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External Rotation Strength After TSA in Osteoarthritic Shoulders with Eccentric Deformity Is Not Impacted by Posterior Rotator Cuff Deficiency

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Background: Patients with persistent glenohumeral osteoarthritis symptoms despite nonoperative management may pursue anatomic total shoulder arthroplasty (TSA). TSA revision rates are higher in patients with preoperative eccentric (asymmetric posterior erosion) compared with concentric (symmetric) glenoid deformity. If posterior rotator cuff deficiency demonstrated preoperatively in patients with eccentric deformity persists after TSA, it may manifest as relative weakness in external compared with internal rotation secondary to deficient activity of the shoulder external rotator muscles. Persistent posterior rotator cuff deficiency is hypothesized to contribute to TSA failures. However, it remains unknown whether rotational strength is impaired after TSA in patients with eccentric deformity. Our goal was to determine if patients with eccentric deformity exhibit relative external rotation weakness that may be explained by posterior rotator cuff deficiency after TSA.

Methods: Patients who were >1 year after TSA for primary glenohumeral osteoarthritis and had had preoperative eccentric or concentric deformity were prospectively recruited. Torque was measured and electromyography was performed during maximal isometric contractions in 26 three-dimensional direction combinations. Relative strength in opposing directions (strength balance) and muscle activity of 6 shoulder rotators were compared between groups.

Results: The internal (+) and external (−) rotation component of strength balance did not differ in patients with eccentric (mean internal-external rotation component of strength balance: −7.6% ± 7.4%) compared with concentric deformity (−10.3% ± 6.8%) (mean difference: 2.7% [95% confidence interval (CI), −1.3% to 6.7%]; p = 0.59), suggesting no relative external rotation weakness. Infraspinatus activity was reduced in patients with eccentric (43.9% ± 10.4% of maximum voluntary contraction [MVC]) compared with concentric (51.3% ± 10.4% of MVC) deformity (mean difference: −7.4% [95% CI, −13.4% to −1.4%] of MVC; p = 0.04).

Conclusions: A relative external rotation strength deficit following TSA was not found, despite evidence of reduced infraspinatus activity, in the eccentric-deformity group. Reduced infraspinatus activity suggests that posterior rotator cuff deficiencies may persist following TSA in patients with eccentric deformities. Longitudinal study is necessary to evaluate muscle imbalance as a contributor to higher TSA failure rates.

Level of Evidence: Prognostic Level III. See Instructions for Authors for a complete description of levels of evidence.

Anatomic total shoulder arthroplasty (TSA) is a viable option when symptoms persist despite nonoperative management in patients with end-stage glenohumeral osteoarthritis. TSA outcomes vary according to whether the preoperative deformity type involves symmetric (concentric deformity; Walch A1 or A2) or asymmetric (eccentric deformity; Walch B1, B2, or B3)[1] glenoid bone wear. Patients with eccentric deformity have a higher TSA revision rate compared with the overall rate after TSA[2,3]. Deficiency of the posterior (infraspinatus and teres minor) relative to the anterior (subscapularis) rotator cuff (RC) muscles is theorized to contribute to the development of eccentric deformity and affect the outcome of surgery[4,5]. Studies have demonstrated greater intramuscular fat in the posterior RC muscles of patients with eccentric compared with concentric deformity before TSA[4,6-8]; the greater fat would impair external rotation strength[9,10] and support this theory. Persistent weakness in

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external relative to internal rotation after TSA may alter glenoid load transmission[11], leading to glenoid component loosening and TSA failure[12,13]. Additionally, although RC muscle activity changes, serving as markers of RC deficiency, have not been evaluated as a potential contributor to altered loading, they may play a role[14]. Persistent rotational weakness after TSA may result from deficient posterior RC muscle activity and affect TSA failure rates.

Despite the importance of understanding relative external (compared with internal) rotation strength following TSA, current evidence is limited. Existing studies of patients with unspecified deformity showed that subscapularis strength after TSA remains inferior to that in the contralateral shoulder[15,16] and to normative values[17]. These studies have advanced our knowledge of postoperative strength recovery, but they have limitations. First, strength was measured using 1-dimensional (1D) handheld dynamometers[15-17], which may overestimate strength as patients maximize torque in a direction of interest (e.g., internal rotation) by generating off-axis torques (e.g., adduction)[18]. Furthermore, 1D dynamometers cannot discern the contribution of rotation to functional combined motions (e.g., adduction with rotation). Prior work has demonstrated posterior RC muscle deficiency in patients with eccentric deformity before TSA[7,19], which is theorized to persist and contribute to TSA failures in this subgroup. However, postoperative strength has not been compared according to preoperative deformity types, to our knowledge, so it remains unknown whether patients with eccentric deformity exhibit relative external rotation weakness after TSA.

Therefore, our primary goal was to determine whether patients with eccentric deformity demonstrate relative external rotation weakness after TSA compared with patients with concentric deformity. Additionally, we used electromyography (EMG) to evaluate whether patients with eccentric deformity exhibit signs of posterior RC deficiency, as shown by reduced muscle activity after TSA. Determining whether deficits exist postoperatively is the first essential step in evaluating whether posterior RC deficiency is a potential contributor to TSA failure rates that requires targeted intervention.

