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Differences in glenohumeral joint morphology between patients with anterior shoulder instability and healthy, uninjured volunteers

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Background: Traumatic glenohumeral joint (GHJ) dislocations are common, resulting in significant shoulder disability and pain. Previous research indicates that bony morphology is associated with an increased risk of injury in other joints (eg, the knee), but the extent to which bony morphology is associated with traumatic GHJ dislocation is unknown. This study assessed GHJ morphology in patients with anterior GHJ instability and in a control population of healthy volunteers.

Methods: Bilateral computed tomography scans were used to measure GHJ morphology in both shoulders of 11 patients with instability and 11 control subjects. Specific outcome measures included the glenoid radius of curvature (ROC) in the anterior/posterior (A/P) and superior/inferior (S/I) directions, humeral head ROC, A/P and S/I conformity index, and A/P and S/I stability angle.

Results: Compared with the control subjects, the glenoid of the instability the injured shoulder in patients with instability was flatter (ie, higher ROC) in the A/P ($P = .001$) and S/I ($P = .01$) directions and this finding was also true for uninjured, contralateral shoulder (A/P: $P = .01$, S/I: $P = .03$). No differences in GHJ morphology were detected between the instability patients' injured and contralateral shoulders ($P > .07$). Similarly, no differences in GHJ morphology were detected between the control subjects' dominant and nondominant shoulders ($P > .51$).

Conclusions: There are significant differences in GHJ morphology between healthy control subjects and both shoulders (injured and uninjured, contralateral) of patients diagnosed with anterior instability after GHJ dislocation. These findings are important clinically because they suggest that glenoid morphology may influence the risk of GHJ dislocation.

Level of evidence: Level II, Retrospective Design, Prognosis Study.

Keywords: Glenohumeral instability; anterior instability; bony morphology; glenoid morphology; humeral head morphology; radius of curvature; traumatic instability

Normal, healthy shoulder function has been previously described as the ''perfect compromise between mobility and stability.''[31] Mobility of the shoulder is accomplished primarily at the glenohumeral joint (GHJ), whereas GHJ

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stability is achieved through a complex interaction of anatomic structures that passively or actively stabilize the joint. The anatomic structures providing passive stability include the geometry of the articulating surfaces and subchondral bone, the labrum, the glenohumeral ligaments, and passive tension of the surrounding muscle groups. Dynamic stability is provided through the active contraction of muscles crossing the GHJ. The individual contributions of the various stabilizing structures have been studied extensively.[4,5,8,13,16,17] It is generally understood that the geometry of the articulating surfaces and subchondral bone have only a modest effect on stability[24,27] but that the labrum provides additional stability by increasing the depth of the glenoid.[12,14,22] The glenohumeral ligaments provide stability at end ranges of motion, and the rotator cuff muscles provide dynamic stability in the mid ranges of motion when glenohumeral ligaments are lax.[8,16,17]

Unfortunately, it is not uncommon for the delicate balance between mobility and stability to be upset because dislocation of the GHJ accounts for approximately half of all major joint dislocations each year.[1] Although a small portion of these dislocations occur chronically in the absence of trauma, approximately 80% to 90% of all GHJ dislocations occur as a result of trauma, such as falling on an outstretched arm.[26] These traumatic dislocations often damage the soft tissues that help stabilize the joint, with injury to any of these stabilizing structures (most often the labrum) leading to an increased risk of recurrent dislocation or subluxation. Traumatic dislocations are also often associated with bony defects in the glenoid or humerus, or both, with glenoid defects being reported in 5% to 56% of traumatic anterior instability cases.[15] Recurrent instability is one of the most common pathologic conditions of the shoulder and results in pain, decreased activity levels, and decreased quality of life.[18]

Whether some individuals are at increased risk for GHJ dislocation is not known, but previous research has hypothesized that glenoid morphology—specifically, having a shallow glenoid—may predispose individuals to atraumatic GHJ dislocation.[32] This hypothesis is consistent with previous research that has shown that bony morphology can influence the likelihood of traumatic injury in other major joints. For example, morphologic features of the femoral notch and the tibial slope have been identified as risk factors for anterior cruciate ligament rupture in the knee.[2,3,19,33] In the shoulder, previous research has shown that GHJ morphology can vary significantly between individuals. For example, an anatomic study by McPherson et al[20] suggested that 95% of 93 cadaveric shoulder specimens analyzed had a glenoid radius of curvature (ROC) ranging from 17 mm to 47 mm. Given this high anatomic variability, it is plausible that GHJ morphology may influence the likelihood of an individual sustaining a joint dislocation in response to trauma.

