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## Acromial and glenoid morphology in glenohumeral osteoarthritis: a three-dimensional analysis

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**Background:** The purpose of this study was to determine the association between glenohumeral osteoarthritis (GHOA) and three-dimensional acromial and glenoid morphology.

**Methods:** In this retrospective study, we compared computed tomographic studies of three groups of scapulae: normal healthy, mild GHOA (Samilson-Prieto grade 1), and severe GHOA (Samilson-Prieto grade 3). All scans were segmented to create three-dimensional reconstructions. From these models, critical shoulder angle and acromial offset were measured, as normalized to scapular height. The coronal plane inclination of the glenoid was measured using a glenoid sphere-fit method. Reliability was confirmed via intraclass correlation coefficients > 0.75.

**Results:** Eighty scapulae were included: 30 normal, 20 mild GHOA, and 30 severe GHOA. There were no differences in acromial offset between the normal group and either the mild-GHOA group or the severe-GHOA group. The severe-GHOA group had a smaller critical shoulder angle than either the normal ( $30 \pm 5^\circ$  vs.  $34 \pm 4^\circ$ ,  $P = .003$ ) or mild-GHOA groups ( $34 \pm 4^\circ$ ,  $P = .020$ ), but the normal and mild-GHOA groups did not differ ( $P = .965$ ). The severe-GHOA group had more inferiorly inclined glenoids than either the normal ( $7 \pm 6^\circ$  vs.  $12 \pm 5^\circ$ ,  $P = .002$ ) or mild-GHOA groups ( $14 \pm 5^\circ$ ,  $P \leq .001$ ), but the normal and mild-GHOA groups did not differ ( $P = .281$ ).

**Conclusion:** Normal and severe-GHOA shoulders differ in critical shoulder angle and glenoid inclination but not acromial offset. The lack of a difference in critical shoulder angle or inferior inclination between mild-GHOA and normal groups calls into question whether inclination and critical shoulder angle differences predate severe GHOA.

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The incidence of glenohumeral (GH) osteoarthritis (OA) in those 70–80 years of age is >15%,<sup>15,22</sup> and the incidence is increasing.<sup>8,17</sup> GHOA causes substantial pain and disability,<sup>19</sup> with a similar decrease in overall health status to congestive heart failure and a greater decrease than myocardial infarction or clinical depression.<sup>13</sup> The pathogenesis of GHOA remains unknown. OA progression

Each author certifies that his or her institution approved the human protocol for this investigation, that all investigations were conducted in conformity with ethical principles of research, and that informed consent for participation in the study was not required by our institutional review board.

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likely involves a complex interaction between genetic predisposition and altered biomechanics, synovium, cartilage, muscle, and bone.<sup>1,7,10,16,18,20,24,26,28,29</sup> There are no current treatment options to prevent or slow OA progression.

Prior research has suggested that the critical shoulder angle differs in scapulae with and without OA.<sup>4,5,21,26,27,32</sup> The critical shoulder angle measurement incorporates contributions from both glenoid inclination and the extent of the lateral protrusion of the acromion in the coronal-plane.<sup>6,21</sup> Both increased superior inclination and a more lateral acromion relative to the glenoid has been theorized to increase the compressive force and decrease the shear force on the glenoid as generated by the deltoid.<sup>14</sup> It is unclear which of these factors is the primary driver, or if they contribute equally, as there are few prior studies to examine both concepts.<sup>3</sup> Furthermore, many prior anatomic comparisons have been between patients with GHOA and rotator cuff tears, and it remains

unclear whether one or both of these pathologies is related to the anatomic difference.<sup>2</sup> It is also unclear whether the differences in critical shoulder angle are present in individuals with mild GHOA, before progression to severe GHOA. Severe GHOA may be associated with glenoid wear which directly influences glenoid inclination and may thus be a consequence, and not cause, of GHOA. Glenoid wear also creates medialization of the glenoid fossa and thus shortens the length of the supraspinatus, infraspinatus, and subscapularis fossae. A three-dimensional (3D) analysis is helpful both to examine these topics and to better characterize the entirety of the lateral aspect of the acromion, as compared with just the most lateral acromial point, which may not truly reflect the composite force generated by the deltoid.

Thus, the purpose of this study was to determine the association between GHOA and 3D acromial and glenoid morphology. We hypothesized that both mild and severe GHOA would be associated with a smaller critical shoulder angle, increased inferior inclination, and a decreased distance between the glenoid center and the lateral acromial edge.