Materials and Methods

Patients

Participants provided written informed consent for this institutional review board-approved prospective, cross-sectional study. From November 2021 to March 2022, patients >1 year after anatomic TSA for primary glenohumeral osteoarthritis performed by 1 of 2 fellowship-trained orthopaedic surgeons were recruited. Preoperative glenoid deformity was

TABLE I Participant Demographics and Preoperative Scores*

Characteristic	Preoperative Deformity†			Mean Difference (95% CI)			Chi-Square Statistic	P Value			
	Concentric (N = 18)	Eccentric (N = 18)	Control (N = 18)	Eccentric Vs. Concentric	Eccentric Vs. Control	Concentric Vs. Control		Overall	Eccentric Vs. Concentric	Eccentric Vs. Control	Concentric Vs. Control
Age† *(yr)*	67.8 ± 6.9	70.2 ± 8.1	64.1 ± 13.8	2.4 (−5.7, 10.5)	6.2 (−1.9, 14.3)	3.7 (−4.3, 11.9)		0.19			
Male gender§	10 (56%)	11 (61%)	12 (67%)				0.5	0.79			
Right hand dominance§	16 (89%)	16 (89%)	18 (100%)				2.2	0.34			
Dominant side tested§	12 (67%)	7 (39%)	8 (44%)				3.1	0.21			
Follow-up† *(mo)*	35.7 ± 24.8	48.1 ± 32.0	NA	−12.4 (−31.8, 7.0)				0.20			
BMI† *(kg/m²)*	31.3 ± 4.5	29.5 ± 6.3	26.3 ± 5.2	−1.8 (−6.1, 2.6)	3.2 (−1.1, 7.6)	5.0 (0.7, 9.3)		0.02	0.58	0.18	0.02
Penn shoulder score†											
Total	88.7 ± 12.7	89.3 ± 9.1	98.1 ± 2.4	0.6 (−6.7, 7.9)	−8.8 (−16.1, −1.4)	−9.4 (−16.7, −2.0)		<0.01	0.98	0.02	0.01
Pain subscore (0-30)	28.1 ± 2.3	28.0 ± 2.3	29.7 ± 0.8	−0.1 (−1.6, 1.5)	−1.7 (−3.2, −0.1)	−1.6 (−3.2, −0.0)		0.02	0.99	0.03	0.04
Satisfaction subscore (0-10)	8.9 ± 1.6	8.9 ± 1.7	9.4 ± 0.9	0.0 (−1.2, 1.2)	−0.4 (−1.6, 0.7)	−0.5 (−1.7, 0.7)		0.56			
Function subscore (0-60)	51.8 ± 9.3	52.4 ± 5.8	59.1 ± 2.3	0.6 (−4.5, 5.8)	−6.7 (−11.8, −1.5)	−7.3 (−12.4, −2.1)		<0.01	0.95	0.01	<0.01
Active flexion† *(°)*	123 ± 14	121 ± 11	128 ± 12	1.8 (−8.3, 11.8)	−7.0 (−17.1, 3.0)	−5.3 (−15.3, 4.8)		0.22			
Active abduction† *(°)*	132 ± 24	131 ± 19	130 ± 19	0.4 (−16.1, 17.0)	1.5 (−15.0, 18.1)	1.9 (−14.6, 18.5)		0.96			
Active external rotation† *(°)*	60 ± 12	60 ± 12	58 ± 11	−0.4 (−9.7, 8.8)	2.3 (−6.9, 11.5)	1.8 (−7.3, 11.0)		0.82			

*NA = not applicable, BMI = body mass index. †The values are given as the mean ± standard deviation or as the number with the percentage in parentheses. ‡Compared between groups with 1-way analysis of variance. §Compared between groups with a chi-square test.

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classified as eccentric or concentric with use of the Walch system[12] by a fellowship-trained orthopaedic surgeon using available imaging. The 2 surgeons contributed equal numbers of cases to each deformity (concentric and eccentric) group. One surgeon used a subscapularis tenotomy, and the other used a subscapularis peel. Both surgeons used a stemmed, non-eccentric humeral component and reamed to correct as much retroversion as possible (targeting <10°) while maintaining adequate subchondral bone. Routine intra- and postoperative radiographs were used to ensure alignment. Exclusion criteria included additional shoulder surgery, prior shoulder fracture or infection, or resting shoulder pain of >6 of 10 on a visual analog scale (to eliminate pain as a confounder). Age-matched, pain-free (0 of 10) adults who had never sought shoulder care were recruited from the community as control participants. Exclusion criteria for all groups included neurological disease, systemic inflammatory conditions, shoulder pain with cervical spine motion, prior breast cancer treatment, or active cancer. Enrolled participants underwent ultrasound imaging. A musculoskeletal radiologist confirmed RC tendon integrity, which was another inclusion requirement. Thirty-six of 105 patients who had undergone TSA and 18 of 21 control participants who were screened satisfied the criteria and participated.

Thus, the study had a total of 54 participants, with 18 in each of 3 groups: eccentric deformity, concentric deformity, and no deformity (controls) (Table I). Participants completed demographic questions and the Penn shoulder score[20]. Groups did not differ by age, gender, dominance of the tested side, duration of follow-up, or range of motion.