The extent to which bony morphology is associated with traumatic GHJ instability events is unknown. Therefore, the primary objective of this study was to assess GHJ morphology in 2 populations: (1) patients diagnosed with anterior instability as a result of a traumatic GHJ instability event and (2) volunteers with normal shoulder function. We hypothesized that the injured shoulders of patients with instability would have a flatter glenoid in the anterior/posterior (A/P) direction than the shoulders of healthy volunteers. As a secondary objective, we also assessed differences in GHJ morphology between the injured and contralateral shoulders of the instability patients and between the dominant and nondominant shoulders of healthy volunteers.

Materials and methods

The study enrolled 22 individuals who provided informed consent. Eleven were patients (2 female, 9 male; age, 20.5 $\pm$ 4.9 years) who had been diagnosed with at least 1 acute anterior GHJ instability event before enrolling in the study. As defined previously by Owens et al,[25] this included dislocations requiring manual reduction and subluxations. Each patient had experienced an average of 0.9 dislocations that required manual reduction (range, 0-3), with an average of 1.5 subluxations (range, 0-2). To participate in the study, these patients were required to have a positive apprehension test, no more than 3 instability events in the year before the diagnosis, bony defects estimated to be less than 5% of the glenoid width, and an asymptomatic contralateral shoulder. The size of any bony defect was measured using the technique previously reported by Sugaya et al,[28] with 9 of the 11 patients exhibiting no bony defect and the remaining 2 patients with bony defects measured at 5% of the glenoid width. Also enrolled in the study were 11 healthy volunteers (1 woman, 10 men; age, 27.0 $\pm$ 4.2 years) with no history of shoulder injury, surgery, or symptoms.

GHJ morphology was measured from computed tomography (CT) scans of the humerus and scapula. CT scans of the entire humerus and scapula were acquired (LightSpeed 16; GE Medical Systems, Piscataway, NJ, USA) in both shoulders of all instability patients and healthy volunteers. The scans had a slice thickness of 1.25 mm and an in-plane resolution of approximately 0.5 mm/pixel. The humerus and scapula were manually segmented from other bones and soft tissue and reconstructed into 3-dimensional (3D) bone models using Mimics 10.1 software (Materialise, Leuven, Belgium).

Custom software was used to measure GHJ morphology from the 3D bone models. The first step involved establishing a glenoid coordinate system. To accomplish this, a glenoid region of interest (ROI) was manually identified as all points in the 3D bone model comprising the glenoid surface. The software then automatically defined a glenoid contour line (ie, those points within the ROI that defined the outer border of the glenoid) and calculated the glenoid coordinate system origin as the average of the glenoid contour points. Next, the software determined the plane of the glenoid. This was accomplished by fitting a plane through the glenoid coordinate system origin that minimized the sum of squares distance between the plane and the glenoid contour line points. Finally, the glenoid contour line points were projected onto the

glenoid plane, and the superior/inferior (S/I) axis was positioned such that the anterior/superior quadrant of the glenoid had the same projection area as the posterior/superior quadrant. The anterior/posterior (A/P) axis was defined as perpendicular to the S/I axis in the glenoid plane, and the medial/lateral axis was perpendicular to the glenoid plane.

To calculate the glenoid's ROC in the S/I direction, the software first established a plane that was perpendicular to the glenoid plane and contained the S/I axis. The software then searched this S/I-aligned plane to find the center point and the radius of a circle that minimized the sum of squares error between the circle and the points in the glenoid bone model that intersected the S/I-aligned plane (Fig. 1). This process was repeated in an A/P-aligned plane to determine the ROC in the A/P direction (Fig. 1).

A similar approach was used for determining the humeral head ROC. Briefly, a ROI was manually identified to include the points in the bone model that comprised the humeral head surface. Next, the custom software searched the interior of this ROI to find the center point and radius of a sphere that minimized the sum of squares error between the spherical surface and the humeral head ROI.

