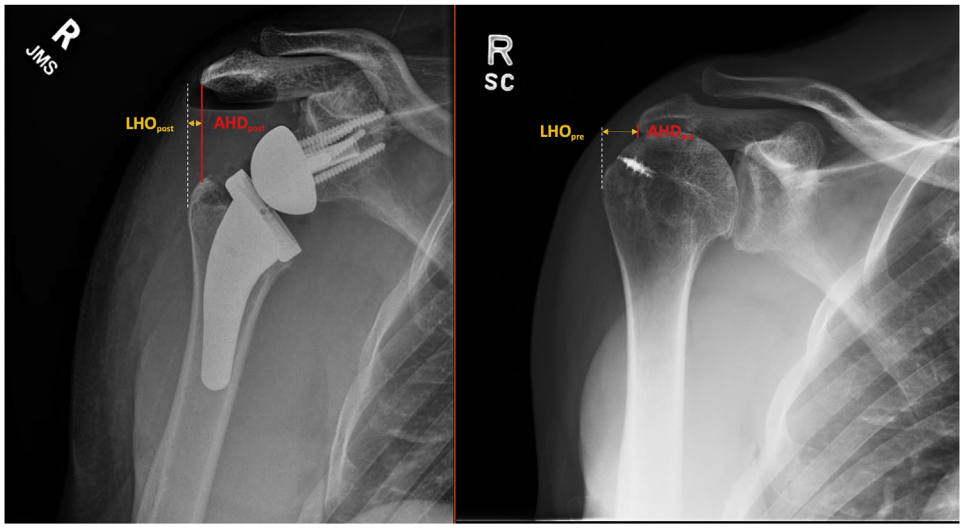

Figure 1 A diagram illustrating humeral distalization (*left*) and lateralization measurements (*right*). Distalization was measured as the absolute difference between post- and preoperative measurements of acromiohumeral distance (AHD), whereas lateralization was measured as the absolute difference between postoperative and preoperative lateral humeral offset (LHO).

Several authors have attempted to elucidate the etiology of nerve injury after shoulder arthroplasty. Studies using intraoperative neurophysiologic monitoring have shown that traction injuries of the brachial plexus can result from arm position, retractor placement, and distraction of the arm.[1,7,12,18,19] Furthermore, instrumentation of the glenoid or humerus in preparation for implant placement have been described as a possible etiology.[7,18] Finally, toxicity from peripheral nerve block has been shown to be a risk factor for nerve injury after shoulder arthroplasty.[5,15,22,23]

Few studies have discussed clinical outcomes of nerve injury after shoulder arthroplasty, focusing instead on etiology and pathology of the neurologic lesion. Reassuringly, authors have reported spontaneous recovery with observation in small patient populations. Aleem et al and Nagda et al described spontaneous resolution of nerve injury after shoulder arthroplasty in 2 (out of 282) and 5 (out of 30) patients, respectively.[1,18] In a larger study, Kim et al observed 34 nerve injuries in 182 shoulders, all of whom completely recovered.[10] Overall, most of these studies focus on the etiology and pathology of the neurologic injury rather than the treatment course and outcome after the complication.

Although academic interest has highlighted the prevalence and etiology of nerve injury after shoulder arthroplasty, descriptions of treatment or outcome data for these nerve injuries are absent. As such, the goal of our study was to describe on the prevalence, treatment course, and outcome of these neurological injuries.

Material and methods

This is a retrospective case-control study performed at a single, urban, academic, institution between 2014 and 2020. After approval by the institutional review board, data were extracted through a retrospective review of patient medical charts and available radiographic studies. The study group included patients aged >18 years who underwent primary shoulder arthroplasty (total or reverse, Current Procedural Terminology code 23472) or revision shoulder arthroplasty (Current Procedural Terminology code 23473) performed by the senior author and had evidence of postoperative neurologic injury documented in the medical records (palsy on clinical examination, nerve conduction study [NCS] results). Patients with pathologic fractures from neoplastic disease and <6 months of clinical and radiographic data were excluded. Subsequently, a control group was randomly selected in a 2:1 ratio matching for age, sex, race, body mass index (BMI), type of arthroplasty (RSA vs. TSA), and primary vs. revision procedure.

