Posterior Glenoid Osteotomy With Capsulolabral Repair Improves Resistance Forces in a Critical Glenoid Bone Loss Model

Stephen E. Marcaccio,* MD, Ryan M. O'Donnel,* MD, Rachel Schilkowsky,* Meng Brett D. Owens,* MD, and Steven L. Bokshan,*† MD

Investigation performed at the Department of Orthopaedic Surgery, Brown University, Warren Alpert School of Medicine, Providence, Rhode Island, USA

Background: There is no widespread consensus on the surgical treatment of posterior shoulder instability with critical posterior glenoid bone loss.

Hypothesis: That opening posterior glenoid wedge osteotomy with soft tissue repair would improve the resistance forces of instability when compared with soft tissue repair alone in the setting of 20% critical bone loss.

Study Design: Controlled laboratory study.

Methods: Native glenoid retroversion was measured on 9 shoulders using computed tomography (CT) scans. The humerus was potted in 90° of forward flexion and 30° of internal rotation relative to the scapula, and a posterior dislocation was performed to create a posterior capsulolabral injury model. The specimens were each taken through a fixed sequence of testing: (1) posteroinferior capsulolabral tear, (2) no glenoid bone loss with posteroinferior capsulolabral repair, (3) 20% posterior glenoid bone loss with posteroinferior capsulolabral repair, and (4) 20% glenoid bone loss with posterior glenoid opening wedge osteotomy and posteroinferior capsulolabral repair. Bone loss was created using a sagittal saw. The resultant peak forces with 1 cm of posterior translation were measured. A 1-way repeated-measures analysis of variance was used to compare mean force values.

Results: After the initial dislocation event, all shoulders had a resultant posterior capsulolabral injury. The resulting labral injury was extended from 6- to 9-o’clock in all specimens to homogenize the extent of injury. Repairing the capsulolabral complex in the 20% posterior glenoid bone loss group did not result in a statistically significant increase in resistance force compared with the labral deficient group (34.1 vs 22.2 N; \( P = .068 \)). When 20% posterior bone loss was created, the posterior glenoid osteotomy with capsulolabral repair was significantly stronger (43.8 N) than the posterior repair alone both with (34.1 N) and without (31.8 N) bone loss (\( P = .008 \) and .045, respectively).

Conclusion: In the setting of critical posterior glenoid bone loss, an opening wedge posterior glenoid osteotomy with capsulolabral repair improved resistance to posterior humeral translation significantly compared with capsulolabral repair alone.

Clinical Relevance: The results of this biomechanical cadaveric study may aid in surgical planning for this complex patient population.

Keywords: dislocation; glenoid; instability; osteotomy; posterior stabilizers

Posterior shoulder instability is an increasingly recognized clinical phenomenon.\(^1,3,5,24,26\) This has been shown to be particularly true in overhead athletes and military personnel, in whom the incidence has been found to be as high as 0.096 per 1000 person-years, with 5.2% of all glenohumeral instability cases being posterior.\(^3\) Contact and overhead athletes have also been shown to be at an increased risk.\(^6,9,14,15,33\) While initial treatment begins with dedicated physical therapy, active patients with continued instability are candidates for surgical intervention.\(^5,10,26\) Surgical management consists of either a soft tissue procedure, a bony procedure, or a combination of the two.\(^1,5,8,10,26\) Arthroscopic posterior capsulolabral repair has been shown to be an effective surgical treatment for recurrent posterior instability.\(^4,25,26,28\) However, failure of index surgery for posterior shoulder instability has been quoted in up to 10% of patients, with posterior glenoid bone loss of greater than 20% being a significant risk factor for failure of arthroscopic soft tissue repair.\(^10,17,22,27\)

