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A biomechanical confirmation of the relationship between critical shoulder angle (CSA) and articular joint loading

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Background: The critical shoulder angle (CSA) has been shown to be correlated with shoulder disease states. The biomechanical hypothesis to explain this correlation is that the CSA changes the shear and compressive forces on the shoulder. The objective of this study is to test this hypothesis by use of a validated computational shoulder model. Specifically, this study assesses the impact on glenohumeral biomechanics of modifying the CSA.
Methods: An inverse dynamics 3-dimensional musculoskeletal model of the shoulder was used to quantify muscle forces and glenohumeral joint forces. The CSA was changed by altering the attachment point of the middle deltoid into a normal CSA (33°), a reduced CSA of 28°, and an increased CSA of 38°. Subject-specific kinematics of slow and fast speed abduction in the scapular plane and slow and fast forward flexion measured by a 3-dimensional motion capture system were used to quantify joint reaction shear and compressive forces.
Results: Increasing the CSA results in increased superior-inferior forces (shearing forces; integrated over the range of motion; $P < .05$). Reducing CSA results in increased lateromedial (compressive) forces for both the maximum and integrated sum of the forces over the whole motion ($P < .01$).
Discussion/Conclusion: Changes in the CSA modify glenohumeral joint biomechanics with increasing CSA producing higher shear forces that could contribute to rotator cuff overuse, whereas reducing the CSA results in higher compressive forces that contribute to joint wear.
Level of evidence: Basic Science Study; Computer Modeling

Keywords: Critical shoulder angle (CSA); glenohumeral joint; joint wear; rotator cuff; computational shoulder model

The shape of the scapula and especially of the acromion has historically been considered as a potential etiology for shoulder pathologies including those of the glenohumeral joint (GHJ). Codman,[9] Armstrong,[2] and Neer[24] described the association of specific acromion shapes with

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Institutional review board approval was not required for this basic science study.

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degenerative rotator cuff tear (RCT), leading to the well-known impingement syndrome and the extrinsic mechanical conflict theory. Since then, a broader understanding of degenerative RCT physiopathology[33] (ageing of the tendon, modification of local vascularization, genetic disposition) has challenged this theory, thus questioning the absolute role of the acromion shape in this process. Research over the past 15 years has continued to focus on the shape of the acromion in the coronal plane, including proposed radiological parameters to describe the lateral extension of the acromion.[4,26] A recent and widely cited study from Moor et al[22] proposed the concept of the critical shoulder angle (CSA), a measure that takes into account the tilt of the glenoid (inclination) and the lateral extension of the acromion. In a population of 279 patients, they found that the mean CSA was significantly different between a disease-free shoulder group (33.1°), an RCT group (38.0°), and a primary osteoarthritis group (OA, 28.1°). Numerous clinical observational studies have confirmed these findings.[3,4,7,10-12,14,15,21,23,36,40]

The link between the clinical observations and the CSA is hypothesized to be biomechanical in nature,[1,25,20,13,38,39] by changing the magnitude and direction of the deltoid force. The hypothesis of this study is that an increased CSA would result in higher shearing forces (SF) that would be associated with RCT and a decreased CSA would result in higher compressive forces (CF), associated with primary OA.

The objective of this study is to test the hypothesis by assessing the impact on GHJ biomechanics of modifying the CSA by use of a validated computational shoulder model.

Material and methods

The United Kingdom National Shoulder Model (UKNSM)

An inverse dynamics 3-dimensional musculoskeletal model of the upper limb[8] was used to quantify muscle and GHJ forces. This model quantifies forces in 87 muscle elements, 3 ligaments, and joint reaction forces of the sternoclavicular, acromioclavicular, scapulothoracic, glenohumeral, and elbow joint (Fig. 1). The analysis begins by solving the determinate inverse dynamics intersegmental moments based on measured kinematics. Then an optimization algorithm is used to minimize the sum of muscle stresses squared to solve the muscle load-sharing redundancy. The model has been validated for GHJ force measures through comparison with instrumented anatomical shoulder replacement measurements[6,41] for a driving task[27] and other tasks of daily living[18] and for muscle forces through comparison with electromyography.[17] The model is customized to each subject by scaling segment lengths and body segment parameters.[16] In brief, clavicle and scapula segments were homogeneously scaled based on relative segment lengths between the model and subject. An ellipsoid represented the scapulothoracic gliding plane; this was nonhomogeneously scaled using an optimization procedure to minimize the difference between digitized anatomical landmarks and the final ellipsoid. A partially closed chain method is used to optimize scapula and clavicle kinematics, in which the least-squares difference to the measured scapular and clavicle kinematics is minimized and the scapula medial border is constrained not to penetrate the thorax wall (represented by an ellipsoid).[30]