Data collected included patient demographics, medical and social comorbidities, American Society of Anesthesiologists (ASA) scale, Charlson Comorbidity Index (CCI), perioperative data and surgical technique, implant characteristics, radiographic measurements (modified Walch and Favard classifications of glenoid morphology,[4,6] preoperative posterior humeral subluxation, postoperative humeral distalization and lateralization, pre- and postoperative pain scores (numerical rating scale), Single Assessment Numeric Evaluation (SANE) score[24] and American Shoulder and Elbow Surgeons shoulder score (ASES),[21] and perioperative complications. Distalization was measured as the absolute difference between post- and pre-operative measurements of acromiohumeral distance, while lateralization was measured as the absolute difference between postoperative and preoperative lateral humeral offset (Fig. 1). The degree of posterior humeral subluxation was measured as the percentage of humeral head posterior to a line through the axis of the scapular spine exiting the mid-glenoid.

The primary outcome measure was the prevalence of neurological deficits following shoulder arthroplasty calculated by the number of nerve injuries divided by the total number of cases over the defined study period. The secondary outcome measures gathered by the postoperative outpatient visits included prevalence of nerve injury by surgery type, type of nerve injured, time to nerve injury diagnosis, type, evaluation of nerve injury (electromyography/NCS or MRI), treatment for neurological deficits (splint, therapy, hand surgeon referral, and surgery), resolution of nerve injury, time to initial sign of nerve recovery, and time to ultimate nerve recovery (plateau/full improvement).

Results

From 2014 to 2020, 923 shoulder arthroplasties were performed. Of 923 patients, 33 (3.6%) sustained a clinically relevant, iatrogenic nerve injury: 10 (2.1%) after TSA, 23 (5.0%) after RSA, and

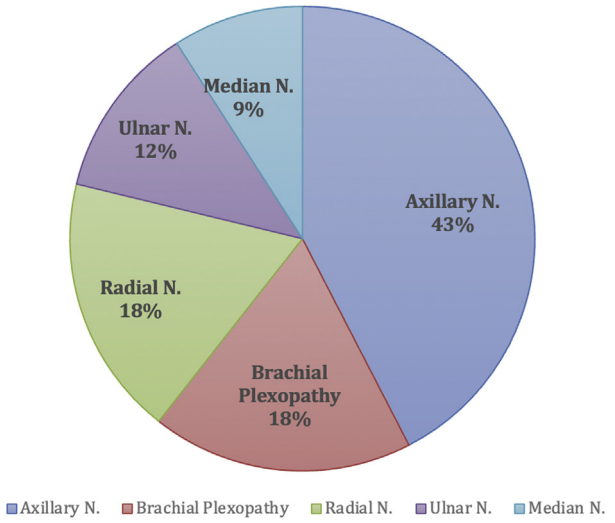

Figure 2 Distribution of nerve injury following reverse shoulder arthroplasty. *N*, nerve.

3 (7.8%) after revision arthroplasty. The nerve injury cohort had a mean age of 65 years (standard deviation [SD] 9.4), mean BMI of 29.8 kg/m^2 (SD 5.5), was 61% female, and reported mean (SD) follow-up duration of 18 (10) months. Compared with controls, there was no significant difference in age, sex, race, BMI, or surgery type (Table I). By ASA class and CCI, there was a higher rate of medical comorbidity (ASA3+, CCI 3+) in the control group (39 vs. 18%, $P = .03$ and 59% vs. 33%, $P = .02$). There were similar rates of tobacco, alcohol, and drug use between groups (Table I).