## Methods

### Patient groups

This was a retrospective radiographic study. We collected data from three groups of shoulders: (1) normal healthy shoulders, (2) shoulders with mild GHOA, and (3) shoulders with severe GHOA. Because GHOA is more common in the elderly,<sup>22</sup> we specifically examined shoulders of similar ages (50–65 years of age). Because the measurements required the entirety of the scapula and its 3D osseous morphology, we only included shoulders with computed tomographic (CT) scans of the complete scapula that could be exported from the medical record and imported into the 3D analysis software. The groups were binned based on the Samilson-Prieto classification,<sup>25</sup> which is based on osteophyte size, with no osteophytes being a normal healthy shoulder, <3 mm osteophyte size (Samilson-Prieto grade 1) being mild GHOA, and >7 mm osteophyte size (Samilson-Prieto grade 3) being severe GHOA. This system has previously been demonstrated to have excellent reliability on plain radiographs.<sup>11</sup> However, we additionally tested the reliability of the Samilson-Prieto staging system here. Two orthopedic shoulder surgeons graded 51 scans blinded to each other's grades, and the kappa statistic was calculated. Only 1 shoulder per subject was included if bilateral scans were available, with the laterality selected randomly. For each included shoulder, the following information was collected: age, side of scan, sex, height, and weight.

### Normal group

Scans from two subgroups were included in this group: cadaver shoulders and living subjects. Our laboratory has previously obtained CT scans on all cadaver shoulders used in laboratory experiments. These scans were reviewed, along with the records from the individual experiments where each shoulder underwent a dissection, and those shoulders with evidence of either OA or a rotator cuff tear on imaging or gross dissection were excluded. Our laboratory has also recruited control subjects with no history of shoulder injuries, surgery, or shoulder pain for other studies. These deidentified scans were also available and reviewed. An attending surgeon with fellowship training in shoulder and elbow surgery (P.N.C.) reviewed all scans to exclude any subjects with radiographic abnormalities. Only shoulders older than the age of 50 years were included.

### Mild-GHOA group

The cadaver scan archive was also reviewed to identify those with mild GHOA (<3 mm osteophytes, Samilson-Prieto grade 1). We excluded shoulders with rotator cuff tears, and those over the age of 65.

### Severe-GHOA group

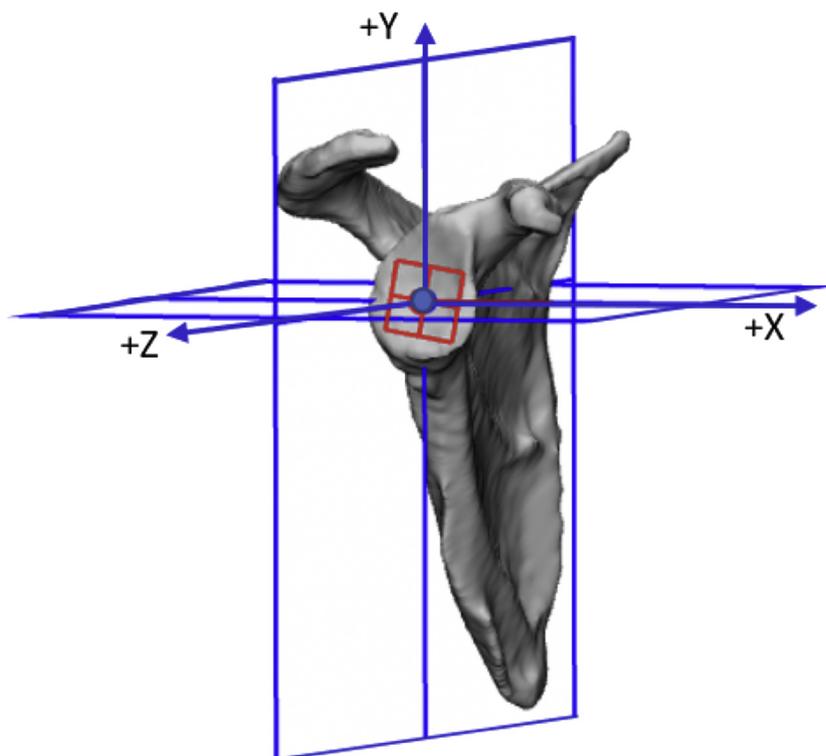
We searched our medical record for all patients who underwent an anatomic total shoulder arthroplasty (CPT code 23472) between January 2009 and March of 2018 and those with a preoperative CT scan were identified. Exclusion criteria included a diagnosis other than OA, patients found intraoperatively to have a rotator cuff tear, and patients with CT scan that could not be exported from our medical records. The imaging for each of these patients was then reviewed and was only included if the patients had severe GHOA (osteophyte size >7 mm, Samilson-Prieto grade 3). We again excluded shoulders older than the age of 65 years.