Tests conditions

The scapula within the UKNSM was imported into computer-aided engineering software to allow the effect of medializing and lateralizing the acromion to be modeled, thus changing the CSA and therefore the 3-dimensional position of the attachment point of the middle deltoid. This was implemented in the software by sketching new points and lines parallel to the plane of measurement of the CSA and coincident with the original middle deltoid attachment point. The changes were made to create 3 different CSAs: a normal CSA of 33°, a reduced CSA of 28°, and an increased CSA of 38°, which reflect the changes previously reported as clinically significant.[22]

To test the effect of different subject-specific kinematics, a previously obtained dataset of kinematics was used.[18] These kinematics were measured using a 9-camera optical motion tracking system with a set of 21 retroreflective markers used to track the thorax, clavicle, humerus, and forearm segments.[34,35,42] The kinematics of 6 healthy male subjects were used (aged 25 years ± 2 years) who each performed 4 motions with a 2 kg hand load to provide a resistance to motion without fatiguing the subjects. These motions were slow and fast speed abduction (in the scapular plane) and slow and fast forward flexion.[32] Each subject performed 3 trials per motion. These datasets were passed through the UKNSM after appropriate subject scaling.[30]

Outcomes measures

All output measures were normalized to humerothoracic elevation, and all forces were normalized to the subject's body weight and integrated over the range of motion from 30° to 120° of humerothoracic elevation. The following parameters were analyzed to test the effects of CSA changes on GHJ biomechanics:

1. the magnitude (maximum and integrated over the whole motion) of the GHJ SF, representing the superior-inferior (SI) forces and the anteroposterior (AP) forces acting in the sagittal plane that are exerted on the joint during motion
2. the magnitude (maximum and integrated over the whole motion) of the GHJ CF, representing the forces acting in the frontal plane (lateromedial) that are exerted on the joint during motion.

Statistical analysis

Repeated measures 2-way analysis of variance, with Bonferroni post hoc correction, was conducted on the results using SPSS software (2014; IBM Corp., Armonk, NY, USA). The assumptions of the repeated measures 2-way analysis of variance method were tested for all measures. The independent variables are CSA (normal, increased, reduced) and motion (fast and slow, forward flexion, and scapular plane abduction); the dependent variables are the joint forces (integrated AP shear, max AP shear, integrated SI shear, max SI shear, integrated CF, max CF). Mauchly's test indicated that the assumption of sphericity had been violated for the main effects of CSA and motion for several measures; therefore, degrees of freedom were corrected using Greenhouse-Geisser estimates of sphericity (ε).

Results

Statistically significant differences were found for the integrated SI shear force, the integrated CF, and the maximum CF (Table I). These statistical results are presented in Fig. 2. In summary, an increased CSA angle resulted in 111% higher integrated SI shear forces ($P < .05$) when compared with normal. For the CF, a reduced CSA angle resulted in a significant increase in the CF (integrated: 2.8% increase, $P < .01$; maximum: 2.8% increase, $P < .05$) compared with normal CSA and also when compared with increased CSA (integrated: 5.5% increase, $P < .01$; maximum: 5.3% increase, $P < .05$). There were no main effects found for motion, nor an interaction effect of motion and CSA.

The mean maximum SI forces were 13% BW, 12% BW, and 21% BW for reduced, normal, and increased CSA, respectively. The maximum CF were 132% BW, 136% BW, and 129% BW for reduced, normal, and increased CSA, respectively.

Discussion

This is the first study to assess the effects of changes in CSA on GHJ biomechanics through a computational shoulder model to test the mechanically based hypothesis. The results confirm the initial hypothesis based on literature,[38] that is to say increasing the CSA results in increased SI forces (SF), whereas decreasing the CSA results in increased lateromedial (CF) forces (Fig. 3).

Our results are also consistent with those of 2 cadaver studies previously published about the subject. Even if the model and analysis method are different, Viehöfer et al[38] found that with an increased CSA, the ratio of GHJ shear to joint compression forces increased (peak difference of 23% at 50° of thoracohumeral abduction compared with a normal CSA), requiring substantially increased compensatory supraspinatus loads (increased by 13%-33% between 33° and 37° of elevation compared with a normal CSA).[13]

These cadaver studies combined with the study presented here provide simple mechanical explanations for the results:

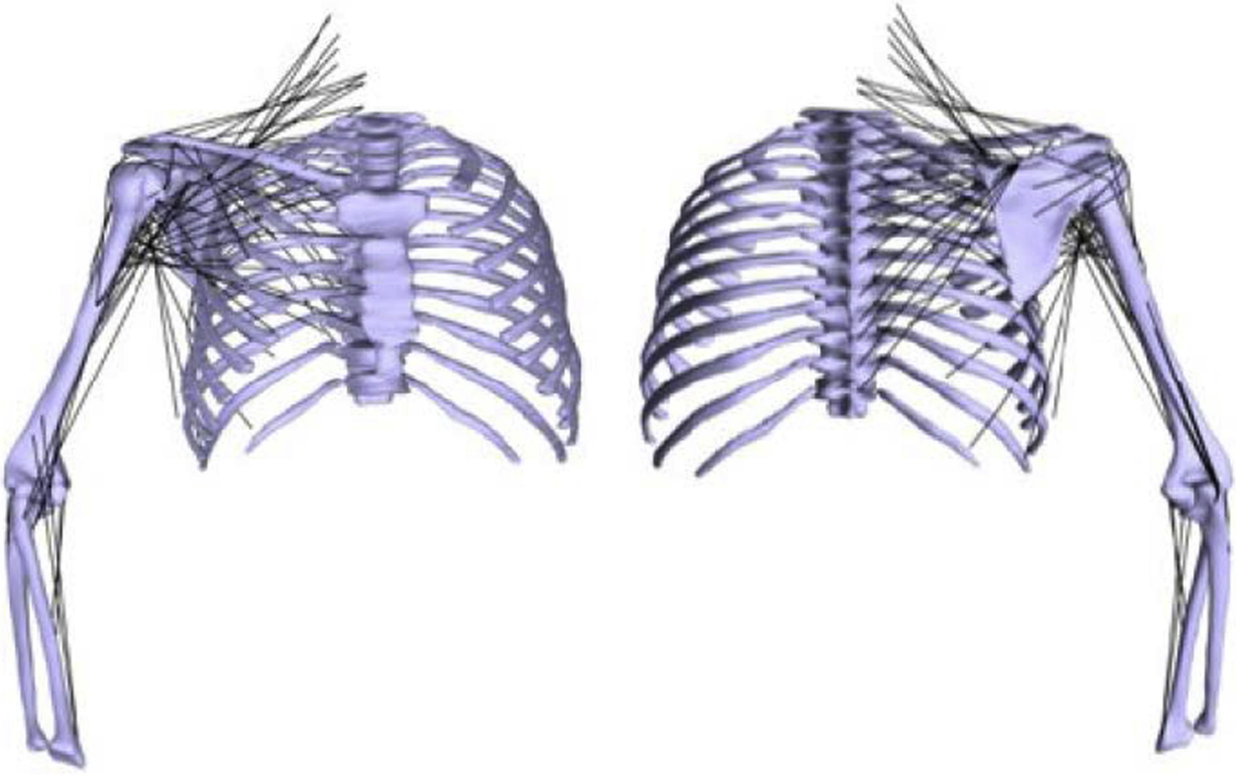

Figure 1 Illustration of the United Kingdom National Shoulder Model.

Table I Repeated measures ANOVA

Variable	Mauchly's sphericity	Main effect		Pairwise CSA		
				Nor-Red	Nor-Inc	Red-Inc
Integrated AP shear	$\chi^2(2) = 9.5$, $P < .01$, $\varepsilon = 0.52$	$P = .598$				
Max AP shear	$\chi^2(2) = 11.6$, $P < .01$, $\varepsilon = 0.51$	$P = .595$				
Integrated SI shear		$\mathbf{P = .026}$	$\mathbf{F(2, 10) = 5.34}$		$P = .044$	
Max SI shear	$\chi^2(2) = 5.14$, $P < .05$, $\varepsilon = 0.55$	$P = .068$				
Integrated CF	$\chi^2(2) = 6.23$, $P < .05$, $\varepsilon = 0.56$	$\mathbf{P = .003}$	$\mathbf{F(1.1, 5.6) = 24.55}$	$P = .001$		$P = .01$
Max CF	$\chi^2(2) = 6.84$, $P < .05$, $\varepsilon = 0.55$	$\mathbf{P = .009}$	$\mathbf{F(2, 10) = 14.97}$	$P = .008$		$P = .03$

ANOVA, analysis of variance; *AP*, anteroposterior; *SI*, superior-inferior; *CF*, compressive force; *CSA*, critical shoulder angle; *Nor*, normal CSA (33°); *Red*, reduced CSA (28°); *Inc*, increased CSA (38°).

The main effects for the separate force directions (AP, SI, and CF), and the pairwise comparisons for the force directions that significantly differed between CSA conditions.