Most patients were indicated for surgery for glenohumeral osteoarthritis (61%) and rotator cuff tear arthropathy (34%), and there were no differences between the 2 cohorts ($P = .14$). Ninety (91%) patients were primary arthroplasty procedures. Further surgical characteristics are outlined in Table II. There was no significant difference in glenoid morphology, percent posterior humeral head subluxation, and distalization or lateralization between cohorts (Table II). All but one control underwent preoperative regional nerve block. There was a nonstatistically significant increase in operative time (128 [SD 47] vs. 118 [32] min, $P = .27$) and estimated blood loss (227 [SD 129] vs. 204 [SD 96] cc, $P = .37$) in the nerve injury cohort.

The most common presentations of nerve injury after shoulder arthroplasty was axillary nerve mononeuropathy (14, 42%) and brachial plexopathy (6, 18%), although 5 distinct presentations were noted (Fig. 2). All median and ulnar nerve injuries were peripheral compressive neuropathies. The average time to diagnosis was 26.2 (28.8) days postoperation. Most patients (30, 91%) were treated conservatively with observation alone, 11 (33%) with bracing to stabilize a wrist drop, and 9 (27%) with occupational therapy (Table III). Eighteen patients (55%) received hand surgery consultation, and 3 (9%) underwent an additional procedure for compressive peripheral neuropathy: carpal tunnel release (1, 3%), ulnar nerve decompression (1, 3%), and ulnar nerve transposition (1, 3%). At the final follow-up, 19 (57%) nerves fully recovered, and 14 (43%) experienced improvement but showed residual sign of sensorimotor dysfunction at the final follow-up. The mean (SD) time to first documented sign of recovery was 11 (7.2) weeks, whereas ultimate recovery was 36 (23.5) weeks. By nerve type, the median time to first (final) sign of recovery was 8.4 (46.8) weeks (brachial plexus), 11.9 (29.0) weeks (axillary), 8.5 (16.8) weeks (radial), 7.3 (26.9) weeks (ulnar), and 5.6 (57.7) weeks (median). Patients with nerve injury reported statistically significant worse postoperative visual analog pain scores (2.2 ± 2.2 vs. 1.0 ± 1.5, $P<.001$), subjective shoulder values (62 ± 31.5 vs. 77 ± 25.2, $P = .009$), and ASES scores (67.8 ± 27.2 vs. 84.8 [14.2], $P < .001$) at final follow-up (Table IV).

Discussion

The goal of our study was to present a comprehensive evaluation of nerve injuries following shoulder arthroplasty, including the epidemiology, risk factors for injury, and management and recovery. We report a prevalence of 3.6%, with majority presenting as axillary mononeuropathies. RSA (5%) and revision arthroplasty (7.8%) demonstrated higher rates than conventional TSA (2.1%). History of cervical spinal stenosis and prior open shoulder surgery were associated with higher rates of nerve injury; however, we were underpowered to detect a difference. In spite of nerve injury, 100% of patients experienced complete (57%) and partial (43%) recovery. All proximal nerve injuries were managed conservatively, whereas just 3 of 10 peripheral compressive neuropathies required carpal tunnel or cubital tunnel surgery.

Incidence

The prevalence of clinically significant iatrogenic nerve injury in our study was 3.6%. The most common presentation was axillary mononeuropathy (43%) and brachial plexopathy (18%), although radial, ulnar, and median mononeuropathies were also reported. Our prevalence closely resembles that of an early study by Lynch et al who found a 4% rate of nerve palsy following 417 shoulder arthroplasties. Their distribution, however, included 17 of 18 (94%) brachial plexopathies and one carpal tunnel syndrome (6%).[17] Subsequent investigations reported varying rates of nerve palsy. Barlow et al identified 2 nerve injuries in a smaller cohort of 91 patients (2.2%) undergoing RSA, both of which were brachial plexopathies.[3] Ball identified a 21% rate of nerve injury in 211 RSA, TSA, and hemiarthroplasty cases. Similarly, most injuries (86%) were brachial plexopathies followed by cervical radiculopathies (5%) and cubital tunnel (5%) and carpal tunnel compressive neuropathies.[2] Kim et al reported a 19% prevalence in 182 RSAs, most common injuries to the axillary nerve (41%), radial nerve (18%), and brachial plexopathies (18%), mirroring the results of our study.[10]