Three-Dimensional Strength

To test shoulder strength, the arm was fitted with a premade fiberglass cast extending from the upper arm to the wrist that held the elbow in 90° of flexion. The casted arm was fixed to a 6-degrees-of-freedom load cell (45E15A4; JR3) in 45° of elevation in the scapular plane and neutral rotation (Fig. 1). Torque and force

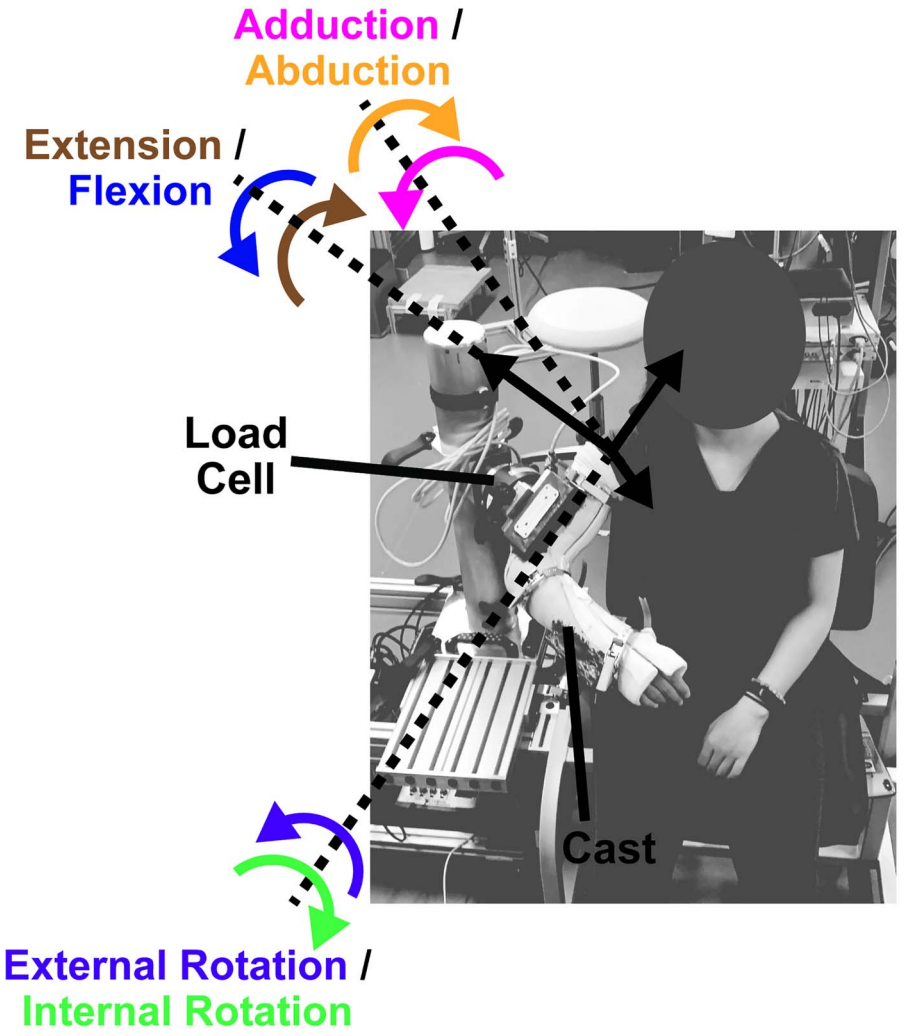

Fig. 1

Experimental setup. Participants were seated with the trunk secured by straps while they performed maximal isometric contractions. The arm was fixed to a 6-degrees-of-freedom load cell via a premade fiberglass cast.

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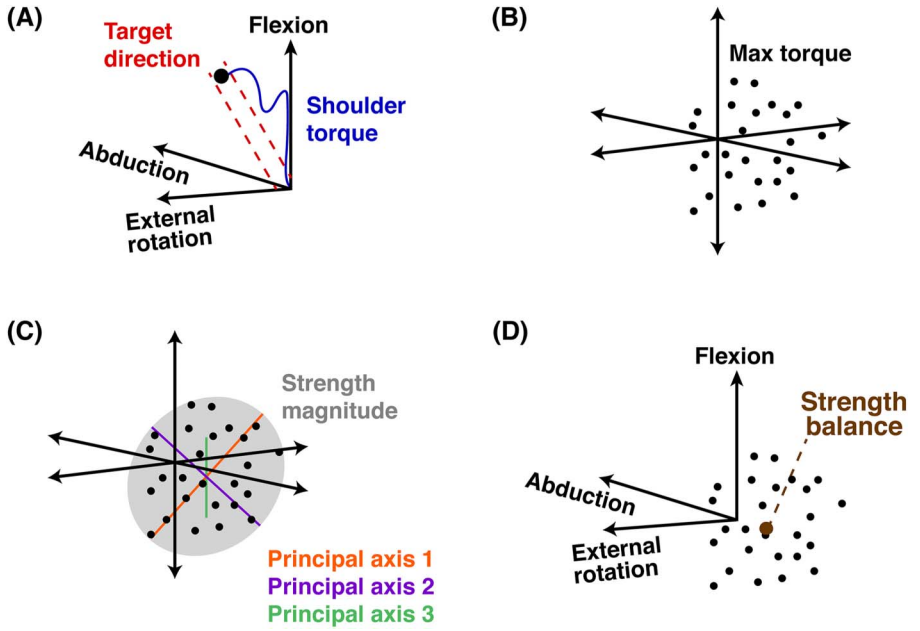

