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Subject-Specific Finite Element Modeling of the Human Shoulder Complex Part 2: Quantitative Evaluation of the Effect of Rotator Cuff Tear Propagation on Glenohumeral Joint Stability

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ABSTRACT The objective of this paper is to quantitatively evaluate the effects of rotator cuff tear propagation on glenohumeral joint stability in a previously constructed and validated finite element shoulder model. Rotator cuff tears with a sequence of increasing sizes were created from the anterior portion of the supraspinatus osseous insertion site and propagated posteriorly through the infraspinatus tendon until a complete tear extended through the entire teres minor tendon. Finite element simulations were performed in the same physiological loading and boundary conditions as in the original model. A novel integrative stability index was proposed and used for quantitative analysis of the simulated results. By defining the healthy condition as the baseline (100%), the stability index decreased slightly with small tear sizes but declined suddenly after half tear of the infraspinatus accompanied by a complete tear of the supraspinatus tendon until the full tear condition, when the index reached 0.41%. These results confirm the clinical and cadaveric findings that glenohumeral joint stability generally decreases as the size of the rotator cuff tear increases and that the critical tear size which leads to the loss of normal shoulder biomechanics was half tear of the infraspinatus accompanied by a complete tear of the supraspinatus tendon. It is concluded that the finite element shoulder model and the proposed novel stability ratio can accurately predict shoulder biomechanics in the investigated rotator cuff condition. Both the model and ratio may have the potential to be used to improve diagnostic and therapeutic strategies for clinicians.

INDEX TERMS Rotator cuff tear, glenohumeral joint stability, shoulder complex, subject-specific, finite element analysis.

I. INTRODUCTION

The rotator cuff (RC), which includes the supraspinatus, infraspinatus, teres minor and subscapularis, is a group of muscles that surround the glenohumeral (GH) joint. The RC is considered as the most important stabiliser of the GH joint [1], [2], and pain, including tears, is the most common source of shoulder complaints [3]. Tears of the, especially large tears, can result in severe pain and functional loss [4], [5]. RC tears commonly initial at the anterior portion of the osseous insertion of the SUP tendon and extend posteriorly [6]–[8]. During the early stages of tear propagation, small sizes of tear (for example: an isolated supraspinatus tear) have little effect on the biomechanics of the GH joint [9]–[11]. Upward migration of the humeral head during elevation of the limb is typically seen in patients

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with massive tears [12]–[14]; however, the exact size of RC tear that leads to the loss of normal shoulder biomechanics remains unclear [10].

Because excessive proximal migration of the humeral head is a clinical marker for a loss of GH joint stability [15], most studies have investigated variations in superior-inferior and/or anterior-posterior migration of the humeral head following RC tears in either cadaveric simulations [11], [16]–[20] or via direct measurement of patients [6], [21]. Recent studies have measured the *in-vivo* three-dimensional GH and scapular kinematics after RC tears [22], [23]. Some cadaveric and computational studies also investigated variations in the GH joint bone-on-bone contact forces (BOBFs) or muscle forces following RC tears [5], [9], [24]–[26]. However, few studies have reported the simultaneous determination of variations in the GH kinematics and the BOBFs and the contact pressure and contact area due to the propagation of RC tears. It is nearly impossible to mechanically address these results of multiple tear sizes with one specimen. Therefore, computational methods are promising, especially a subject-specific finite element (FE) computational framework capable of integrating *in-vivo* kinematic and kinetic data. Previous FE studies on RC tears were mainly conducted to investigate the aetiological mechanism due to the distribution of stress in the supraspinatus tendon [27]–[30]. In these studies, from early FE models that used two-dimensional geometries of the humeral head and supraspinatus tendon [27] to recent models that used three-dimensional geometries of the humerus and scapula and all RC tendons [30], an increasing trend in accuracy and complexity was seen. However, few FE studies have been conducted to evaluate the effects of a change in the stability of the entire GH joint due to RC tear propagation. The main limitations are the lack of a comprehensive anatomical model that contains the major musculoskeletal components and the lack of physiological loading and boundary conditions [31], [32].