Risk factors

Of the existing studies available, few have evaluated the risk factors of iatrogenic nerve injury during shoulder arthroplasty. Many propose that the risk of nerve injury is higher in RSA than TSA due to arm lengthening causing a traction neurapraxia.[1,17,18] In our study, we controlled for procedure type and could not evaluate this as a risk factor, although overall, our historical rates were higher in RSA (5.0%) than TSA (2.1%). In a prospective study evaluating patients with nerve injury, Läderman found a higher rate after RSA than TSA (47% vs. 4%). They determined that patients undergoing RSA are at 10 times higher risk for nerve injury. Similarly, using IONM, Parisien et al reported 5 times as many nerve alerts during RSA than TSA and attributed this difference to lengthening.[19] However, Ball and Lowe et al failed to identify a difference in rates of nerve injury after RSA and TSA (23 vs. 20% and 4% vs. 4%, respectively).[2,16]

Kim et al specifically evaluated the effect of distalization during RSA by measuring the comparing change in acromiohumeral distance postoperatively between patients with and without injury. They found this to be an independent risk factor, and patients with nerve injury were on average 4 mm longer than patients without injury.[10] However, they did not identify lateralization (change in lateral humeral offset) as a risk factor. Furthermore, Lowe et al

Table I
Patient characteristics and demographics.

Variable	Nerve injury (n = 33)		Control (n = 66)		*P* value	Significance
	n	%	n	%		
Age*	65	9	69	9	.05	NS
Sex						
Female	20	61	37	56		
Male	13	39	29	44	.83	NS
Race						
White	30	91	61	92		
Non-white	3	9	5	8	.79	NS
Body mass index	29.8	5.5	8.3	7.2	.52	NS
Surgery						
TSA	10	30	20	30		
RSA	23	70	46	70	.58	NS
ASA						
1	0	0	1	2		
2	27	82	41	65		
3	6	18	21	33	.20	*
CCI						
0	0	0.0	0	0.0		
1	7	21	8	12		
2	15	46	15	23		
3+	11	33	43	65	<.001	***
Cervical spine pathology						
Yes	5	15	4	6		
No	28	85	62	94	.13	NS
Prior trauma						
Yes	13	39	21	32		
No	20	61	45	68	.45	NS
Prior open shoulder surgery						
Yes	7	33	14	33	1.0	NS
No	26	66	52	33		
Prior neuroleptic use						
Yes	3	9	8	12		
No	30	91	58	88	.65	NS
Tobacco use						
Yes	2	6	3	5		
No	31	94	63	95	.54	NS
EtOH use						
Yes	18	55	0	0		
No	15	45	0	0	.2	NS
Illicit drug use						
Yes	2	6	0	0		
No	31	94	66	100	.11	NS

TSA, total shoulder arthroplasty; *RSA*, reverse shoulder arthroplasty; *ASA*, American Society of Anesthesiologists; *CCI*, Charlson Comorbidity Index; *EtOH*, alcohol; *NS*, not significant.
*Mean (standard deviation).
***Statistically significant.

Table II
Comparison of surgical characteristics of patients with and without nerve injury.