Fig. 2

Quantification of strength magnitude (SM) and 3-dimensional (3D) strength balance. **Fig. 2-A** Sample trajectory of the torque generated during a trial involving combined flexion and abduction, demonstrating the maximum torque (black dot) that was achieved in the target direction (red dotted channel). **Fig. 2-B** The points represent the maximal torque achieved in the 26 directions tested. **Fig. 2-C** Our measure of overall 3D strength (SM) was computed by first performing a principal components analysis of the 26 maxima, which yielded 3 principal axes defining the 3D space of achievable torques. The Euclidian norm of the 3 principal axis magnitudes represented the patient's overall SM (in Nm) across all 26 directions. The weight-normalized overall SM (in Nm/kg) was then calculated by dividing by the patient's weight. **Fig. 2-D** Our measure of relative strength in opposing directions (strength balance) was determined by computing the 3D center of the torque space, by first taking the vector mean of the patient's 26 maximum torques. This vector mean was then normalized by the patient's overall SM to obtain the 3D strength balance (% of unnormalized SM] along the 3 axes of interest). Strength balance along the internal-external rotation axis was of primary interest.

measurements were transformed from the load cell's coordinate system to a glenohumeral joint coordinate system[21] to determine shoulder adduction and abduction, internal and external rotation, and flexion and extension torques.

Participants performed submaximal practice trials to familiarize themselves with the visual feedback. Participants then performed 3-second maximal isometric contractions in each of 26 randomly ordered, equally spaced directions spanning the 3D space surrounding the shoulder, with 30-second breaks between trials. Directions encompassed 1D targets (e.g., external rotation) and 2D and 3D targets that combined 2 or 3 directions (e.g., flexion and adduction with or without internal rotation).

TABLE II Surface Electromyography Electrode Placement

Muscle	Action	Placement
Anterior deltoid	Internal rotation	1 finger width distal and anterior to the acromion, oriented along the line between the acromion and the thumb[23]
Pectoralis major	Internal rotation	2 finger widths below the midpoint of the clavicle, oriented along the line between the sternoclavicular joint and the anterior axillary fold[24]
Latissimus dorsi	Internal rotation	3 finger widths distal to and along the posterior axillary fold, oriented along the line between the posterior axillary fold and L3[24]
Teres major	Internal rotation	3 finger widths above the inferior angle of the scapula, along the lateral border, oriented along the line between the posterior axillary fold and inferior angle[24]
Infraspinatus	External rotation	2 to 3 finger widths below the scapular spine, at the midpoint between the posterior acromion and the trigonum spinae, oriented parallel to the scapular spine[24]
Posterior deltoid	External rotation	2 finger widths behind the angle of the acromion, oriented along the line between the acromion and the little finger[23]

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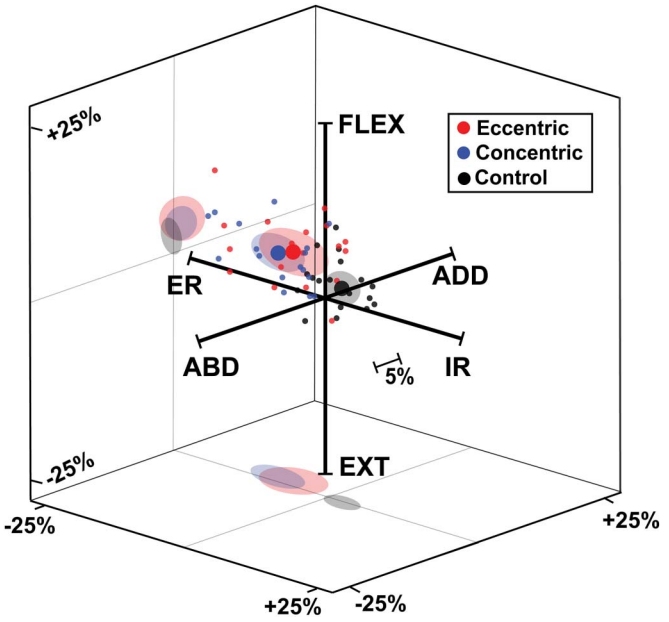

Fig. 3

Three-dimensional strength balance for each participant (smaller dots) and group means (larger dots), which did not differ between the eccentric and concentric groups. Both TSA groups demonstrated weakness in internal relative to external rotation compared with control participants. Shaded ellipses represent the 95% confidence intervals of the group means, and 2-dimensional projections of these intervals (onto the ADD/ABD-IR/ER and ADD/ABD-FLEX/EXT planes) are also shown. ADD/ABD = adduction/abduction; IR/ER = internal/external rotation; FLEX/EXT = flexion/extension.