### Measurement protocol

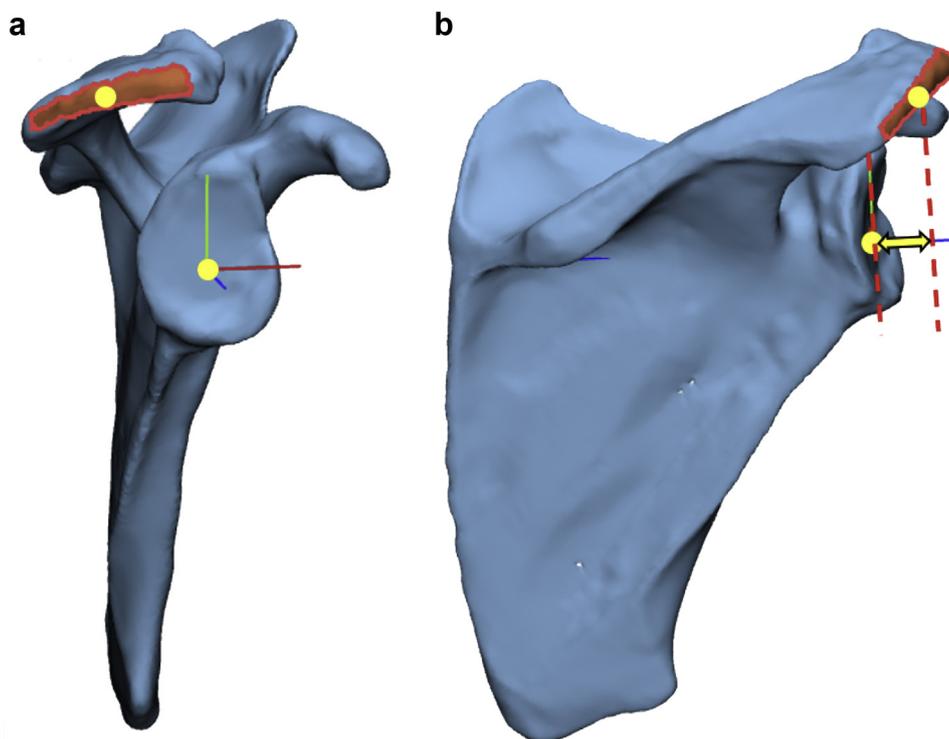
All measurements were performed in 3D analysis software (Mimics and 3matic; Materialise, Leuven, Belgium and Horos [horosproject.org]). Plain radiographs were not available, and thus, the critical shoulder angle was measured as previously described on CT slices reoriented into a plane that contained the superior pole of the glenoid, the inferior pole of the glenoid, and the lateralmost point on the acromion. This method has been previously described to nearly perfectly replicate measurements made radiographically.<sup>6</sup> Each CT scan Digital Imaging and Communications in Medicine file was imported and reconstructed into a 3D surface. This reconstruction process used a combination of semiautomatic and manual segmentation to ensure reconstruction quality followed the identifiable cortical boundaries. These surfaces were then wrapped and smoothed to correct any surface defects owing to the segmentation process. All subsequent measurements were made on these reconstructions.

Each scapula was reoriented to a global coordinate system. We identified the center of the glenoid, defined as the center of an arc fit to the lower, circular portion of the rim of the glenoid.<sup>30,31</sup> A line was then drawn between the center of this arc and the trigonum spinae, where its intersection at the osseous surface of the glenoid was defined as the center of the glenoid. Next, we reoriented the coordinate system relative to the scapula using the center of the glenoid, the trigonum spinae, and the inferior-most portion of the inferior angle to define the scapular plane (coronal). The axial plane was defined perpendicular to the scapular plane, intersecting both the center of the glenoid and the trigonum spinae. The sagittal plane was perpendicular to both the axial and scapular planes. Anterior (X), superior (Y), and lateral (Z) were all considered as positive values relative to the sagittal (XY), coronal (YZ), and axial (XZ) planes (Fig. 1).