Variable	Nerve injury (n = 33)		Control (n = 66)		*P* value	Significance
Diagnosis					.14	NS
Osteoarthritis	18	55	40	61		
Posttraumatic arthritis	0	0	0	0.0		
Cuff tear arthropathy	9	27	17	26		
Avascular necrosis	0	0	0	0.0		
Acute fracture	3	9	3	5		
Failed arthroplasty	3	9	6	9		
Glenoid morphology						NS
A1	6	18	6	10	.23	
A2	4	12	14	21		
B1	4	12	1	2		
B2	4	12	16	24		
B3	7	21	11	17		
C	2	6	0	0		
D	0	0	1	2		
E0	0	0	0	0		
E1	0	0	3	5		
E2	2	6	8	12		
E3	2	6	3	4		
Posterior subluxation (%)	62	13	61	14	.73	NS
Primary vs. revision						
Primary	30	91	60	86		
Revision	3	9	6	14	1.0	NS
Regional block						
Yes	32	97	64	97		
No	1	3	2	3	.33	NS
Subscapularis takedown						
LTO	11	33	26	39		
Subscapularis peel	22	67	32	49	.82	NS
Tenotomy	0	0	8	12		
Operative time	132	47	128	22	.61	NS
EBL	227	129	199	78	.18	NS
Lateralization (all)	8.9	7.2	5.5	7.3	.06	NS
TSA	9.7	5.4	5.9	7.4	.12	
RSA	8.6	8.1	5.3	7.3	0	
Distalization (all)	19.2	3.6	20.4	4.7	.62	NS
TSA	7.0	1.9	6.2	4.6	.61	
RSA	24.5	4.2	26.6	4.7	.29	

LTO, lesser tuberosity osteotomy; *EBL*, estimated blood loss; *TSA*, total shoulder arthroplasty; *RSA*, reverse shoulder arthroplasty.

found the rate of sensorimotor nerve alerts using IOMN to be similar when comparing a lateralized glenoid RSA designed compared with anatomic arthroplasty in spite of a mean 10 mm difference in arm lengthening. The rate of iatrogenic nerve injury was also similar in each group (4%).[16] Using the same method as Kim et al, we did not find any statistically significant difference in distalization or lateralization between patients with and without nerve injury. Though, we did observe a nonstatistically significant increase in lateralization in all comers (RSA and TSA) with nerve injury compared with controls (mean 8.9 [7.2] mm vs. 5.5 mm [7.3], $P = .06$). We were likely underpowered to detect a difference.

Prior studies have evaluated the role of RSA implant preparation and placement in iatrogenic nerve injury. In a cadaveric study, Läderman et al found the axillary nerve in direct contact with the humeral metaphyseal component in 3 of 6 specimens, raising concern of direct trauma during reaming and/or implantation of the components.[13] Lenoir et al evaluated the role of arm positioning on nerve stress experienced during RSA.[14] Via tensiometer, they found that nerve stress significantly increased for the entire plexus during humeral exposition with increasing arm extension and external rotation. Furthermore, nerve stress increased for all nerves, except median and ulnar during glenoid exposure. Finally, axillary nerve stress increased with increasing polyethylene thickness during component trialing. The results of this study further corroborate the theory that injury arises from traction neuropraxia rather than neurotmesis or axonotmesis.[1,17,18]

We evaluated other surgical-related factors and their potential role in iatrogenic nerve injury. It is believed that revision surgery or prior open shoulder surgery may increase the risk of nerve injury secondary to scarring, decreased nerve tissue compliance, and prolonged arm positioning. We controlled for revisions and thus could not directly evaluate its effect, although overall, it did demonstrate a higher rate (7.8%) of nerve injury. Ball et al found a lower rate of nerve injury in revision arthroplasty (18%) compared with primary RSA (23%), TSA (20%), and hemiarthroplasty (9%).[2] Although Patel et al reported 22% rate of nerve alerts using IOMN in a cohort of 44 revision shoulders, they reported no clinically significant nerve injuries, suggesting IOMN overestimates the rate of nerve pathology.[20] A systematic review of 341 revision shoulder arthroplasties with minimum 24-month follow-up by Knowles et al identified just 3 (0.8%) nerve injuries (all radial), suggesting the risk is low.[11] Evaluating patients with prior nonarthroplasty shoulder

Table III
Evaluation and management of nerve injury.