Currently, there is no widespread consensus on the surgical treatment of critical posterior glenoid bone loss. When substantial posterior glenoid bone loss or significant...
glenoid retroversion is present, a bony glenoid procedure may be needed to fully address the underlying pathology.\textsuperscript{1,5,8,16} Posterior glenoid bone block augmentation has been described with the use of iliac crest bone graft, scapular spine autograft, and distal tibial allograft, with biomechanical analyses showing no strength difference between these alternatives.\textsuperscript{8,11,19,21,23} In addition to the posterior bone block, posterior glenoid osteotomy is another potential bony procedure that simultaneously addresses posterior glenoid bone loss and glenoid retroversion.\textsuperscript{8,13,16,18,29} Small clinical studies have shown posterior glenoid osteotomy to be a reliable procedure with low rates of clinical failure for patients with recurrent instability.\textsuperscript{12,13,16,18} However, due to higher complication rates in the setting of a technically challenging surgery, many authors have advocated for this procedure to be performed primarily by specialized surgeons at tertiary care centers.\textsuperscript{10,16,26}

As it stands, there is a paucity of literature examining the biomechanical effect of posterior glenoid opening wedge osteotomy on posterior instability, particularly in the setting of critical posterior glenoid bone loss. Therefore, the purpose of our study is to compare the biomechanical resistance force of posterior opening wedge glenoid osteotomy using a scapular spine autograft in the setting of 20\% posterior bone loss. We hypothesized that opening posterior glenoid wedge osteotomy with soft tissue repair would improve the resistance forces of instability when compared with soft tissue repair alone in the setting of 20\% critical bone loss.

**METHODS**

**Cadaveric Specimen Preparation and Mounting**

An a priori power analysis was conducted: With a power level of 0.8 and effect size $f$ of 0.5, 9 total shoulders were required for a significance level of .05. As such, 9 fresh-frozen cadaver shoulders were obtained from a local tissue bank. The mean age ($\pm$SD) was 73.2 $\pm$ 8.45 years. There were 5 male and 4 female specimens. Computed tomography (CT) scans were performed on all specimens to assess for any pre-existing glenoid bone deficiency and calculate baseline glenoid version (using the Friedman technique) and width.\textsuperscript{30} Specimens were excluded if they had pre-existing posterior glenoid bony deficiency. The skin and subcutaneous tissues were dissected sharply from the specimen with care to preserve the rotator cuff muscles and insertions. The humerus was then potted in a 2-part urethane compound (300Q, Smooth-On) (Figure 1) and attached to the actuator of an axial-torsion servohydraulic Instron testing system (model 8521; Instron). The scapula was then mounted in a vice affixed to the lower crosshead of the testing system. The humeral and scapular components were positioned such that the scapular surface was parallel to the ground, whereas the humerus was positioned relative to the scapula in 90\° of forward flexion and 30\° of internal rotation (Figure 1).

**Test Conditions**

To create a posterior instability biomechanical model, the humeral head was frankly dislocated posteriorly before testing using the Instron 8521 Axial-Torsion Servohydraulic Load Frame (Instron). Shoulder arthroscopy was then performed using a direct anterior portal to confirm the presence of a resulting posterior inferior capsulolabral injury. The resulting labral injury was extended from 6-to-9-o’clock in all specimens to homogenize the extent of injury. The 9 specimens were then each taken through a fixed sequence of testing, which included (1) posteroinferior

---

\*Address correspondence to Steven L. Bokshan, MD, Department of Orthopaedic Surgery, Brown University, Warren Alpert School of Medicine, 2 Dudley Street, Providence, RI 02905, USA (email: steven.bokshan@gmail.com).

\*Department of Orthopaedic Surgery, Brown University, Warren Alpert School of Medicine, Providence, Rhode Island, USA.

\textsuperscript{†}One or more of the authors has declared the following potential conflict of interest or source of funding: B.D.O. has received consulting fees from DePuy/ Medical Device Business Services, Linvatec, Musculoskeletal Transplant Foundation, Rotation Medical, and Verici; royalties from Linvatec and Verici; and is a paid associate editor for The American Journal of Sports Medicine. S.L.B. has received personal fees from Stryker and Zimmer Biomet. AOSSM checks author disclosures against the Open Payments Database (OPD). AOSSM has not conducted an independent investigation on the OPD and disclaims any liability or responsibility relating thereto.