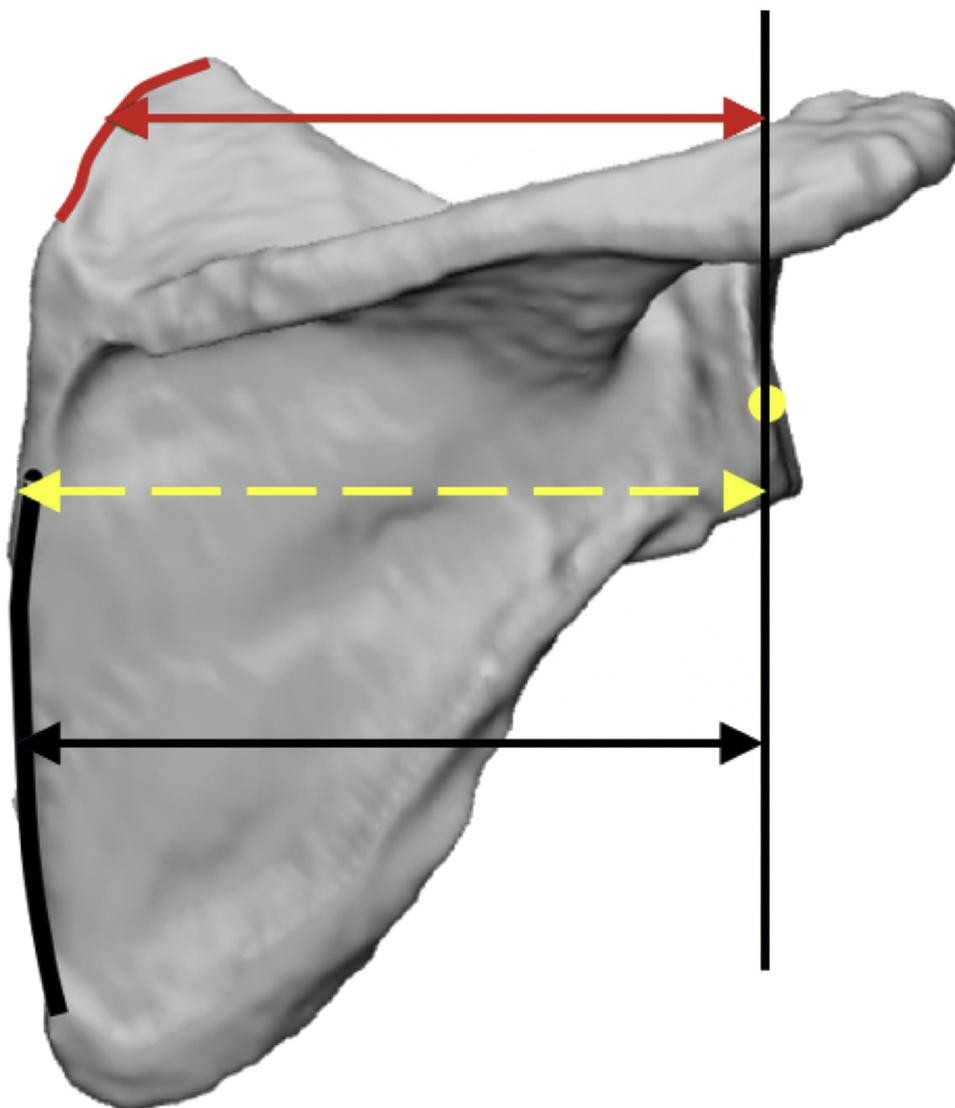
Next, a series of linear measures was made. The critical shoulder angle was measured as the angle between a line from the inferior to the superior glenoid poles and a line between the inferior glenoid rim and the most lateral acromial point, as previously described.<sup>6</sup> The lateral acromial offset was measured as the mediolateral distance (ie, distance projected onto the YZ, coronal plane) between the glenoid center and the mean mediolateral (Z) position of the lateral acromial edge. This measurement differed from the traditional measurement of critical shoulder angle<sup>21</sup> to include the entire lateral acromial edge instead of only using the most lateral point, as our goal here was not to replicate the critical shoulder



**Figure 1** The coordinate system of the scapula. The scapular plane (coronal, YZ) was defined by the points at the glenoid Center (blue circle), the trigonum spinae, and the most distal point on the inferior angle. The transverse plane (XZ) and sagittal plane (red, XY) were perpendicular to the scapular plane, where anterior (+X), superior (+Y), and lateral (+Z) formed the principle axes of the scapular coordinate system.



**Figure 2** The lateral (a) and posterior view (b) of a three-dimensional scapular reconstruction demonstrates the acromial offset measurement (yellow arrow) between the glenoid Center (yellow dot) and the Center of gravity of the lateral acromial edge (orange area, yellow dot). The acromial offset was the distance along the +Z axis, between the red lines oriented in the YZ plane (Fig. 1).



**Figure 3** This posterior view of a three-dimensional scapular reconstruction illustrates the methodology for measurement of the mediolateral distance between the glenoid Center and the origins of the supraspinatus (red arrow), infraspinatus (black arrow), and subscapularis (dashed yellow arrow shown as this measure was made from anterior views). The origins of the rotator cuff muscle were highlighted and the Center of gravity function was used to determine where to measure with respect to the glenoid Center. The difference in Z coordinates was used so the measure was taken as projected onto the YZ plane.