Another obstacle to a thorough evaluation of the effect of RC tears on the stability of the GH joint is the lack of a method for quantitative analysis. The generally accepted concept of normal shoulder joint stability function, proposed by Lippitt and Mstsen [33], breaks down the comprehensive stability of the GH joint into two mechanisms: the concavity compression mechanism and the scapulohumeral balance mechanism. The concavity compression mechanism describes the stability function as a convex object (humeral head) that is pressed into a concave surface (glenoid fossa), whereas the scapulohumeral balance mechanism indicates that the surrounding soft tissues dynamically position the GH joint so that the BOBFs are balanced within the glenoid fossa. Based on the first mechanism, a commonly used stability ratio was defined as the translational force at dislocation divided by the artificially defined compressive load [34]. This stability ratio has been used in cadaveric and computational studies to investigate the influence on joint stability from changes in the bony geometry due to Bankart and Hill-Sachs lesions [25], [35]–[38]. Normally, this stability ratio calculation requires the experiment or simulation to be performed until the joint dislocates, which can be troublesome and unnecessary in some cases. Another method used to study GH joint stability is the average contact point/area [39], but it was merely a rough description of the joint stability. Excessive humeral head migration was used as a clinical marker [6], [11], [16]–[21]. However, it is more of a descriptive symptom than a ratio, and it commonly occurs in more extensive tears such as massive tears, which makes it difficult to use in less severe pathologies. No quantification ratio or method is currently capable of quantifying both the concavity compression and scapulohumeral balance mechanisms, which function together in the normal GH joint. In addition, the conformity function from the articular surfaces of the humeral head and glenoid fossa has not been accounted in the joint stability mechanism previously. This joint conformity mechanism would be the third mechanism after the traditional two mechanisms, because changes in the bony geometry caused by disorders can vary the stability via variation of the bony conformity. To conduct a thorough quantitative analysis of GH joint stability, a new stability index is needed to account for all of these mechanisms.

The objective of this study is to quantitatively investigate the effects of the propagation of RC tears on GH joint stability. We hypothesise that the humeral head migrates in a superior direction and that the stability of the GH joint decreases as the size of the cuff tear increases. We further hypothesise that a critical tear size (defined as when the stability index dramatically changes) exists which leads to the loss of normal shoulder biomechanics.

II. METHODS

A. SIMULATION OF RC TEAR PROPAGATION

The constructed and validated subject-specific FE model in 30° scapular abduction in the first part of this study, was used for this investigation (see Figure 1). In this model, detailed representations of the major musculoskeletal components around the GH joint were constructed based on

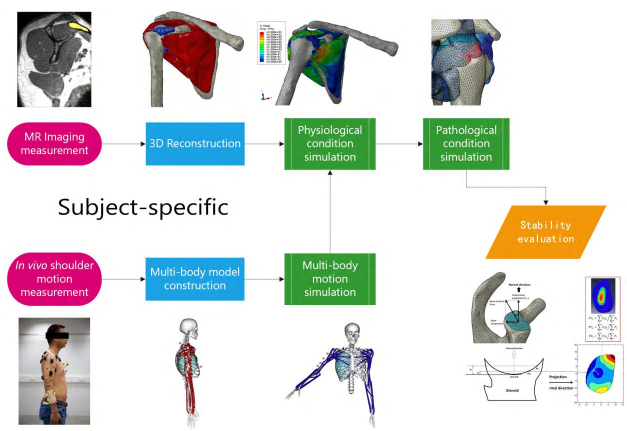

FIGURE 1. Schematic diagram of the construction and validation of the subject-specific finite element model of the shoulder complex.

TABLE 1. (a) Comparison of the bone-on-bone contact forces of the simulation results at 0°, 10°, 20° and 30° of abduction between this study and previous computational and experimental results from the literature. (b) Comparison of the superior-inferior movement of the humeral centre with respect to the scapula of the simulation results at 0°, 10°, 20° and 30° of abduction between this study and previous experimental results from the literature.