Ethical approval was not sought for the present study.
capsulolabral tear (labrum deficient), (2) no glenoid bone loss with posteroinferior capsulolabral repair, (3) 20% posterior glenoid bone loss with posteroinferior capsulolabral repair, and (4) 20% glenoid bone loss with posterior glenoid opening wedge osteotomy and posteroinferior capsulolabral repair (Figure 2).

The posterior critical bone loss model was created by removing 20% of the posterior glenoid width (measured based on glenoid width from the CT scan) at a 30° angle relative to the long axis of the glenoid using a small sagittal saw (Stryker). To create a posterior wedge osteotomy, the soft tissue repair was subsequently taken apart and rerepaired following the wedge procedure. The glenohumeral joint was accessed through the posterior inferior capsulolabral injury resulting from the initial dislocation event. The posterior inferior labrum as repaired using bone tunnels and No. 2 Ethibond suture (Ethicon) with 3 separate horizontal mattress-type sutures spanning 1 cm of tissue each. These were evenly distributed at the 7-, 8-, and 9-o’clock glenoid positions.

Finally, the glenoid opening wedge osteotomy (Figure 2) was created using a triangular wedge harvested from the scapular spine. This was done by detaching the most medial 3 cm of the posterior deltoid insertion and harvesting the bone block. The interval between the infraspinatus and teres minor was opened to access the posterior glenohumeral joint. The glenoid osteotomy site was created 2.5 cm medial to the glenoid, with care taken not to violate the anterior glenoid cortex. The triangular bone block was shaped to be 1 mm at its widest width for every degree of native retroversion measured on the corresponding CT scan. To prevent displacement of the posterior wedge, a 6-hole, 2.7-mm T-plate (Smith & Nephew) was used, with 2 points of fixation minimum on each side of the osteotomy. The “T” portion of the plate was placed lateral to the osteotomy. Locking screw fixation was used to ensure the osteotomy remained in the desired position.

For every test condition, the humerus was displaced vertically 1 cm posteriorly relative to the scapula at a constant displacement rate of 1 mm/s. This resulted in a subluxation (not dislocation) of the shoulder 1 cm posteriorly. This was done twice for each test group, and the average of the 2 peak resistance forces was calculated. Load data were collected digitally from the Instron testing system (load data recorded from the machine itself) at a frequency of 100 Hz and was measured in Newtons (N).

Statistical Analysis

A 1-way repeated-measures analysis of variance was used to determine the difference in mean peak force values for each test condition. Post hoc Dunnett analyses were performed to examine significant differences among each individual group. $P = .05$ was used to determine significance for all tests (SPSS Statistics Version 21.0, IBM).

RESULTS

The mean and standard deviation of native retroversion of all shoulders was $7.3\pm 3.7^\circ$ (range, $0.3^\circ-12.7^\circ$), and the mean glenoid width was $27.2\pm 3.0$ mm (range, 22.5-30.1 mm). Following the initial dislocation event, all shoulders had a resultant posterior capsulolabral injury. This injury was contained within the 6- to 9-o’clock position and was subsequently extended for the sake of homogenization. The peak resistance force in the posterior capsulolabral deficient group was $22.2\pm 14.9$ N, with 1 cm of posterior translation (Figure 3).
Repairing the posterior capsulolabral injury resulted in a statistically significant increase in resistance force with peak force of the capsulolabral repair group averaging 31.8 ± 20.7 N (P = .047). Repairing the capsulolabral complex in the 20% posterior glenoid bone loss group (34.1 ± 31.4 N) did not result in a statistically significant increase in resistance force compared with the labral deficient group (22.2±14.9 N; P = .068). A high degree of force variability was observed in the capsulolabral repair with 20% bone loss group, with shoulders with greater than 6° of native retroversion having nearly half the average resistance forces following repair than shoulders with less than 6° of retroversion (23.6±21.7 vs 47.3±37.7 N, P = .057). When 20% posterior bone loss was created, the posterior glenoid osteotomy with capsulolabral repair was significantly higher resistant forces (43.8 ± 43.6 N) than the posterior capsulolabral repair alone with bone loss (34.1 ± 31.4 N, P = .008). Furthermore, the posterior glenoid osteotomy with capsulolabral repair showed significantly higher forces than the capsulolabral repair without bone loss (43.8 ± 31.8 N, P = .045).