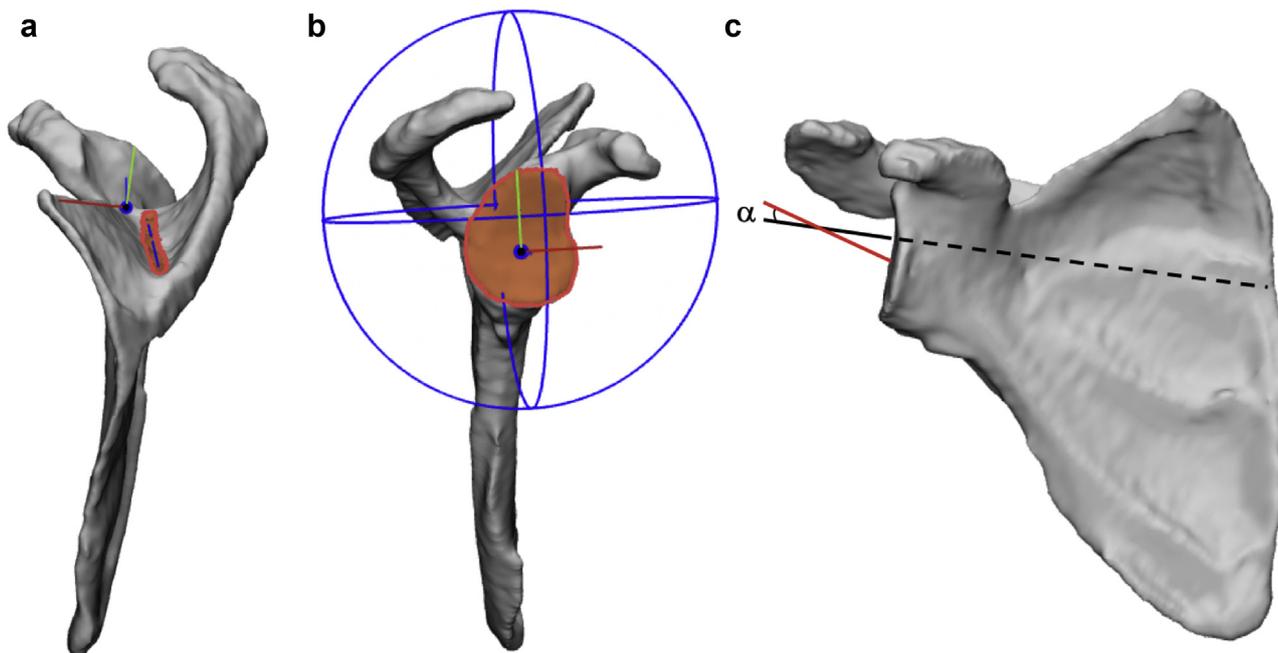
angle but to measure the underlying concepts. We defined the lateral acromial edge by selecting the most lateral surface and calculating center of gravity of the marked area (Fig. 2). The mediolateral distance between the glenoid center and the origins of the supraspinatus, subscapularis, and infraspinatus muscle were also quantified to measure medialization of the glenoid. The origins of the rotator cuff muscles were defined by marking the medial border of the scapula and again calculating the centers of gravity of the marked areas (Fig. 3). The height of the scapula was then measured by selecting the superior and inferior angles and taking the linear difference between the Y coordinates of the superior and inferior extrema. All distance measurements were normalized to scapular height to correct for differences in overall size of the bone.

Finally, we measured glenoid inclination. The portion of the 3D model that comprised the glenoid face was marked. Using the automatic fitting techniques provided by the Mimics software suite, a sphere was fit to the marked area of the glenoid face, and a line was drawn from the center of the glenoid and the center of this

sphere (Fig. 4). The floor of the supraspinatus fossa was selected, and the inertial axis was generated.<sup>9</sup> Glenoid inclination was defined as the angle between the line from the glenoid center to the sphere center and the inertial axis of the fossa projected into the coronal plane. This method of measurement was used as it was felt to be most accurately reflect glenoid inclination including the entirety of the glenoid and scapula in three dimensions.