To assess the accuracy of this technique, ROC values calculated from conventional CT images of a cadaveric shoulder specimen were compared with curvature values computed from micro-CT images of the same shoulder specimen after cartilage removal. The ROC measurements determined from the conventional CT images were within 4% of the micro-CT–based measures of curvature. The specific outcome measures used in this study included glenoid height, glenoid width, glenoid height-to-width ratio, glenoid version, the glenoid ROC in the A/P and S/I directions, the humeral head ROC, the glenohumeral conformity index (humeral head ROC/glenoid ROC) in the A/P and S/I directions, and the glenohumeral stability angle (the angle of the humeral head enclosed by the glenoid[13]) in the A/P and S/I directions. Paired t tests were used to assess differences in morphologic parameters between the healthy volunteers' dominant and nondominant shoulders and the instability between patients' injured and contralateral shoulders. Unpaired t tests were used to assess differences between the healthy volunteers' dominant shoulders and instability patients' injured and contralateral shoulders. Significance was set at $P \leq .05$ for all statistical tests.

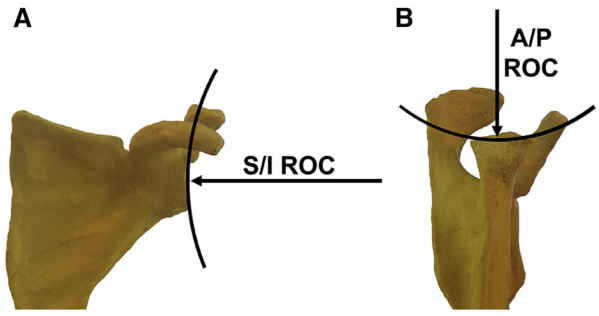

Figure 1 Glenoid radius of curvature (*ROC*) was determined in the (**A**) superior/inferior (*S/I*) and (**B**) anterior/posterior (*A/P*) directions by fitting a circle to the points in a computed tomography–based bone model along the A/P and S/I axes of a glenoid-based coordinate system.

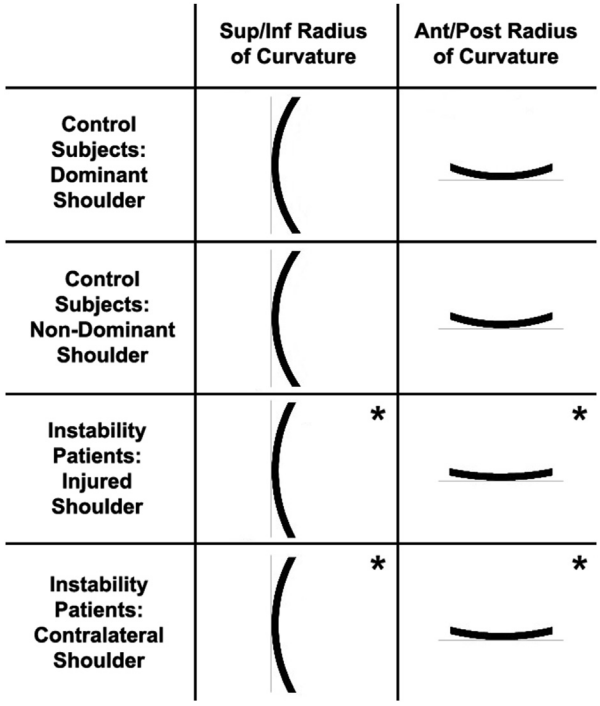

Figure 2 Graphic representation of the mean glenoid radii of curvature in the superior/inferior (*Sup/Inf*) (S/I) and anterior/posterior (*Ant/Post*) (A/P) directions for the 2 groups. No significant differences in S/I radius of curvature or A/P radius of curvature were detected between the control subjects' dominant and nondominant shoulders or between the instability patients' injured and contralateral shoulders. However, the instability patients' injured and contralateral shoulders were both significantly flatter than the control subjects' dominant shoulder in both the S/I ($P < .03$) and A/P direction ($P < .01$). *$P < .05$ compared with control subjects' dominant shoulders.

Results

No significant differences were detected between the healthy volunteers' dominant and nondominant shoulders in any GHJ morphology parameters. Specifically, no significant differences were found in glenoid height ($P = .96$), glenoid width ($P = .73$), glenoid height-to-width ratio ($P = .73$), glenoid version ($P = .27$), and humeral head ROC ($P = .92$). In the S/I direction, no differences were detected in the ROC ($P = .70$, Fig. 2), conformity index ($P = .74$), or stability angle ($P = .66$, Table I). Furthermore, no significant differences were detected between healthy volunteers' dominant and nondominant shoulders in the A/P ROC ($P = .51$, Fig. 2), A/P conformity index ($P = .72$), or A/P stability angle ($P = .69$, Table I).