Variable	N	%
Time to diagnosis (days, mean, SD)	26.2	29
Evaluation		
EMG/NCS	26	79
MRI	2	6
Initial management		
Observation	30	91
Bracing	11	33
OT	9	27
Hand consultation	18	55
Surgical intervention	3	9
Carpal tunnel release	1	3
Ulnar nerve decompression	1	3
Ulnar nerve transposition	1	3
Outcome		
Complete recovery	19	57
Improved	14	43
Time to:		
Initial recovery (weeks)	11	7.2
Ultimate recovery (weeks)	36	23.5

SD, standard deviation; *EMG/NCS*, electromyography/ nerve conduction study; *MRI*, magnetic resonance imaging; *OT*, occupational therapy.

surgery, there was no difference in the rate of prior surgery between patients with and without nerve injury (33 vs. 33%, $P = 1.0$). Frank et al compared outcomes and complications after shoulder arthroplasty between patients with and without prior shoulder surgery. Although overall complication was higher (19 vs. 4%) in patients with prior surgery, the rate of neurologic injury (axillary nerve neurapraxia) was lower (0 vs. 0.2%).[8]

Other preoperative patient variables may be related to nerve injuries after shoulder arthroplasty, including shoulder stiffness, cervical spine pathology, and neuroleptic agents. Parisien et al and Nagda et al evaluated the effect of preoperative range of motion and found that decreased passive forward flexion and external rotation of less than 10 degrees were risk factors for nerve injury.[18,19] In our cohort, we did not find any significant differences in passive range of motion between groups. We hypothesized that preexisting cervical spine pathology may make the cervical nerve roots more susceptible to traction neurapraxia. We identified nonstatitistically significant higher rate of cervical spine pathology in patients with nerve injury (15% vs. 6%, $P = .13$), equating to a 3.5 higher risk of nerve injury. We believe we may have been underpowered to detect a significant difference. We evaluated the use of neuroleptic drugs, given their theorized neurocytotoxic effects; however, we found no association between nerve injury and neuroleptic use. Lynch et al did identify methotrexate to be associated with nerve injury and, while not explicitly discussed, may be possibly because of its cytotoxic effect.[17]

Management, nerve recovery, and outcome

In our study, the mean (median) time until the diagnosis of nerve injury was 26.2 (10) days, coinciding with most patients' first follow-up. Delayed diagnosis might have been explained by the fact that patients remained strictly immobilized for the first 4-6 weeks postoperatively, and deltoid weakness would not be tested or noted until well after. Most (26, 79%) patients received EMG/NCS evaluation, and 18 (55%) were referred to a hand surgeon, peripheral nerve specialist for further evaluation. Overall, 30 (91%) patients were managed conservatively, although one-third (11, 33%) of patients required bracing for radial nerve-related wrist drop. Three patients required decompression of a peripheral nerve compressive neuropathy (one carpal tunnel release, one cubital tunnel release, and one ulnar nerve transposition). At the final follow-up, 100% of patients with nerve injury recovered, with 57% exhibiting full recovery and 43% exhibiting residual sensorimotor deficits at their most recent follow-up. A review of the literature demonstrates that most nerve complications resolve without issue, albeit quite slowly. Kim et al found that although all nerve injuries recovered with expectant management, the mean time to final recovery was 7.4 months and ranged from 2 to 38 months.[10] Simple neurapraxias resolved more quickly (mean 2.4 months), whereas patients with axonotomesis recovered much more slowly (mean 22, 10-38 months).[10] The mean time to recovery was shorter for most mononeuropathies: axillary (3.4 months), radial (4.8 months), and median (5.3 months), whereas polyneuropathies recovered more slowly and was variable depending on nerves involved (mean time 10-19 months).[10] Ball reported that all but 4 in a cohort of 44 nerve injuries fully recovered.[2] The author reported that individual nerve injuries typically resolved within 2-8 months, whereas combined nerve injuries were slower to recover (12-24 months), and the presence of neuropathic pain typically portended a slower recovery.[2] Läderman et al found that 9 of 11 nerve injuries completely recovered at a mean of 6 months, whereas 2 brachial plexus injuries exhibited only partial recovery at 18-month follow-up. In our study, nerves exhibited initial signs of recovery at a mean time of 11 (7.2) weeks. Full recovery or reaching their plateau was noted at a mean time of 36 (23.5) weeks.