#### Included shoulders

By reviewing the laboratory records, we identified 151 shoulder CT scans; of which, 37 were excluded because a rotator cuff tear, 21 because they did not include the full scapula, three because they had moderate GHOA (osteophyte size 3-7 mm, Samilson-Prieto grade 2), 1 because there was a healed fracture, and 1 because a prior surgery distorted the anatomy. There were 63 scans that appeared normal and healthy. Of these, 36 were excluded as they were a contralateral side or were age < 50 years. An additional three normal patient scans were identified that met the criteria,



**Figure 4** The (a) medial, (b) lateral, and (c) anterior views of a three-dimensional scapular reconstruction demonstrates the measurement of glenoid inclination. First, we marked the floor of the supraspinatus fossa (orange area in a) and created a line for the inertial axis of the marked area in the scapular plane (black line in c). To measure glenoid inclination, we marked the glenoid face (orange area in b) and fit a sphere (blue lines in b) to the marked area. The line from the Center of the glenoid and the Center of this sphere (red line in c), and the fossa line, defined inclination ( $\alpha$ ) when projected into the scapular plane.

leaving 30 scapulae for inclusion in the normal group. There were 25 scapulae with mild GHOA (Samilson-Prieto grade 1); of which, we excluded 4 because they were a contralateral side and 1 because age > 65 years, leaving 20 scapulae for inclusion in the mild-GHOA group.

During the study period, 265 shoulder arthroplasties were performed for which there was a preoperative CT scan. One hundred thirteen were excluded as they underwent reverse total shoulder arthroplasty and it was not felt these could be included as this procedure was mostly performed for rotator cuff tear arthropathy and not GHOA at our institution during the study period, 38 were excluded as their preoperative diagnosis was not OA, and 15 were excluded because they could not be uploaded into our analysis software. This left 99 imaging studies; of which, 30 were younger than the age of 65 years with Samilson-Prieto grade 3 for inclusion.

#### Statistical analysis

All analyses were performed in Excel X (Microsoft, Redmond, WA, USA) and SPSS 25 (IBM, Armonk, NY, USA). To determine the reliability of each of the anatomic measurements, all measurements were performed by two observers and compared using intraclass correlation coefficients using a two-way mixed model of absolute agreement type and the single-measures result. *A priori*, intraclass correlation coefficient values of <0.75 were considered unacceptable. To determine the association between scapular morphology and GHOA, each of the radiographic measures were compared between groups using ANOVA or Kruskal-Wallis tests as appropriate depending on data normality as determined using the Kolmogorov-Smirnov test. Post hoc pairwise testing was performed with Student's *t*-tests or Mann-Whitney U tests based on data normality as determined using the Kolmogorov-Smirnov test. Discrete data were compared using  $\chi^2$  tests.

## Results

### Demographics

The groups did not differ in age, height, laterality, or gender (Table I). However, there were differences detected in weight, with the severe-GHOA group having higher weight than the mild-GHOA ( $P = .003$ ) and normal ( $P < .001$ ) groups. The Samilson-Prieto grade reliability was acceptable, with 82% agreement and kappa of 0.693. All anatomic measurements had acceptable interobserver measurement reliability (Table II).

### Anatomic measures

There were no differences in lateral acromial offset between the normal group and either the GHOA group, but the severe-GHOA group had a more laterally offset lateral acromial edge than the mild-GHOA group ( $P = .009$ , Table I). The severe-GHOA group had a smaller critical shoulder angle than either the normal ( $P = .003$ ) or mild-GHOA groups ( $P = .020$ ), but the normal and mild-GHOA groups did not differ from one another ( $P = .965$ ). Examining glenoid inclination, the severe-GHOA group had a more inferiorly inclined glenoid than both the mild-GHOA group ( $P < .001$ ) and the normal group ( $P = .002$ ), but there were no differences between the mild-GHOA group and normal group ( $P = .281$ ). No differences were detected in rotator cuff muscle fossa lengths.

## Discussion

Within our study, severe GHOA was associated with smaller critical shoulder angle, and a more inferiorly inclined glenoid than either normal or mild-GHOA scapulae, but there were no differences in lateral acromial offset between normal healthy scapulae and either the mild or severe GHOA. These findings suggest that the prior studies demonstrating a difference in critical shoulder angle