(a)

Bone-on-bone contact forces in each abduction angle (N)				Reference
0°	10°	20°	30°	
66	140	213	290	Poppen et al., 1978 [48]
5	66	125	176	Van der Helm et al.,1994 [49]
50	160	270	375	Terrier et al., 2008 [50]
30	100	215	450	Favre et al., 2012 [38]
N/A	N/A	152	225	Sins et al., 2015 [51]
12.74	86	175.18	229.32	Bergmann et al., 2007 [52]
8.18	91.45	146.14	408	This study

(b)

Superior-inferior movement of the humeral centre in each abduction angle (mm)				Reference
0°	10°	20°	30°	
N/A	1.43 (0.55)	2.03 (0.3)	1.53 (0.35)	Bey et al., 2008 [53]
0 (1.05)	N/A	N/A	0.77 (0.98)	Matsuki et al., 2012 [54]
N/A	N/A	N/A	2.16 (1.4)	Kijima et al., 2015 [23]
0	1.43	2.08	1.47	This study

*Standard deviation in parentheses

*As a result of the differing measuring techniques and definitions of the coordinate system of the GH joint, only the relative differences in the superior-inferior translations between abduction angles were compared. The data in the literature that did not begin from 0° abduction were changed to equivalent results in this study.

TABLE 2. Nine rotator cuff tear conditions and their FE model abbreviations.

Tear condition description	FE model abbreviations
Heathy condition	INTACT
Tear of the anterior half of the SUP tendon	1/2SUP
Complete tear of the SUP tendon	SUP
Advancement of the tear into anterior half of the INF tendon	SUP+1/2INF
Complete tear of the SUP and INF tendon	SUP+INF
Advancement of the tear into superior half of the TMIN tendon	SUP+INF+1/2TMIN
Advancement of the tear into superior 23/24 of the TMIN tendon	SUP+INF +23/24TMIN
Advancement of the tear into superior 95/96 of the TMIN tendon	SUP+INF+95/96TMIN
Complete tear of all three posterior RC tendons	FULL TEAR

*Fractions represent the proportional area of the tear in the affected tendon.

three-dimensional shoulder motion and geometric data from a young, healthy subject. The model was meshed using tetrahedral elements with sizes ranging from 1.2mm to 2mm and the total element number is 666587. In addition to 30° scapular abduction, quasi-static FE analyses were also conducted to simulate 0°, 10° and 20° of the measured motion. The model was validated by the simultaneously determined results of the BOBFs and the superior-inferior movement of the humeral centre during motion which were found to agree well with the previous experimental and numerical results (Table 1(A) and (B)).

The propagation of tears was defined as initialling from the anterior portion of the osseous insertion site of the SUP tendon and extending in a posterior direction until all three posterior RC tendons were completely torn. The simulation of RC tear propagation was conducted by performing a series of quasi-static FE simulations by revising the FE model above with a sequence of increasing tear sizes. Specifically, the sequence of tears was created by changing the contact surfaces in the muscle-bone bonding contacts (i.e., the firm attachment areas between the humeral head and the supraspinatus, infraspinatus and teres minor tendons). The tears were created from the anterior portion of the supraspinatus footprint and propagated in a posterior direction through the infraspinatus until a complete tear extended through the entire teres minor tendon. Including the original model, nine FE models were constructed with nine tear sizes, as summarised in Table 2. (Tears were initially created with an increment of 1/4 of the proportional area of the tear in each tendon. If the results showed significant variation, smaller proportions (such as 1/8, 1/24 and 1/96) of the relative tendon were performed. Therefore, many more tear sizes between the nine presented tear sizes were simulated. The nine presented tear sizes were representative of the simulation results.) Figure 2 shows the constructed FE models with these nine tear conditions. The FE simulations were conducted with the same loading and boundary conditions as the original simulation as follows: the clavicle and scapula were fixed, whilst the humerus was defined as free to move with no prescribed artificial control; the humerus was positioned and stabilised actively by the calculated *in-vivo* muscle loadings from multi-body model simulation and passively by the bony and the ligamentous configurations. Each FE simulation took 40 minutes to complete (24 cores and 48 Gigabytes memory).