To evaluate for relative external rotation weakness, the relative strength in opposing directions (strength balance) was quantified across all 26 directions as previously described[22]. The maximum torque achieved in each direction was identified (Figs. 2-A and 2-B). A principal components analysis of the 26 maxima yielded the magnitude (in Nm) and direction of the 3 principal axes defining the 3D space of achievable torques. The Euclidian norm of the principal axis magnitudes represented the patient's overall strength magnitude (SM) across all 26 directions (in Nm). The weight-normalized overall SM (in Nm/kg) was then calculated by dividing by the patient's weight (Fig. 2-C). Finally, the strength balance was determined by computing the 3D center of the torque space by first taking the vector mean of the patient's 26 maximum torques. This vector mean was then normalized by the patient's overall strength magnitude to obtain the 3D strength balance (% of unnormalized strength magnitude [% of SM] along the 3 axes of interest) (Fig. 2-D). Strength balance favoring external rotation (more negative internal-external rotation component) would suggest relative weakness in internal rotation. An absence of between-group differences in strength balance would suggest no relative weakness along the adduction-abduction, internal-external rotation, or flexion-extension axes. Variability in strength balance was defined by the volume of the 95% confidence interval (CI).

Muscle Activity

Artifacts from TSA implants can limit the accuracy of imaging-based quantification of posterior RC fat infiltration. Therefore, posterior RC deficiency in the patients who had undergone TSA was evaluated by using EMG to measure external and internal rotator muscle activity. Surface EMG signals during maximal contractions were recorded from the primary contributors to rotational torque production (Table II)[23,24]. Before placing electrodes (Trigno Avanti; Delsys), the skin was shaved, cleaned, and abraded (NuPrep). EMG signals from the electrodes were sampled at 2,148.1 Hz (Trigno; Delsys) and bandpass-filtered by the EMG system at 20 to 450 Hz.

EMG data were digitally bandpass-filtered at 20 to 500 Hz and then rectified. EMG data for each muscle were normalized to the maximum activity that the muscle achieved across all 26 directions (i.e., reported as a percentage of the maximum voluntary contraction [MVC]). EMG activity was determined at the time of maximal torque production for each muscle for each direction, yielding a mean EMG activity during the 1 second when the maximum torque was achieved (%MVC). Nine of the 26 directions involved internal rotation and 9 involved external rotation, with each of the 2 sets consisting of 1D rotation and 2D or 3D combinations of rotation with flexion or extension and/or with adduction or abduction. To evaluate for posterior RC deficiency, we compared muscle activity across the 9 directions involving internal rotation and across the 9 directions involving external rotation between deformity groups. EMGs from 2

TABLE III Three-Dimensional Strength Balance*

	Control (N = 18)		Concentric (N = 18)				Eccentric (N = 18)					
	Mean ± SD	95% CI of Volume	Mean ± SD	95% CI of Volume	Mean Difference from Control (95% CI)	P Value	Mean ± SD	95% CI of Volume	Mean Difference from Control (95% CI)	P Value	Mean Difference from Concentric (95% CI)	P Value
		152.0%^{3}		344.7%^{3}		<0.01		739.8%^{3}		<0.01		0.59
Add-Abd	−0.3% ± 2.6%		1.6% ± 4.0%		1.9% (0.0%, 3.8%)		1.8% ± 5.8%		2.1% (−0.4%, 4.6%)		0.2% (−2.6%, 3.0%)	
IR-ER	3.3% ± 4.3%		−10.3% ± 6.8%		−13.6% (−16.8%, −10.4%)		−7.6% ± 7.4%		−10.9% (−14.3%, −7.5%)		2.7% (−1.3%, 6.7%)	
Flex-Ext	2.3% ± 3.5%		3.8% ± 3.1%		1.4% (−0.4%, 3.3%)		4.4% ± 4.1%		2.1% (−0.1%, 4.2%)		0.6% (−1.4%, 2.7%)	

*All values are reported as the percent of the unnormalized strength magnitude. SD = standard deviation, add-abd = adduction-abduction, IR-ER = internal-external rotation, flex-ext = flexion-extension.

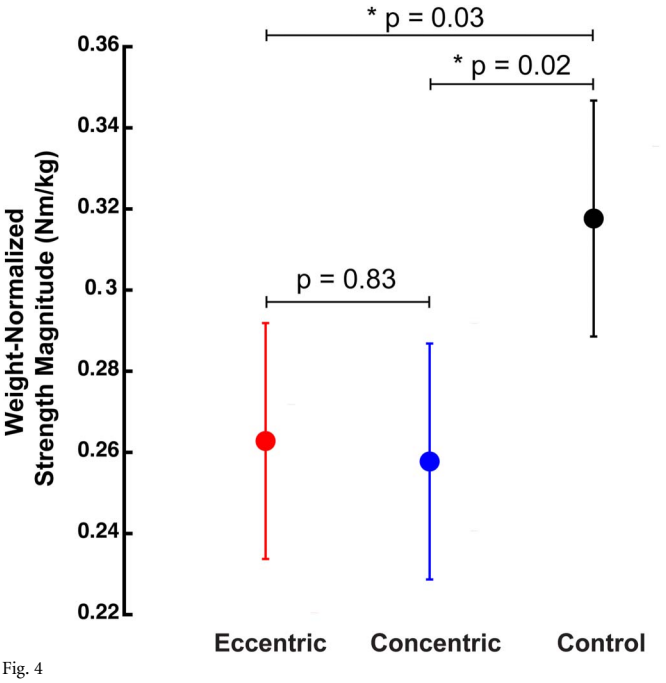

Fig. 4

The mean weight-normalized strength magnitude, and 95% CI, for each group. After TSA, there was no difference between the patients with preoperative eccentric and concentric deformity, but the magnitude was reduced by at least 17% in both groups compared with control participants.

patients (1 with eccentric deformity and 1 with concentric) were not included due to uncommon, excessive noise.