Similar to the control subjects, no significant differences were detected in GHJ morphology parameters between the instability patients' injured and contralateral shoulders (Table I). Again, there were no significant differences in glenoid height ($P = .60$), glenoid width ($P = .33$), glenoid height-to-width ratio ($P = .12$), glenoid version ($P = .81$), humeral head ROC ($P = .78$), S/I ROC ($P = .62$, Fig. 2), S/I

Table I Measures of glenohumeral joint morphology in control subjects and instability patients*

Outcome measure	Control subjects		Instability patients	
	Dominant	Nondominant	Injured	Contralateral
	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD
HH ROC, mm	23.3 ± 2.0	23.3 ± 2.3	25.2 ± 2.7	25.3 ± 2.7
Superior/inferior				
ROC, mm	35.3 ± 4.1	35.0 ± 3.9	43.2 ± 8.4†	42.1 ± 8.7†
Conformity index	0.67 ± 0.1	0.67 ± 0.1	0.60 ± 0.1	0.62 ± 0.1
Stability angle,°	56.9 ± 7.6	57.5 ± 8.1	49.8 ± 9.6	50.5 ± 8.8
Anterior/posterior				
ROC, mm	42.7 ± 6.0	45.5 ± 11.2	77.1 ± 27.5†	63.3 ± 21.9†
Conformity index	0.56 ± 0.1	0.54 ± 0.1	0.36 ± 0.1†	0.44 ± 0.1†
Stability angle,°	35.7 ± 6.33	34.6 ± 8.7	20.0 ± 6.5†	25.1 ± 8.3†
Glenoid				
Height, mm	33.3 ± 3.5	33.2 ± 3.1	35.3 ± 3.5	35.0 ± 4.4
Width, mm	25.3 ± 2.9	25.2 ± 2.5	24.2 ± 1.7	24.9 ± 3.1
Height-to-width ratio	1.31 ± 0.1	1.32 ± 0.1	1.46 ± 0.1†	1.41 ± 0.1†
Glenoid version,°	6.4 ± 4.7	5.3 ± 5.0	4.9 ± 5.8	4.6 ± 6.4

HH, humeral head; *ROC*, radius of curvature; *SD*, standard deviation.

* No significant differences were detected between the control subjects' dominant and nondominant shoulders or between the instability patients' injured and contralateral shoulders.

† $P < .05$ compared with control subjects' dominant shoulders.

conformity index ($P = .56$), or S/I stability angle ($P = .67$, Table I). In addition, no significant differences were detected between shoulders in the instability patients in the A/P ROC ($P = .21$, Fig. 2), A/P conformity index ($P = .07$), or A/P stability angle ($P = .69$, Table I).

Because no statistically significant differences were detected between the healthy volunteers' dominant and nondominant shoulders, we used the healthy volunteers' dominant shoulders for comparison with the instability patients' injured and contralateral shoulders. This comparison showed the instability patients' injured shoulders had a significantly greater glenoid height-to-width ratio ($P = .003$), S/I ROC ($P = .01$, Fig. 2), A/P ROC ($P = .001$, Fig. 2), lower A/P conformity index ($P < .001$), and lower A/P stability angle ($P < .001$, Table I). However, no significant differences were detected between the healthy volunteers' dominant shoulders and the instability patients' injured shoulders in glenoid height ($P = .18$), glenoid width ($P = .27$), glenoid version ($P = .50$), humeral head ROC ($P = .07$), S/I conformity index ($P = .13$), or S/I stability angle ($P = .07$, Table I).

Compared with healthy volunteers' dominant shoulders, the instability patients' uninjured contralateral shoulders had a significantly greater glenoid height-to-width ratio ($P = .01$), S/I ROC ($P = .03$, Fig. 2), greater A/P ROC ($P = .01$, Fig. 2), lower A/P conformity index ($P = .02$), and lower A/P stability angle ($P = .004$, Table I). However, no significant differences were detected between the healthy volunteers' dominant shoulders and the instability patients' contralateral shoulders in glenoid height ($P = .30$), glenoid width ($P = .72$), glenoid version ($P = .46$), humeral head ROC ($P = .06$), S/I conformity index ($P = .18$), or the S/I stability angle ($P = .08$, Table I).