DISCUSSION

In this study, we found that repairing the posterior capsulolabral complex in the 20% posterior glenoid bone loss model did not result in a statistically significant increase in resistance force compared with a labral deficient group (34.1 vs 22.2 N, P = .068). As such, labral repair alone did not restore resistance forces compared with a no bone loss group. When 20% posterior bone loss was created, the posterior glenoid osteotomy with capsulolabral repair was significantly stronger (43.8 N) than the posterior repair alone with bone loss (P = .008 and P = .045, respectively).

Posterior shoulder instability is an increasingly recognized clinical phenomenon in military personnel as well as in contact and overhead athletes.1,3,5,8,10,11,13,16,18,19,21,29 However, no consensus exists on the management of patients with recurrent instability in the setting of critical posterior glenoid bone loss, with surgical options including bony procedures such as glenoid bone block augmentation with various autograft and allograft options as well as posterior glenoid opening wedge osteotomy.4 In this study, we evaluated the biomechanical resistance force of posterior opening wedge glenoid osteotomy with scapular spine autograft in the setting of 20% posterior glenoid bone loss, particularly as it compares with the resistance force of posterior capsulolabral repair with and without bone loss.10 In this biomechanical analysis, we found that the posterior opening wedge glenoid osteotomy with capsulolabral repair had significantly higher resistance forces than the posterior capsulolabral repair alone in a critical bone loss model and was able to restore resistance force superior to the posterior capsulolabral repair without bone loss group.

In a recent study, Nacca et al22 measured the peak force required to translate the humeral head posteriorly in the setting of increasing bony defects in the posterior glenoid. In this latter study, it was found that posterior glenoid bone loss of greater than 20% required a significantly lower peak force to displace the humerus and concluded that critical posterior glenoid bone loss of 20% or greater remains unstable after capsulolabral repair alone.22 Similar to this previous work, the results of our study demonstrate that, in specimens without posterior glenoid bone loss, repair of the capsulolabral injury resulted in a statistically significant increase in resistance force when compared with the posterior capsulolabral deficient group. However, with 20% posterior glenoid bone loss present, there was no statistically significant difference between the capsulolabral repair group and capsulolabral deficiency group. This was likely explained by the high degree of variability in repair force following capsulolabral repair in the critical bone loss group. Specifically, shoulders with less than 6° of native retroversion had nearly twice the force following capsulolabral repair compared with shoulders with greater than 6° of retroversion (47.3 ± 37.7 N vs 23.6 ± 21.7 N), although this finding was only trending toward significance (P = .057). These results suggest that, in patients with critical posterior glenoid bone loss and significant retroversion, capsulolabral repair alone provides the same stability to posterior translation as a patient with a torn posterior labrum without posterior glenoid bone loss, leaving this population susceptible to further posterior instability.

Currently, there is a paucity of literature evaluating the biomechanical effect of the opening wedge posterior glenoid osteotomy on posterior instability of the shoulder, particularly in the setting of critical posterior glenoid bone loss. With regard to clinical results, Lacheta et al16 evaluated 12 patients who underwent a posterior glenoid osteotomy for persistent posterior shoulder instability in the setting of increased glenoid retroversion (defined as >10°) for a minimum of 12 months postoperatively and found a significant improvement in functional outcomes and no postoperative redislocations or revision surgeries.16 However, the complexity of the posterior glenoid osteotomy was emphasized in this study, as 4 intraoperative fractures occurred during the actual glenoid osteotomy, all which were managed without further osteosynthesis and did not affect that functional outcomes of the patients.16 While this study evaluated the opening wedge posterior glenoid osteotomy in the setting of excessive glenoid retroversion rather than critical posterior glenoid bone loss, these findings show that the procedure can lead to improved functional outcomes while highlighting the importance of leaving sufficient space between the osteotomy site and the glenoid articular surface. These results also highlight the clinical complexity that may arise in patients with significant posterior glenoid bone loss, as these patients frequently have coexisting glenoid retroversion as a result of the nonorthogonal bone loss with respect to the glenoid axis.7