Statistical Analysis

Multivariable regression was used to test our hypothesis that patients with eccentric deformity exhibit relative external rotation weakness compared with patients with concentric deformity. Strength balance was modeled as dependent on the group and on confounding effects of age, gender, and dominance of the side tested. Between-group differences in strength balance were tested with the Hotelling t^2 statistic. In addition to that test of our primary hypothesis, we also compared strength magnitude between groups using a univariate linear model with the same confounders (age, gender, dominance of the side tested).

Linear mixed-effects models with random effects of patient and direction were used to test our hypothesis that patients with eccentric deformity demonstrate reduced posterior RC muscle activity compared with patients with concentric deformity. All direction combinations involving external rotation were used to test for between-group differences in muscle activity of the infraspinatus and posterior deltoid. A similar analysis was performed using all direction combinations involving internal rotation to test for between-group differences in internal rotator activity.

An a priori power analysis based on pilot data revealed that 18 subjects per group would be required to provide 80% power to detect between-group differences in strength balance along the internal-external rotation axis, given an anticipated effect size of 0.97. A significance level of alpha = 0.05 was used for all tests.

Source of Funding

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Results

Effects of Glenoid Deformity on 3D Strength

Patients with eccentric deformity did not demonstrate relative external rotation weakness ($p = 0.59$) compared with patients with concentric deformity (mean difference in internal-external rotation component of strength balance: 2.7% of SM [95% CI, −1.3% to 6.7% of SM]) (Fig. 3, Table III). However, patients in both deformity groups had relative internal rotation weakness ($p < 0.01$) compared with control participants (eccentric versus control: −10.9% [95% CI, −14.3% to −7.5%] of SM; concentric versus control: −13.6% [95% CI, −16.8% to −10.4%] of SM). Of note, strength balance was more variable in patients with eccentric (volume of 95% CI, 739.8% of SM^3) compared with concentric deformity (volume of 95% CI, 344.7% of SM^3) and control participants (volume of 95% CI, 152.0% of SM^3). There was no difference in overall weight-normalized strength magnitude ($p = 0.83$) between deformity groups (mean difference: 0.01 [95% CI, −0.04 to 0.05] Nm/kg) (Fig. 4). However, strength magnitude was reduced by 19% ($p = 0.02$) in the concentric compared with the control group (−0.06 [95% CI, −0.10 to −0.02] Nm/kg) and 17% ($p = 0.03$) in the eccentric compared with the control group (−0.05 [95% CI, −0.10 to −0.01] Nm/kg).

Effects of Glenoid Deformity on Muscle Activity

Activity of the internal rotator muscles did not differ between deformity groups across the directions involving internal rotation (Figs. 5-A, 5-B, and 5-C), suggesting no changes in internal rotator muscle activity in the eccentric group. Posterior deltoid activity did not differ between deformity groups across the directions involving external rotation. However, infraspinatus activity was reduced ($p = 0.04$) in the eccentric compared with the concentric group (43.9% ± 10.4% of MVC) compared with the concentric group (51.3% ± 10.4% of MVC; mean difference, −7.4% [95% CI, −13.4% to −1.4%] of MVC) (Figs. 5-B, 5-C, and 5-D). This finding supports the theory that posterior RC deficiency may exist >1 year following TSA in patients who had preoperative eccentric deformity.

Discussion

Deficiency of the posterior RC muscles relative to the anterior RC muscles is theorized to contribute to higher TSA failure rates in patients with eccentric compared with concentric deformity and has been demonstrated before TSA. We evaluated 3D strength and EMG to compare external relative to internal rotation strength and muscle activity between deformity types after TSA. No difference in strength balance, and thus no relative external rotation weakness, was observed in patients with eccentric compared with concentric deformity when controlling for gender, age, and dominance of the side tested. Yet, evidence of posterior RC deficiency with reduced normalized