Discussion

The objective of this study was to compare GHJ morphology between instability patients and healthy volunteers as well as between shoulders within each of the 2 groups. When comparing the dominant and nondominant shoulders of the healthy volunteers, we found no differences in morphology of the humeral head or glenoid, as hypothesized. Because no significant differences were detected between dominant and nondominant healthy shoulders, the instability patients' injured shoulders were compared with the healthy volunteers' dominant shoulders. This comparison indicated that the instability patients' injured shoulders had a flatter glenoid in the A/P and S/I directions compared with the healthy control subjects. These patients also exhibited a lower conformity index and stability angle in the A/P direction. Lastly, we compared the measures of bony morphology between the instability patients' injured and contralateral shoulders. Similar to the control subjects, no significant differences were detected between the injured and contralateral shoulders of the instability patients. These findings indicate that there are significant differences in GHJ morphology between patients who have sustained a traumatic GHJ dislocation and healthy volunteers but that no statistically significant side-to-side differences in GHJ morphology were detected in either group.

The values reported here for humeral head ROC and glenoid ROC of healthy volunteers are comparable to those reported previously in the literature. Von Eisenhart-Rothe et al[32] used magnetic resonance imaging to measure GHJ morphology in healthy volunteers and reported that the glenoid had an average generalized ROC (ie, a ROC not specific to the A/P or S/I directions) of 41.7 mm. This value is in good agreement with the average S/I ROC (35.3 ± 4.1 mm) and A/P ROC (42.1 ± 6.0 mm) of the control subjects' dominant shoulder reported in the current study. McPherson et al[20] calculated GHJ morphology from 2D radiographs of cadaveric shoulder specimens and reported a humeral head ROC of 23 mm, an S/I glenoid ROC of 32.2 mm, and an A/P glenoid ROC of 40.6 mm. For comparison, the values reported in the current study for these parameters, averaged across dominant and nondominant shoulders of the healthy volunteers, were 23.3 ± 2.1 mm, 35.1 ± 3.9, mm and 44.1 ± 8.9 mm, respectively.

Perhaps the most interesting finding from this study is that the instability patients have flatter glenoids (ie, higher A/P ROC and S/I ROC) than the control subjects (Table I). This is particularly interesting given that significant side-to-side differences in GHJ morphology were not detected in either group. This indicates that the instability patients had flatter, less conforming glenoids in both shoulders, which may suggest that the differences in GHJ morphology observed between the 2 groups was not a result of the instability patients' injury. Rather, these data suggest that certain individuals (ie, those with a flatter glenoid) may be at higher risk for GHJ dislocation in response to a traumatic event. We acknowledge that the relatively small sample size does not exclude the possibility that small differences in joint morphology may exist between the instability patients' injured and uninjured shoulders and that joint morphology may have been influenced by the traumatic dislocation or subluxation.

However, the statistically significant differences between the instability patients' uninjured shoulders and the control subjects' dominant shoulders provides convincing evidence that differences in joint morphology certainly exist between these 2 groups. This conjecture is further supported by a recent study by Kawasaki et al[11] who reported that a history of dislocation in one shoulder was a significant risk factor for dislocation in the contralateral shoulder. Furthermore, Owens et al[25] recently conducted a prospective cohort study to determine risk factors for traumatic anterior instability. They found that in addition to the clinical risk factors, such as the apprehension sign and relocation sign, significant anatomic risk factors existed as well. Specifically, they found the coracohumeral distance and, in accordance with our findings, the glenoid height-to-width ratio were significant risk factors for anterior instability. In addition, they did not find glenoid depth was a significant risk factor but did not assess the ROC.