**Table I**  
Study group attributes.

Variable	Normal	Mild GHOA	Severe GHOA	Overall P value	Norm. vs. Mild	Norm. vs. Severe	Mild. vs. Severe
<b>Demographics</b>							
Age	57 ± 5 (50 to 66)	59 ± 5 (45 to 67)	60 ± 8 (27 to 66)	.209	0.087	0.127	0.967
Height (cm)	161 ± 47 (145 to 193)	174 ± 11 (152 to 191)	175 ± 11 (152 to 198)	.633	0.237	0.104	0.635
Weight (kg)	72 ± 27 (45 to 125)	75 ± 21 (40 to 109)	96 ± 25 (55 to 152)	<b>.001</b>	0.634	<b>&lt;0.001</b>	<b>0.003</b>
Right-sided	67% (20/30)	85% (16/19)	50% (15/30)	.084	0.175	0.217	0.030
Female – gender	27% (8/30)	37% (7/19)	27% (8/30)	.696	0.451	1.000	0.451
<b>Anatomic measurements</b>							
Scap. Ht. (mm)	155 ± 12 (129 to 176)	154 ± 15 (119 to 178)	161 ± 12 (136 to 187)	.097	0.658	0.073	0.064
Acromial Offset	0.22 ± 0.04 (0.13 to 0.31)	0.21 ± 0.03 (0.13 to 0.26)	0.24 ± 0.07 (0.10 to 0.37)	<b>.049</b>	0.065	0.107	<b>0.009</b>
CSA (°)	34 ± 4 (26 to 40)	34 ± 4 (24 to 42)	30 ± 5 (16 to 38)	<b>.002</b>	0.965	<b>0.003</b>	<b>0.020</b>
Inclination (°)	12 ± 5 (0 to 21)	14 ± 5 (1 to 20)	7 ± 6 (-5 to 21)	<b>&lt;.001</b>	0.281	<b>0.002</b>	<b>&lt;0.001</b>
Subscapularis	1.00 ± 0.14 (0.72 to 1.28)	0.98 ± 0.13 (0.71 to 1.20)	1.06 ± 0.14 (0.81 to 1.34)	.122	0.586	0.124	0.063
Supraspinatus	0.93 ± 0.13 (0.65 to 1.19)	0.91 ± 0.12 (0.66 to 1.11)	0.98 ± 0.13 (0.77 to 1.25)	.184	0.678	0.161	0.093
Infraspinatus	1.01 ± 0.14 (0.72 to 1.27)	1.00 ± 0.15 (0.72 to 1.23)	1.08 ± 0.15 (0.81 to 1.39)	.087	0.711	0.070	0.060

CSA, critical shoulder angle; GHOA, glenohumeral osteoarthritis; Scap. Ht., scapular height.

Significant differences are bolded.

Continuous data are presented as mean ± standard deviation [range] and categorical data are presented as percentage.

Acromial offset, subscapularis length, supraspinatus length, and infraspinatus length are normalized to scapular height and are thus dimensionless.

**Table II**  
Reliability statistics for each radiographic measure.

Variable	ICC [95% CI]
Critical shoulder angle	0.831 [0.748 to 0.889]
Acromial offset	0.823 [0.735 to 0.883]
Subscapularis length	0.966 [0.948 to 0.979]
Supraspinatus length	0.904 [0.854 to 0.938]
Infraspinatus length	0.926 [0.885 to 0.952]
Scapular height	0.959 [0.926 to 0.974]
Inclination	0.905 [0.855 to 0.938]

CI, confidence interval; ICC, intraclass correlation coefficient.

related to GHOA may be explained by glenoid inclination and not by acromial morphology. While further research will be needed to prospectively follow up patients with pre-OA and mild OA, the lack of inferior inclination in the mild-GHOA group calls into question whether inferior inclination predates severe OA. When compared with healthy control scapulae, mild GHOA scapulae did not differ for any other anatomic metrics except for increased patient body weight.

There were no differences between the normal scapulae and either GHOA groups for lateral acromial offset. While the mild-GHOA and severe-GHOA groups differed, where the severe-GHOA group had a more laterally offset lateral acromion edge than the mild-GHOA group, this was the opposite of our hypothesis. Prior research has suggested that the critical shoulder angle differs in scapulae with and without OA.<sup>4,5,21,27,32</sup> The critical shoulder angle measurement incorporates contributions from both glenoid inclination and the extent of the lateral protrusion of the acromion in the coronal plane. A more medial deltoid origin due to a more medial acromial offset has been theorized to increase the compressive force and decrease the shear force on the glenoid as generated by the deltoid. Our findings suggest that severe GHOA is not associated with a more medial acromial offset and therefore does not play a role in the etiology of GHOA and that the prior studies demonstrating a difference in critical shoulder angle<sup>4,5,21,26,27,32</sup> may not be explained by this aspect of acromial morphology. This is in contrast to a prior study performed by Beeler et al,<sup>3</sup> which suggested that the lateral extent of the acromion may play a bigger role in explaining critical shoulder angle than glenoid inclination. This study only incorporated the most lateral acromial point and not the entirety of the lateral acromion as we did and this difference in measurement technique most likely explains the difference in findings.