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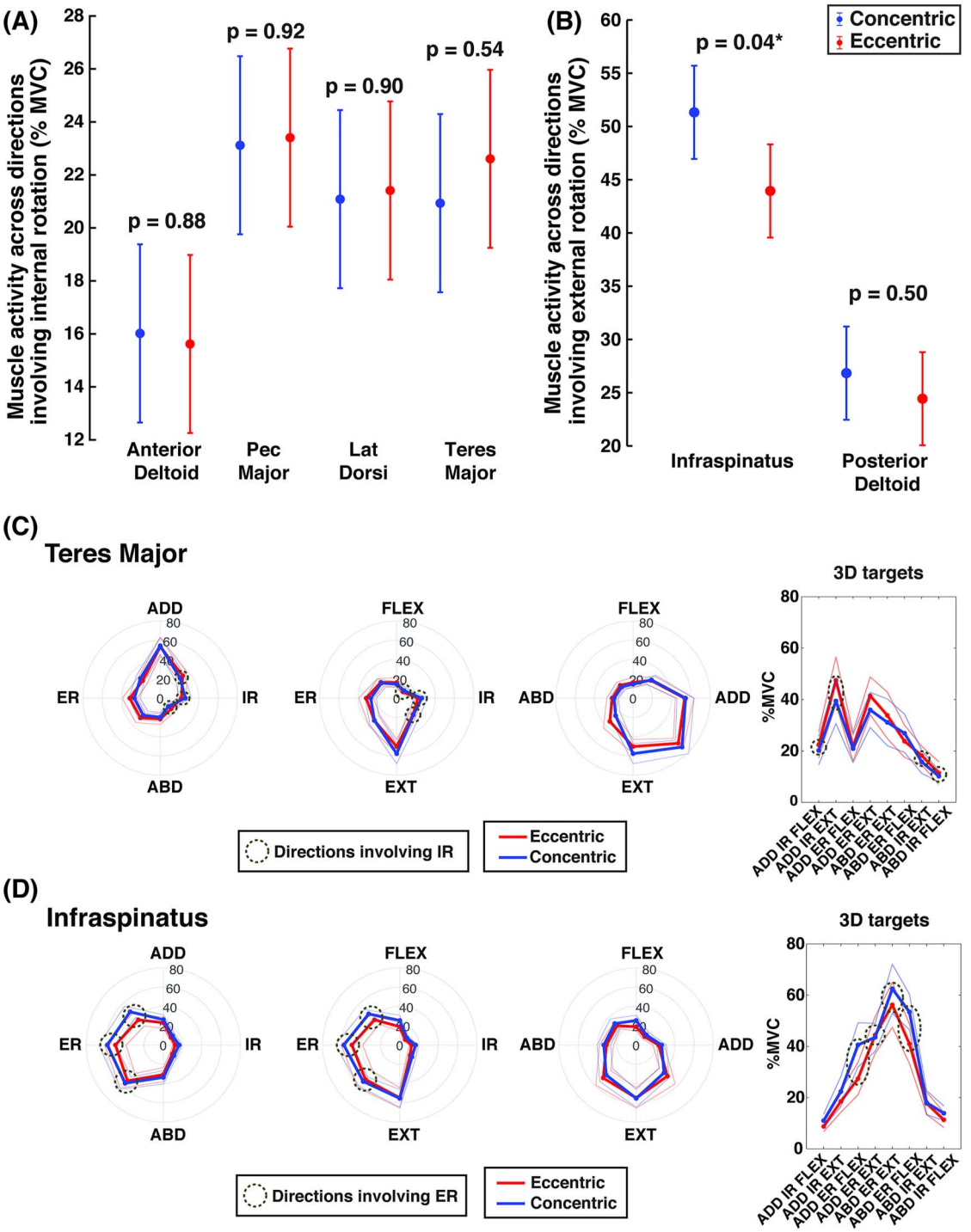

Fig. 5

Figs. 5-A and 5-B Group results for the mean muscle activity, and 95% CI, of the internal and external rotators based on linear mixed-effects models. Muscle activity was compared between the preoperative deformity groups across all 9 directions involving internal rotation for the internal rotators (**Fig. 5-A**), and across all 9 directions involving external rotation for the external rotators (**Fig. 5-B**). The only difference according to preoperative deformity was reduced muscle activity in the infraspinatus in the eccentric group. **Figs. 5-C and 5-D** Raw (un-modeled) group data for muscle activity of the teres major, an internal rotator, and the infraspinatus, an external rotator, across all 26 directions tested. Three 2D slices for each muscle demonstrate the direction combinations involving 1 or 2 directions. At the far right, the combinations involving 3 directions are shown. For the teres major, the directions involving internal rotation are designated by dashed circles or ovals. For the infraspinatus, the directions involving external rotation are designated by dashed circles or ovals. Lat = latissimus, Pec = pectoralis, MVC = maximum voluntary contraction, ADD/ABD = adduction/abduction, IR/ER = internal/external rotation, FLEX/EXT = flexion/extension.

infraspinatus activity was found in the eccentric group. This finding supports prior theories that posterior RC deficiency may exist in patients with eccentric deformity >1 year after TSA.

Effects of Glenoid Deformity on 3D Strength

Current findings agree with existing work measuring strength after TSA without considering preoperative deformity. One study measured external and internal rotation strength in the same position, allowing for consideration of relative rotational strength. Across all deformity types, patients 1 year after TSA had 42% weaker relative internal rotation compared with normative values at 45° of shoulder abduction and 90° of elbow flexion[17]. Similarly, we found that patients who had had TSA had 11% to 14% weaker relative internal rotation than control participants. The magnitude of this weakness may differ due to variations in surgical procedure, postoperative rehabilitation, and strength assessment methods[25].