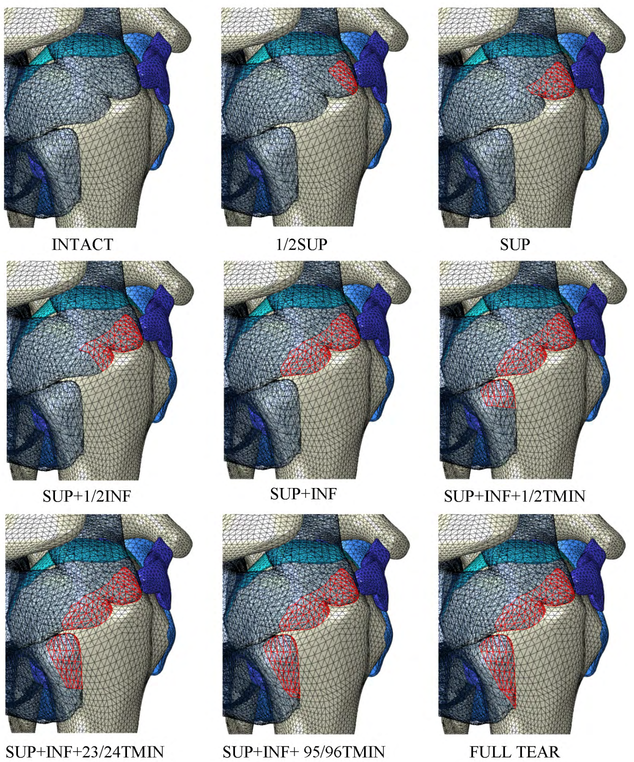

FIGURE 2. Visualisation of the finite element models of the nine rotator cuff tear conditions. (The area in red represents the affected tendon.)

B. NOVEL STABILITY INDEX DEFINITION FOR GH JOINT STABILITY ANALYSIS

A novel integrative stability index was defined to perform the GH joint stability analysis. This stability index is defined as the product of the four stability ratios (i.e., $S = S_N{}^*S_F{}^*S_D{}^*S_C$) that account for the four independent mechanisms that influence the stability of the GH joint. If any of the four stability mechanisms fail, the GH joint stability is lost. It is assumed that each stability ratio contributed equally to the joint stability. The four stability ratios are as follows: (1) S_C: the stability ratio that quantifies the effects of articular conformity on joint stability; (2) S_N: the stability ratio that quantifies the effects of the compressive forces between the articular surfaces on joint stability; (3) S_F: the stability ratio that quantifies the effects of shear force (i.e., the translational forces parallel to the articular surface) between the articular surfaces on joint stability; and (4) S_D: the stability ratio that quantifies the effects of the depth of the humeral head in the glenoid fossa on joint stability. The healthy condition is set as the baseline for analysis of each tear case.

1) S_C: THE EFFECT OF ARTICULAR CONFORMITY ON JOINT STABILITY

The first stability ratio S_C is defined as the ratio between the GH contact area of each tear case, A_i (**i** represents the tear size sequence number), and that of the healthy condition, A_o (**o** represents the cuff being intact); i.e., $S_C = (A_i/A_o)^*100\%$. This ratio quantifies the articular conforming function between the glenoid fossa and the humeral head on joint stability. The underlying mechanism is that a greater area of contact between articular surfaces results in better joint stability. Failure of this mechanism occurs when the GH contact area drops to 0, which indicates GH dislocation.

2) S_N: THE EFFECT OF THE COMPRESSIVE FORCES BETWEEN ARTICULAR SURFACES ON JOINT STABILITY

The second stability ratio S_N is defined as the ratio between the magnitude of the compressive component of the BOBF of each tear case, F_{Ni}, and that of the healthy condition, F_{No}; i.e., $S_N = (F_{Ni}/F_{N0})^*100\%$ (see Figure 3(a)). This ratio quantifies the compressive function of the BOBF on the articular surfaces on joint stability. The underlying mechanism is that a larger compressive component results in better joint stability. Specifically, $F_{Ni} = F_M{}^*\cos\theta_i$, where F_M is the magnitude of the BOBF, and θ_i is the angle between the direction of the BOBF and the normal direction of the glenoid in the corresponding tear case. The normal direction is defined as the line perpendicular to the glenoid fossa at its centroid. Failure of this mechanism occurs when the compressive component of the BOBF drops to 0, which indicates GH dislocation.