Malik et al18 performed a systematic review of the literature to evaluate the rate of recurrent instability after performing a posterior glenoid osteotomy for recurrent instability and found an overall recurrence rate of 22%. However, the recurrence rate was reduced to 11% with the removal of 2 studies that were performed in the 1980s and

---

1References 1, 5, 8, 10, 11, 13, 16, 18, 19, 21, 29.
included patients with congenital instability and habitual dislocators, suggesting that more recent improvements in surgical approach and patient selection have made the opening wedge posterior glenoid osteotomy an acceptable option for carefully selected patients with recurrent posterior instability. While this represents a technically challenging procedure, our study found that, in patients with significant posterior glenoid bone loss, a posterior opening wedge glenoid osteotomy can significantly improve the biomechanical stability of the shoulder, further supporting the current literature that has demonstrated improved clinical outcomes in this patient population.

When compared with posterior bone block augmentation, the open wedge posterior glenoid osteotomy has potential advantages. Both procedures have demonstrated improved clinical outcomes for patients with recurrent posterior instability.\textsuperscript{16,18,32} Struck et al\textsuperscript{32} evaluated 15 patients in short- and long-term follow-up after undergoing posterior glenoid augmentation with scapular spine (11 patients) or distal tibia (4 patients) allograft, and found good to very good outcomes for all patients in both short- and long-term subjective and objective functional scores, with only 1 case of recurrent instability and 1 patient developing symptomatic glenohumeral arthritis. However, in a retrospective long-term study with 18-year follow-up, Meuffels et al\textsuperscript{30} reported overall poor results, with glenohumeral arthritis reported in all cases and a redislocation rate of 36%, as well as significant deterioration in clinical function when compared with the same study group when evaluated 6 months postoperatively. Further, there is emerging concern for posterior irritation caused by the prominent screws 6 months postoperatively. Further, there is emerging concern for posterior irritation caused by the prominent screws associated with bone block procedures. In a recent study, 4 of 15 patients (26%) became symptomatic from screw irritation and required surgical removal.\textsuperscript{32} Sirveaux et al\textsuperscript{31} performed screw removal in 4 of 9 patients for similar symptoms, with an additional study reporting symptomatic screw removal in 3 of 8 patients.\textsuperscript{2}

There are several limitations to this study. First, this biomechanical study utilized older cadaveric specimens, which may not represent the tissue quality found in younger patients who typically present with posterior shoulder instability. To that degree, we did not test the posterior osteotomy alone without a soft tissue repair due to the radiative nature of each test, which represents a weakness. Similarly, while a comparison with posterior glenoid bone block would have been informative, we were unable to perform such a comparison as trial testing revealed that tissue degradation prevented performing both procedures on a single specimen. Finally, another limitation of this study is that preinjury data of the uninjured specimen was not collected before the dislocation event. Because the native rotator cuff tendons were not loaded dynamically, it is possible that this model does not entirely recreate the in vivo dynamics of a real patient. Likewise, no data for load deformation were collected, which also represents a limitation of this study. With that being said, the decision to test the force of the various repair techniques with 1 cm of posterior displacement at a rate of 1 mm/s was based on a previously established method of biomechanical testing for posterior instability, the results of which are still able to yield clinically relevant information.\textsuperscript{22} Finally, the use of cadaveric specimens did not allow us to account for any potential additional stability that may be obtained with healing of the osteotomy or capsulolabral repair site. Despite these limitations, this study provides valuable biomechanical data regarding a scarcely studied subject.

CONCLUSION

In the setting of critical posterior glenoid bone loss, an opening wedge posterior glenoid osteotomy with capsulolabral repair significantly improved resistance to posterior humeral translation compared with capsulolabral repair alone.