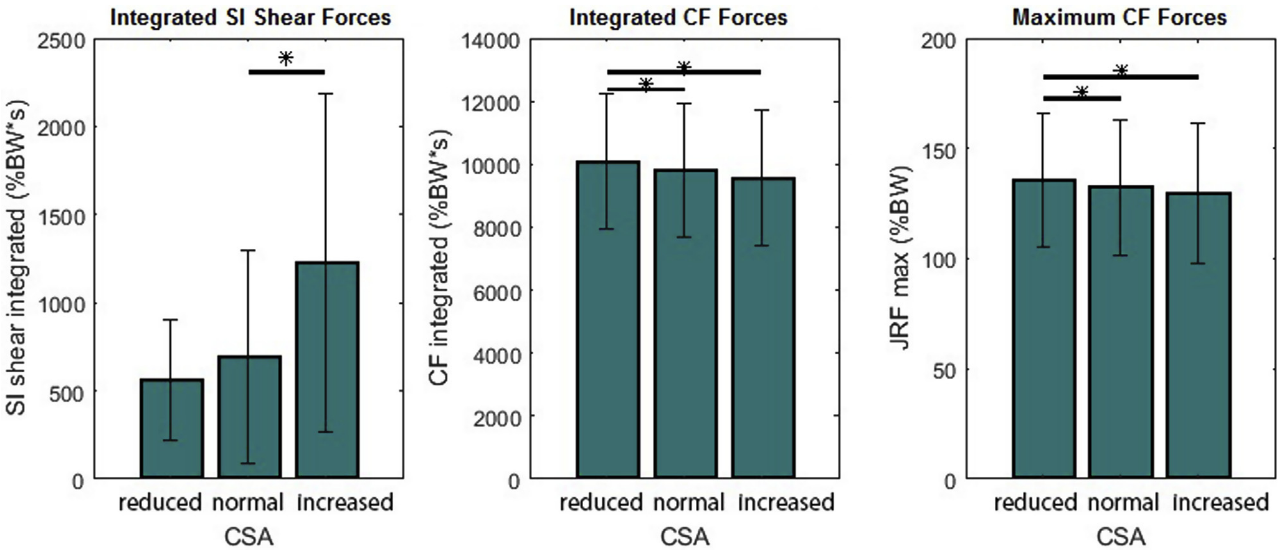

Figure 2 Statistically significant changes in joint forces due to changed CSA. These conditions showed a main effect in the ANOVA analysis. The error bars indicate standard deviation. *CSA*, critical shoulder angle; *ANOVA*, analysis of variance; *CF*, compressive force; *JRF*, joint reaction forces; *SI*, superior-inferior; *, significant difference.

1. An increased CSA would result in a lateralized proximal deltoid insertion. Consequently, the vector force of the deltoid during abduction would be more vertical, requiring a greater horizontal force from the cuff to stabilize the joint (by counterbalancing the SF). Ultimately, this increase in muscle use could potentially, in combination with other factors, lead to a degenerative RCT in the long term.
2. A reduced CSA would result in a medialized proximal deltoid insertion. Consequently, the vector force of the deltoid during abduction would be more oblique (resultant internal translation), which in addition to the vector force of the cuff (mostly horizontal) could increase the load (CF) on the GHJ. Finally, this could lead to OA in the long term.

It is known that small elevated mechanical loading is associated with the instigation and progression of OA,[1,28] suggesting that, although small in percentage terms, the statistical differences in key mechanical variables found here may also be clinically significant.

Computational simulation models are frequently used to analyze human joint biomechanics and have been validated in studies that quantified articular loading in activities of daily living, and the biomechanical