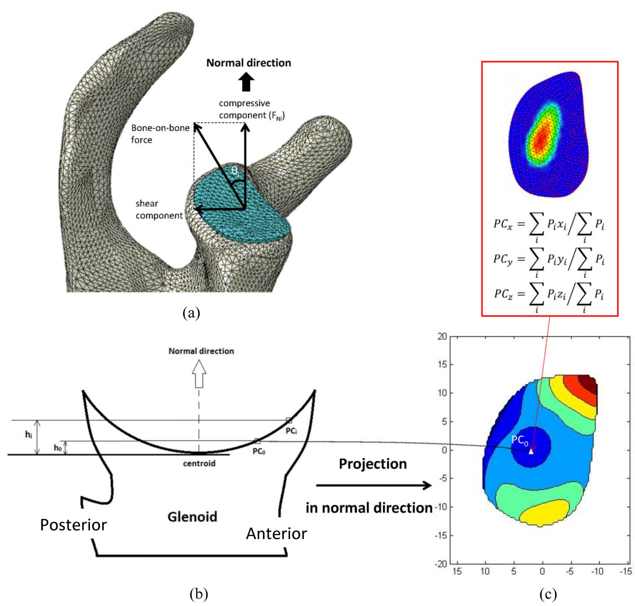

FIGURE 3. Schematic diagram of the stability ratios SN, SF and SD. (a) Glenoid structure and bone-on-bone contact force; (b) schematic of the effective depth; and (c) projection of the glenoid structure in its normal direction (contour line represents the same effective depth).

3) S_F: THE EFFECT OF SHEAR FORCE BETWEEN ARTICULAR SURFACES ON JOINT STABILITY

The third stability ratio S_F is defined as the ratio between the cotangential value of angle θ_i (the quotient of the compressive component over the shear component) in each tear case, $\cot\theta_i$, and that of the healthy condition, $\cot\theta_0$; i.e., $S_F = (\cot\theta_i / \cot\theta_o)^* 100\%$. This ratio quantifies the destabilising function of the shear component of the BOBF on the articular surfaces on joint stability, which is close to the definition in Walia's study [35]. The underlying mechanism is that a closer direction of the BOBF to the glenoid normal direction results in better joint stability. Failure of this mechanism occurs when the direction of the BOBF is perpendicular to the glenoid normal direction, which indicates GH dislocation.

4) S_D: THE EFFECT OF THE DEPTH OF THE HUMERAL HEAD IN THE GLENOID FOSSA ON JOINT STABILITY

The last stability ratio S_D is defined as the ratio between the effective depth (i.e., the distance from the pressure centre and the glenoid centroid in the normal direction of the glenoid) of the healthy condition, H_0, and that of each tear case, H_i; i.e., $S_D = (H_0/H_i)^* 100\%$ (see Figure 3(b)). This ratio quantifies the function of the depth of the humeral head in the glenoid fossa on joint stability. The underlying mechanism is that a deeper pressure centre in the glenoid concavity results in better joint stability. The pressure centre was calculated from the contact pressure area using the equations below [39]:

$$PC_x = \sum_i P_i x_i \Big/ \sum_i P_i$$

$$PC_y = \sum_i P_i y_i \Big/ \sum_i P_i$$

$$PC_z = \sum_i P_i z_i \Big/ \sum_i P_i$$

Where P_i is the contact pressure at node i of the glenoid surface; x_i, y_i and z_i are the coordinates of node i; and PC_x, PC_v and PC_z are the Cartesian coordinates of the contact pressure centre (see Figure 3(c)). Failure of this mechanism occurs when the pressure centre lays outside the glenoid region, where we define S_D as 0, which indicates GH dislocation.