REFERENCES

1. Antosh IJ, Tokish JM, Owens BD. Posterior shoulder instability: current surgical management. Sports Health. 2016;8:520-526. doi:10.1177/1941738116672446.
2. Barbier O, Olliat D, Marchalond JP, Versier G. Iliac bone-block autograft for posterior shoulder instability. Orthop Traumatol Surg Res. 2009;95(2):100-107. doi:10.1016/j.otsr.2008.09.008.
3. Bokshan SL, Kotchman HM, Li LT, DeFroda SF, Cameron KL, Owens BD. Incidence of posterior shoulder instability in the United States military: demographic considerations from a high-risk population. Am J Sports Med. 2021;49(2):340-345. doi:10.1177/0363546520976143.
4. Bradley JP, McClync MP, Amer JW, Tejwani SG. Arthroscopic capsulolabral reconstruction for posterior instability of the shoulder: a prospective study of 200 shoulders. Am J Sports Med. 2013;41(9):2005-2014. doi:10.1177/0363546513493599.
5. Brelin A, Dickens JF. Posterior shoulder instability. Sport Med Arthrosc Rev. 2017;25(3):136-143. doi:10.1007/978-3-662-49114-0_14.
6. Chang ES, Greco NJ, McClync MP, Bradley JP. Posterior shoulder instability in overhead athletes. Orthop Clin North Am. 2016;47(1):179-187. doi:10.1016/j.joc.2015.08.026.
7. Dekker TJ, Peebles LA, Goldenberg BT, Millett PJ, Bradford JP, Provencher MT. Location of the glenoid defect in shoulders with recurrent posterior glenohumeral instability. Am J Sports Med. 2019;47(13):3051-3056. doi:10.1177/0363546519876282.
8. DeMaris S, Bokshan SL, Nacca C, Owens BD. History of surgical stabilization for posterior shoulder instability. JSES Open Access. 2019;3(4):350-356. doi:10.1016/j.jses.2019.08.008.
9. Estes GL, Zarzour R, “HapSpeer KP. Posterior labral injuries in contact athletes. Tech Shoulder Elbow Surg. 2000;1(3):169-176. doi:10.1097/00132589-200001030-00005.
10. Frank RM, Romeo AA, Provencher MT. Posterior glenohumeral instability: evidence-based treatment. J Am Acad Orthop Surg. 2017;25(9):610-623. doi:10.5435/JAAOS-D-15-00631.
11. Frank RM, Shin J, Saccamanno MF, et al. Comparison of glenohumeral contact pressures and contact areas after posterior glenoid reconstruction with an iliac crest bone graft or distal tibial osteochondral allograft. Am J Sports Med. 2014;42(11):2574-2582. doi:10.1177/0363545414545860.
12. Graichen H, Koydl P, Zichne L. Effectiveness of glenoid osteotomy in traumatic posterior instability of the shoulder associated with excessive retroversion and flatness of the glenoid. Int Orthop. 1999;23(3):95-99.
13. Hawkins RH. Glenoid osteotomy for recurrent posterior subluxation of the shoulder: assessment by computed axial tomography. J Shoulder Elbow Surg. 1996;5(5):393-400. doi:10.1016/S1058-2746(96)00071-1.
14. Kang RW, Mahony GT, Harris TC, Dines JS. Posterior instability caused by batter’s shoulder. Clin Sports Med. 2013;32(4):797-802. doi:10.1016/j.csm.2013.07.012.
15. Kaplan LD, Flanigan DC, Norwig J, Jost P, Bradley J. Prevalence and variance of shoulder injuries in elite collegiate football players. Am J Sports Med. 2005;33(8):1142-1146. doi:10.1177/0363545605274718.

16. Lacheta L, Singh TSP, Hovsepian JM, Braun S, Imhoff AB, Pogorzelski J. Posterior open wedge glenoid osteotomy provides reliable results in young patients with increased glenoid retroversion and posterior shoulder instability. Knee Surg Sport Traumatol Arthrosc. 2019;27(1):299-304. doi:10.1007/s00167-018-5223-9.

17. Longo UG, Rizzello G, Locher J, et al. Bone loss in patients with posterior gleno-humeral instability: a systematic review. Knee Surg Sport Traumatol Arthrosc. 2016;24(2):612-617. doi:10.1007/s00167-014-3161-8.

18. Malik SS, Jordan RW, Tahir M, MacDonald PB. Does the posterior glenoid osteotomy reduce the rate of recurrence in patients with posterior shoulder instability - a systematic review. Orthop Traumatol Surg Res. 2021;107(1):102760. doi:10.1016/j.otsr.2020.102760.