Effects of Glenoid Deformity on Muscle Activity

Current results suggest that infraspinatus activity may be reduced in patients with eccentric compared with concentric deformity. Previous work identified chronic infraspinatus denervation changes in 27% of patients at least 1 year after TSA[14], regardless of preoperative deformity, and attributed these changes to surgical factors such as soft-tissue releases, retraction, or regional anesthesia. Denervation concurrent with postoperative glenoid version alterations or persistent posterior subluxation may contribute to reduced infraspinatus activity in the eccentric group.

Undiminished Relative External Rotation Strength Despite Reduced Infraspinatus Activity

External rotation strength was not diminished in patients with eccentric deformity despite reduced infraspinatus activity, perhaps due to compensation from synergistic muscles. Teres minor hypertrophy has been identified in patients undergoing reverse TSA[26,27] and was associated with lower 2-year postoperative ASES (American Shoulder and Elbow Surgeons) scores[26]. Another study suggests that there is hypertrophy of the posterior RC muscles relative to the anterior RC muscles in patients with eccentric deformity[28], which could be driven by teres minor changes. Normalized EMG activity would not reflect hypertrophy. Thus, compensation by increased force contributions from the teres minor or scapulothoracic muscles during external rotation in the eccentric group may partially explain the results of the current study.

Relative Internal Rotation Weakness After TSA

Both patient groups had relative internal rotation weakness after TSA compared with control participants, which draws attention to considerations of subscapularis management reported previously. Lapner et al. measured strength using a 1D handheld dynamometer in patients before and 24 months after TSA[15]. Internal rotation strength in the operative shoulder improved from baseline to postoperatively. However, 78% of patients did not achieve internal rotation strength that was comparable with (within 15% of) the uninvolved shoulder, suggesting that subscapularis manipulation during TSA may negatively impact strength recovery. The findings of the current study support this theory, as patients who had undergone TSA had relatively weaker internal rotation compared with control participants. While relative internal rotation weakness has been shown to exist in patients before TSA compared with control participants (range of mean difference, 2.9% to 7.6% of SM)[19], the degree of weakness was greater after TSA (range, 10.9% to 13.6% of SM). Relative subscapularis weakness after TSA may offset underlying external rotator weakness.

Strengths and Limitations

The current study had several strengths. First, we used a robust 3D method[22] to measure shoulder strength, overcoming limitations of 1D analyses and evaluating for relative external rotation weakness. Additionally, we measured RC muscle activity as a potential source of deficiency of the posterior RC muscles relative to the anterior RC muscles, which is theorized to contribute to TSA failures in patients with eccentric deformity. Supporting this theory, we found that posterior RC muscle activity may be deficient after TSA in patients with eccentric deformity, which has not been previously identified, to our knowledge. We accounted for confounders (age, gender, and dominance of the side tested) and built on prior preoperative strength and RC muscle quality results[7,19]. Finally, the current study serves as an essential step justifying the time and costs associated with longitudinal studies to evaluate how strength and RC muscle activity change from before to after TSA and whether these factors impact TSA failure rates. Elucidating mechanisms, such as intraoperative component positioning, that contribute to deficits may impact management of eccentric deformity and help prevent failures.

The conclusions drawn from the current study must be considered in the context of its limitations. First, this cross-sectional study does not show whether potential preoperative deficits in posterior RC muscle strength or activity are corrected with TSA. However, this study provides a necessary demonstration of potential posterior RC deficiency after TSA in patients with eccentric deformity. Second, strength was tested in only a single position that replicated previous isometric strength assessments following TSA. As muscle moment arms and EMG activity change with position[25], strength and muscle activity in other positions or during movement cannot be inferred. Third, although we included the major rotators, we did not make EMG recordings from the subscapularis or teres minor, as these muscles are not adequately accessed with surface electrodes and many patients had contraindications to insertion of an indwelling sensor. Cross-talk between EMG sensors or from periscapular muscles is possible. To minimize these effects, we used validated sensor positions[23,24]. The focus of the current study was the evaluation of posterior RC deficiency on the basis of rotational strength and muscle activity. As mentioned, implant artifacts can hinder evaluation of RC muscle quality after TSA; thus, we elected not to quantify postoperative fatty infiltration of the RC muscles. As they were not included in the current study, future studies should consider contributions of radiographic parameters such as corrected glenoid position, persistent posterior subluxation, or glenoid radiolucencies. Finally, we included only patients with TSA performed for primary glenohumeral osteoarthritis, so the results may not translate to TSA for fracture or other indications.

Conclusions

Relative external rotation weakness was not detected >1 year after TSA in patients who had had eccentric deformity preoperatively. Consistent with prior studies, there was relative internal rotation weakness in patients after TSA compared with controls. Despite no differences in strength, infraspinatus muscle activity was reduced in patients with preoperative eccentric compared with concentric deformity after TSA. Extending the results of studies demonstrating posterior RC deficiency preoperatively, the current study suggests potential deficiency postoperatively as well. Given the results of the current study, a longitudinal study of patients undergoing TSA is necessary to determine the extent to which underlying posterior RC muscle deficiencies may be modified following adequate surgical correction and whether these deficiencies contribute to TSA failures in patients with eccentric deformity. ■

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