Severe GHOA was associated with a more inferiorly inclined glenoid than either normal or mild GHOA scapulae. A more inferiorly inclined glenoid has been theorized to result in more compressive joint reaction forces. If such an effect exists, it could be expected to have a dose-response relationship – that is normal → mild GHOA → severe GHOA should exhibit increasing degrees of inferior inclination. The question could then be asked: is this a static morphologic phenomenon where native inclination predisposes a person to development of GHOA or a progressive phenomenon where inclination adapts over time owing to the pathologic process? The present data suggest the latter. Patients with severe GHOA had more inferiorly inclined glenoids, while those with mild GHOA or healthy shoulders did not differ in inclination. While further research will be needed to prospectively follow up patients with pre-OA to confirm, the lack of inferior inclination in the mild GHOA group suggests that the difference in inclination may be more likely owing to acquired glenoid inferior inclination deformity secondary to osteoarthritic wear and not to a preexisting inferior inclination within the glenoid. The cause of wear into inferior inclination is unclear, but certainly, the forces on the shoulder with use, the qualities of the humeral or scapular bone, or the constraints of the soft tissues as they become affected by GHOA could all contribute. We also did not observe any differences in the mediolateral position of the glenoid center, as measured with rotator cuff muscle fossae lengths, between groups, suggesting that scapular width does not play a role in the etiology of GHOA.

The rate of OA progression and the rate of osteophyte growth in GHOA are unknown. It may vary between patients such that OA may progress more rapidly in some as a combination of genetic, biologic, and mechanical factors. Certainly, osteophytes that are >7 mm (Samilson-Prieto Grade 3) were once <3 mm (Samilson-Prieto Grade 1). Our study purposefully was designed to include patients of similar ages with different stages of GHOA. Sequential changes could be suggestive of a dose-response relationship but were not readily observed herein. All detected differences had the common factor of severe GHOA as one group in the comparison. No changes between normal healthy shoulder and mild GHOA were observed. As this is a cross-sectional study and not a longitudinal study, it may be that the mild-GHOA subjects within our cohort would not progress to severe GHOA. To firmly establish causation, future studies will need to demonstrate that the inclination differences described herein predate the development and progression of GHOA.

Severe GHOA was associated with increased patient body weight. Multiple studies have demonstrated that increased patient body mass index is associated with OA in the lower extremity.<sup>12,23</sup> While a larger sample size would be needed to confirm these findings, the authors theorize that increased weight of the arm increases the overall stress within the joint, which increases the risk for OA. Increased patient weight may therefore be a modifiable risk factor for severe GHOA.

### Limitations

Our study has several limitations. First, this is a retrospective study of cadavers and patients available within our laboratory and medical records and is thus subject to the selection biases inherent with this study design. Second, our study describes an association and does not establish causation between morphology and pathology. Prospective studies would be necessary to confirm causation. Third, our severe GHOA group did not include any GHOA severe enough to result in medialization of the glenoid center, as measured by scapular width. Fourth, the Samilson-Prieto grade is imperfect as it does not include joint space narrowing or other hallmarks of OA other than osteophyte size. However, the authors felt that it provided the most complete assessment of OA available with the existing CT data. Many of the cadavers included in the study did not have radiographs and thus an analysis using plain radiographs is not possible with this data set. Finally, these measures were motivated by the use of the critical shoulder angle and the controversy that remains in its predictive capabilities. As a 2-dimensional or simplified 3D measure, these methods may miss more complex 3D interactions of the bones and soft tissues within the shoulder complex.

### Conclusion

Normal and severe GHOA shoulders differ in critical shoulder angle and glenoid inclination but not acromial offset. The lack of a difference in critical shoulder angle or inferior inclination between mild-GHOA and normal groups calls into question whether inclination and critical shoulder angle differences predate severe GHOA.

### Disclaimers:

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**Conflicts of interest:** Peter Chalmers is a paid consultant for Depuy-Mitek, serves on the editorial board for the Journal of Shoulder and Elbow Surgery, receives intellectual property royalties from Depuy, and is a paid speaker for Depuy.

Heath Henninger, Matt Miller, John Wheelwright, Jun Kawakami, and Lindsay Beck certify that they have no commercial associations (eg, consultancies, stock ownership, equity interest, patent/licensing arrangements, and so on) that might pose a conflict of interest in connection with the submitted article.

Robert Tashjian is a paid consultant for Zimmer/Biomet, Wright Medical and Depuy-Mitek; has stock in Conexions, INTRAFUSE, Genesis and KATOR; receives intellectual property royalties from Wright Medical, Shoulder Innovations, and Zimmer/Biomet; receives publishing royalties from Springer and the Journal of Bone and Joint Surgery, and serves on the editorial board for Shoulder &

Elbow and the Journal of the American Academy of Orthopaedic Surgeons.

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