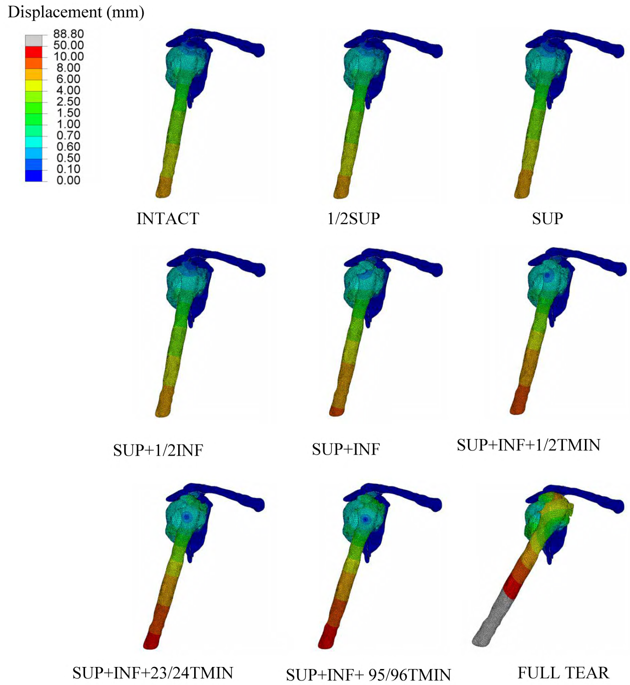

FIGURE 4. Movement of the humerus in the simulation results in each tear case in lateral view.

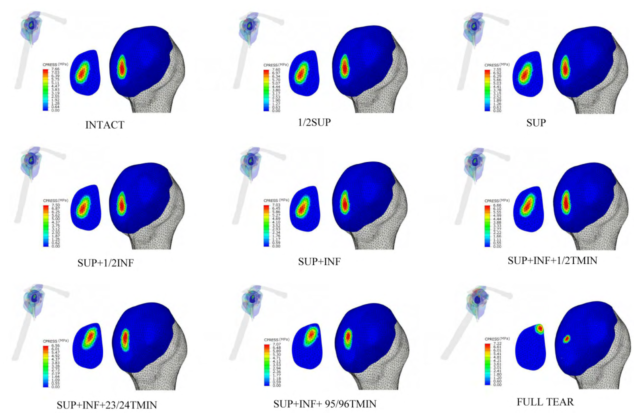

FIGURE 5. Contact pressure distribution at each tear size during rotator cuff tear propagation in the glenohumeral joint open view.

III. RESULTS

A. SIMULATION OF RC TEAR PROPAGATION

FE simulations were conducted successfully for all tear conditions. The results of movement of the humerus can be seen in Figure 4 from the lateral oblique view. Table 3 gives the simultaneously determined compressive component of the BOBF (F_{Ni}), the angle between the BOBF and the glenoid normal direction (θ_i), the effective depth (H_i), the contact area (A_i) and the superior-inferior migration of the humeral centre (U_i) of each tear case, and Figure 5 shows the simultaneous variation of the distribution of the pressures on GH cartilages.

The positional variation of the humerus with detailed superior-inferior migration of the humeral head centre (U_i) is shown in Figure 4 and Table 3. No obvious humeral movement was observed in the first four tear cases in Figure 4. After the tear which comprised of all of the supraspinatus tendon and half of the Infraspinatus tendon (SUP + 1/2INF), this movement became more and more obvious until all three tendons were torn. This humeral head migration also resulted in extension of the humerus in the sagittal plane. U_i also demonstrated the same trend; it was almost unchanged in small tear cases until the tear when half of infraspinatus was torn (SUP + 1/2INF). After the next tear, SUP + INF, the humeral head migration was found to be 0.25 mm; this was followed by dramatic increases and finally reached 6.05 mm when all tendons were torn. This superior-inferior migration result was compared with the results of two *in-vitro* studies [19], [20] and one *in-vivo* study [23] (see Figure 6). The migration of the

TABLE 3. Simulation results of the bone-on-bone contact forces, its direction to the glenoid normal direction, the contact area, and the superior-inferior migration of the humeral centre during the propagation of rotator cuff tears.