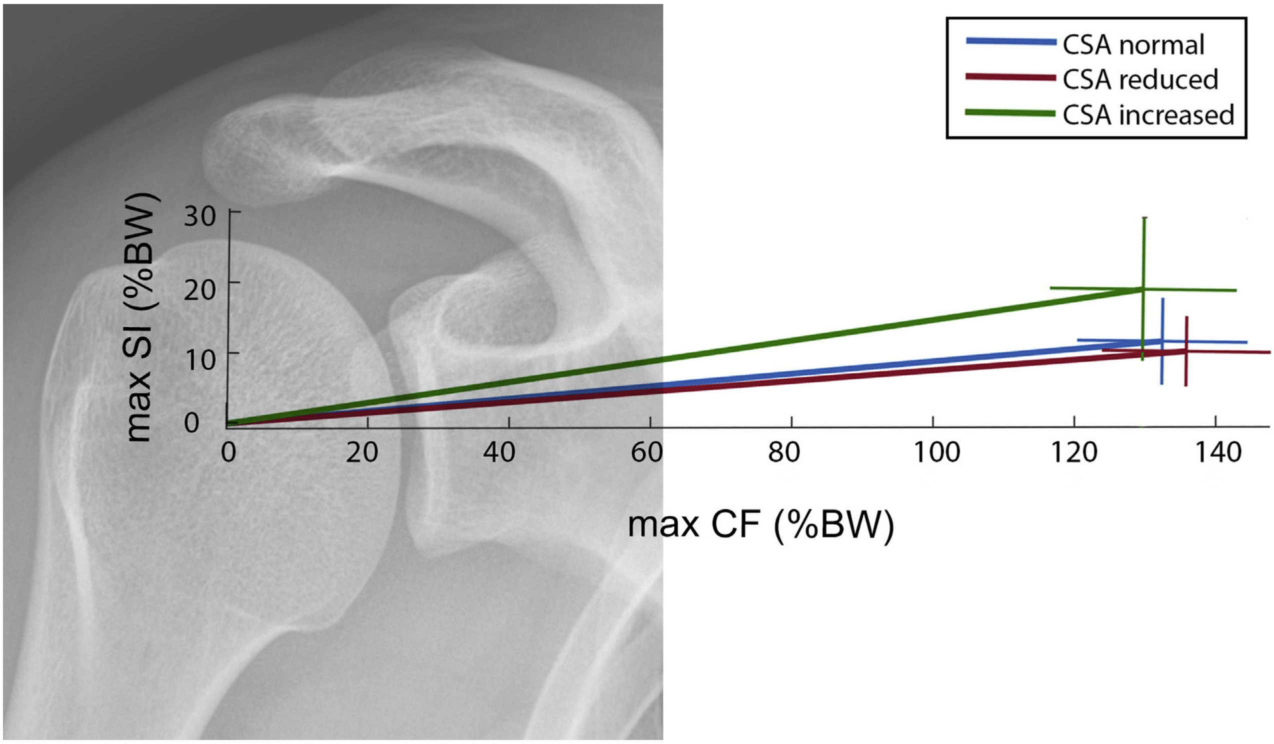

Figure 3 Vector plot of the maximum joint reaction forces in the mediolateral and SI direction for the 3 CSA angles. The integrated shear force in the SI direction was significantly larger for increased CSA compared with normal CSA. The reduced CSA showed significantly higher CF compared with normal CSA and increased CSA. *SI*, superior-inferior; *CSA*, critical shoulder angle; *CF*, compressive force; *BW*, % of body weight.

consequences of a pathology, surgery, and arthroplasty.[8,18,29,37] Concerning shoulder modeling, the UKNSM (formerly Newcastle Shoulder Model)[8] is one the oldest validated inverse dynamics-based models.[31] Body segment parameters are based on a large group of young living patients.[19] There are limitations with such models and so, although the model has been validated by comparison with instrumented joint replacements and electromyography, such computer simulations remain ‘‘models’’ and are, therefore, a surrogate of direct in vivo biomechanical measurements.

Apart from technical considerations of the computational model, this study has some other limitations. Tests were only performed in simplified motions of pure scapular abduction or flexion, whereas most daily activities have ranges of motion that combine abduction and flexion with obligate rotations.[18] Moreover, the model is based on a range of subjects with normal anatomy, and this study varied the CSA in isolation, whereas there might be other changes present with a change in CSA angle, such as glenoid version or muscle stiffness that may influence the model outputs. In addition, a change in anatomy might also result in a further change in kinematics, although this was mitigated in this study by using a set of data from 6 healthy subjects, rather than just from 1 subject. Finally, we could have studied the different components of the CSA separately, but even if the lateral acromial roof extension has a greater influence in pathogenesis of degenerative RCT and concentric OA than acromial height or glenoid inclination, the CSAs remain the best factor to predict these pathologies.[5]

Conclusion

Through a validated computational shoulder model, combined with in vivo motion analysis experiments, this study demonstrates that changes in the CSA modify GHJ biomechanics. Increasing the CSA results in higher shear forces, requiring increased rotator cuff use to neutralize the shear that is potentially damaging in the long term. Decreasing the CSA results in a higher joint CF that leads to increased joint wear.

These findings support previous clinical observational and biomechanical studies that alterations in CSA may have a role in common shoulder pathologies such as RCT or OA. Consequently, surgical restoration to a ‘‘normal’’ CSA is recommended when treating patients with such pathologies, for example, lateral acromioplasty after rotator cuff repair or ensuring control of glenoid inclination when conducting arthroplasty surgery.

Disclaimer

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