Cadaveric studies have defined the contributions of the labrum[12,13,22] and glenohumeral ligaments[8,16,17] to GHJ stability, and the role of glenoid geometry on GHJ stability has been studied extensively within the context of joint arthroplasty.[9,10] Furthermore, cadaveric studies have shown that it is possible to increase GHJ conformity and stability by surgically manipulating the glenoid geometry through posteroinferior glenoplasty[21] or anteroinferior bone grafting.[23] However, the role of glenoid subchondral bone morphology on GHJ stability has not, to our knowledge, been explicitly investigated. Despite this, the measures of glenoid curvature reported here, along with those reported in previously published studies, provide indirect evidence regarding the role of glenoid bony morphology to GHJ stability. Specifically, the current study reports that the A/P ROC of the glenoid was approximately 43 to 45 mm for healthy volunteers and 63 to 77 mm for the anterior instability patients (Table I), whereas von Eisenhart-Rothe et al[32] reported a glenoid ROC of 103.8 mm for patients diagnosed with multidirectional instability. These findings suggest that there is a continuum of glenoid ROC values and that the likelihood of pathology may increase with increasing ROC values. In particular, lower glenoid ROC values imply greater conformity, higher GHJ stability, and more normal GHJ function, whereas higher ROC values imply lower conformity, lower GHJ stability, and perhaps an increased risk of traumatic or atraumatic joint dislocation, or both.

The findings from this study also suggest that the high incidence of GHJ dislocations that occur in the A/P direction—perhaps as high as 96% of all GHJ dislocations[6]—may be influenced to some extent by bony morphology. Specifically, the glenoid's A/P ROC was approximately 7 to 34 mm greater than the S/I ROC, depending on the group and shoulder (Table I), which is consistent with previous findings.[20] Thus, a higher ROC (or flatter glenoid) in the A/P direction, along with a correspondingly lower A/P conformity index and stability angle, may suggest lower GHJ A/P stability and help to at least partly explain why GHJ dislocations are most likely to occur in the A/P direction.

The study failed to detect any differences in GHJ morphology between the control subjects' dominant and nondominant shoulders. Although previous studies have reported differences in GHJ morphology between the dominant and nondominant shoulders of unique subject populations, such as baseball pitchers,[7,29,30] the findings from the current study suggest that GHJ morphology may not be significantly influenced by shoulder dominance in this specific group. In addition, the good agreement between the morphologic measures reported in the current study from young control subjects (average age, 27 years) and the data reported by McPherson et al[20] from "relatively elderly cadaver specimens" (average age was unknown) further suggests that GHJ morphology may not change appreciably over time in healthy, skeletally mature adults.

Although this study selected healthy volunteers and instability patients in anticipation of implicating associations between joint morphology and joint stability, a limitation of this study is that it did not provide a measure of joint stability. Another limitation is that these data were collected on instability patients after they had sustained a dislocation event, and to what extent GHJ morphology was altered as a result of this injury is unknown. However, patients were excluded from participating in the study if they had a bony defect greater than 5% of the glenoid width. Furthermore, the identification of a similarly shaped glenoid on the instability patients' uninjured contralateral shoulder suggests that the differences in bony morphology were not caused by the dislocation event.

The relatively small sample size is a potential limitation of the study, in that it is difficult to exclude the possibility that the study was statistically underpowered for the comparisons between shoulders within each group. In fact, a post hoc power analysis suggests that a sample size of approximately 74 to 128 subjects would be necessary to detect differences in A/P and S/I glenoid ROC between the control subjects' dominant and nondominant shoulders. However, the primary objective of this study was to assess differences in GHJ morphology between the instability patients and the healthy control subjects, and differences were indeed detected in our sample. In addition, the method of morphology measurement was shown to be accurate within 4% of micro-CT measures, and although this difference is small, it still limits the ability to determine if small differences exist between groups. Lastly, we acknowledge that the contributions of the glenoid labrum and cartilage are important. Unfortunately, visualizing soft tissues in a CT scan is not possible without the use of contrast agents, and this is a limitation of our approach.

Conclusions

This study has demonstrated that there are significant differences in GHJ morphology between healthy control subjects and patients with documented traumatic glenohumeral instability events. Specifically, the instability patients have flatter glenoids than the control subjects in the A/P and S/I directions. In addition, the study failed to detect differences in GHJ morphology between the control subjects' dominant and nondominant shoulders and between the instability patients' injured and contralateral shoulders. These findings are important clinically because the results suggest that glenoid morphology may influence the risk of GHJ dislocation due to the finding that both the instability patients' injured and uninjured shoulders showed significant differences in morphology compared with healthy controls. Future research efforts will continue to explore this complex relationship between joint morphology, joint mechanics, and injury.

Disclaimer

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