There are limited data available surrounding functional or PRO scores following shoulder arthroplasty–related nerve injuries. Kim et al reported no difference in VAS (1.7 vs. 1.3, $P = .082$), University of California Los Angeles Shoulder Rating (30.6 vs. 30.8, $P = .463$) or Constant scores (80.6 vs. 81.5, $P = .260$) between patients with and without nerve injury at an average final follow-up of 58 (24-124) months.[10] In our study, we found that on average, VAS pain scores were higher (2.2 vs. 1.0, $P < .001$), whereas ASES (67.8 vs. 84.8, $P < .001$) and SANE (62 vs. 77, $P = .009$) scores were lower at final follow-up in patients with nerve injury. We hypothesize that the difference between our studies could be explained by the shorter follow-up in our patients with nerve injury, capturing their pain and functional outcome scores earlier in recovery. Though, Grubhofer et al reported speed of recovery for common shoulder surgery procedures; VAS, SANE, and ASES scores plateaued between 3-6 months (pain), 6-12 months (SANE, ASES) for RSA and TSA specifically.[9] This, in the context of our reported mean time to nerve recovery (~8 months), suggests our follow-up is long enough to detect a clinically relevant difference in PROMs between patients and without nerve injury or at least suggests that nerve injury delays the time it takes to reach peak PROM scores. Kim et al found that all nerve injuries recovered, so their final follow-up scores likely approach those without nerve injury more closely, given the minimal residual deficit.

Table IV
Comparison of pre- and post-operative patient-reported outcomes.

Variable	Nerve injury		Control		*P* value	Significance
	Value	SD	Value	SD		
Preoperative						
VAS	7.2	1.9	4.9	2.7	<.001	*
SANE	30	14.3	33.4	20.2	.39	NS
ASES	32.4	10.6	43.6	16.7	.02	*
Final follow-up						
VAS	2.2	2.2	1	1.5	<.001	*
SANE	62	31.5	77	25.2	.009	*
ASES	67.8	27.2	84.8	14.2	<.001	*

VAS, visual analog score (pain); *SANE*, Single Assessment Numeric Evaluation; *ASES*, American Shoulder and Elbow Surgeons shoulder score.
*Statistically significant.

Limitations

We acknowledge the limitations of our study. First, our study group size was small, and while we controlled for a number of confounding variables, we were underpowered to detect statistically significant differences in variables of interest (ie, cervical spinal disease, prior open shoulder surgery, and lateralization/distalization). Second, while we required a minimum of 24-month follow-up for control patients, we experienced earlier loss to follow-up in our nerve injury group, mean (SD) 18 (10) months, which potentially limited our ability to study the true natural history of nerve injuries. This could have inherently resulted in misrepresenting their degree of nerve recovery and ultimately their functional outcomes as determined by PRO scores.

Conclusion

Nerve injury after shoulder arthroplasty is rare (3.6%) but plays a significant role in patient's recovery following shoulder replacement surgery. Axillary mononeuropathy, brachial plexopathy, and radial mononeuropathy are the most common presentations. Most patients can be managed expectantly with observation and will recover at least partial nerve function. Improvement in PROs may be delayed or ultimately remain inferior compared with those without nerve complication.

Disclaimers:

Funding: No funding was disclosed by the authors.
Conflicts of interest: The authors, their immediate families, and any research foundation with which they are affiliated have not received any financial payments or other benefits from any commercial entity related to the subject of this article.

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