19. McLaughlin HL. Posterior dislocation of the shoulder. J Bone Joint Surg Am. 1952;24 A-3(3):584-590. doi:10.2106/00004623-195234030-00011.

20. Meuffels DE, Schuit H, Van Biezen FC, Reijman M, Verhaar JAN. The posterior bone block procedure in posterior shoulder instability: a long-term follow-up study. J Bone Joint Surg B. 2010;92(5):651-655. doi:10.1302/0301-620X.92B5.23529.

21. Millett PJ, Schoenahl J-Y, Register B, Gaskill TR, van Deurzen DFP, Martetschläger F. Reconstruction of posterior glenoid deficiency using distal tibial osteoarticular allograft. Knee Surg Sport Traumatol Arthrosc. 2013;21(2):445-449. doi:10.1007/s00167-012-2254-5.

22. Nacca C, Gil JA, Badida R, Crisco JJ, Owens BD. Critical glenoid bone loss in posterior shoulder instability. Am J Sports Med. 2018;46(5):1058-1063. doi:10.1177/0363545618758015.

23. Nacca C, Gil JA, DeFroda SF, Badida R, Owens BD. Comparison of a distal tibial allograft and scapular spinal autograft for posterior shoulder instability with glenoid bone loss. Orthop J Sport Med. 2018;6(7):2325967118786697. doi:10.1097/roj.0000000000000715.

24. Owens BD, Duffey ML, Nelson BJ, DeBerardino TM, Taylor DC, Mountcastle SB. The incidence and characteristics of shoulder instability at the United States Military Academy. Am J Sports Med. 2007;35(7):1168-1173. doi:10.1177/0363545506295179.

25. Provencher MT, Bell SJ, Menzel KA, Mologne TS. Arthroscopic treatment of posterior shoulder instability: results in 33 patients. Am J Sports Med. 2005;33(10):1463-1471. doi:10.1177/0363545605278301.

26. Provencher MT, LeClere LE, King S, et al. Posterior instability of the shoulder: diagnosis and management. Am J Sports Med. 2011;39(4):874-886. doi:10.1177/0363546510384232.

27. Robinson CM, Aderinto J. Current concepts review: recurrent posterior shoulder instability. J Bone Joint Surg A. 2005;87(4):883-892. doi:10.2106/JBJS.D.02906.

28. Sanchez G, Kennedy NI, Ferrari MB, Mannava S, Frangiamore SJ, Provencher MT. Arthroscopic labral repair in the setting of recurrent posterior shoulder instability. Arthrosc Tech. 2017;6(5):e1789-e1794. doi:10.1016/j.eats.2017.06.055.

29. Scott DJ. Treatment of recurrent posterior dislocations of the shoulder by glenoplasty. Report of three cases. J Bone Joint Surg Am. 1967;49(3):471-476. doi:10.2106/00004623-196749030-00005.

30. Sharifi A, Siebert MJ, Chhabra A. How to measure glenoid bone stock and version and why it is important: a practical guide. RadioGraphics. 2020;40(6):1671-1683. doi:10.1148/rg.2020200008.

31. Sirveaux F, Leroux J, Roche O, Gosselin O, De Gasperi M, Molé D. Traitement de l’instabilité postérieure de l’épaule par butée iliaque ou acromiale. Rev Chir Orthop Reparatrice Appar Mot. 2004;90(5):411-419. doi:10.1016/S0035-1040(04)70167-1.

32. Struck M, Wellmann M, Becher C, Pastor MF, Smith T. Results of an open posterior bone block procedure for recurrent posterior shoulder instability after a short- and long-time follow-up. Knee Surg Sport Traumatol Arthrosc. 2016;24(2):618-624. doi:10.1007/s00167-014-3495-2.

33. Wanich T, Dines J, Dines D, Gambardella RA, Yocum LA. “Batter’s shoulder”: can athletes return to play at the same level after operative treatment? Clin Orthop Relat Res. 2012;470(6):1565-1570. doi:10.1007/s11999-012-2264-0.