Tear size	F_{Ni} (N)	θ_i (o)	H_i (mm)	A_i (mm2)	U_i (mm)
INTACT	408.63	1.12	1.03	88.04	0.00
1/2SUP	411.43	1.15	0.98	89.20	-0.01
SUP	408.53	1.20	1.01	89.61	-0.02
SUP + 1/2INF	402.91	1.21	1.02	88.66	-0.01
SUP + INF	358.21	2.03	1.01	84.90	0.25
SUP + INF + 1/2TMIN	356.83	4.06	1.16	84.40	0.58
SUP + INF + 23/24TMIN	311.45	7.72	1.44	79.02	1.02
SUP + INF + 95/96TMIN	286.54	9.80	1.72	71.10	1.33
FULL TEAR	113.40	7.03	2.75	22.15	6.05

F_{Ni}: compressive component of the bone-on-bone contact force;
θ_i: angle between the bone-on-bone contact force and the glenoid normal direction;
H_i: effective depth;
A_i: contact area;
U_i: superior-inferior migration of the humeral centre.

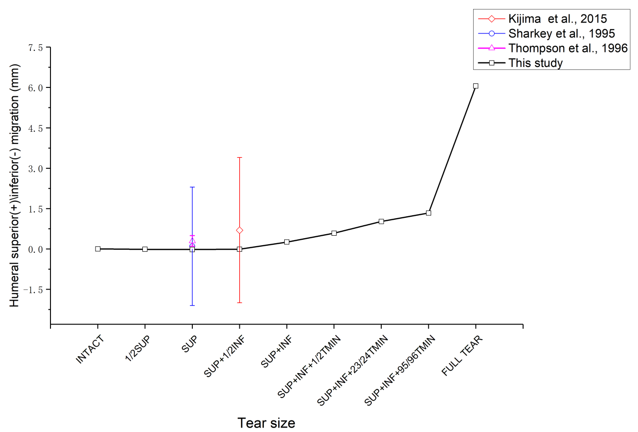

FIGURE 6. Superior-inferior migration of the humeral head centre at each tear size during rotator cuff tear propagation.

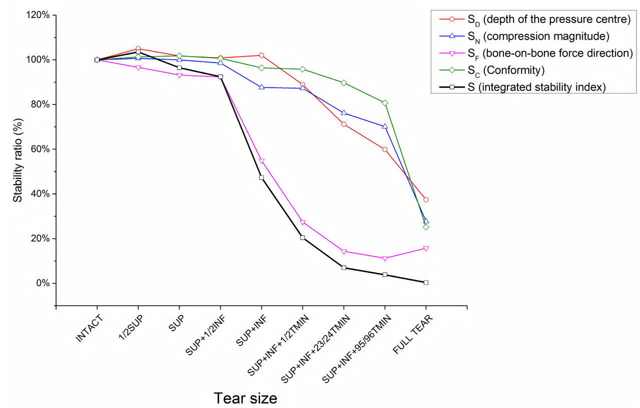

FIGURE 7. Variation in the integrative stability index and individual stability ratios at each tear size during rotator cuff tear propagation.

humeral head centre was found to agree well with the results of previous studies. A similar positional variation trend can also be observed in Figure 5 in the movement of the GH contact area.

B. STABILITY INDEX CALCULATION

A summary of the calculated individual stability ratios and the integrative stability index can be found in Table 4 and Figure 7. In general, the integrative stability index was found to decrease as tear propagation increased, with an exception of half of supraspinatus tear simulation (1/2SUP), where a small increase (3.57%) was found. In addition, the stability index decreased slowly to 92.48% until half of infraspinatus was involved (SUP + 1/2INF) and dramatically afterwards to 0.41% when a full tear occurred. The result of this stability index calculation is consistent with the humeral head migration and contact pressure variation observed above. In addition, the integrative stability index was found to be dominated by the ratio S_F.

IV. DISCUSSION

The objective of this study was to conduct a thorough investigation of the effects of RC tear propagation on GH stability using a previously constructed and validated subject-specific FE model. Simulation results were simultaneously determined for variations in humeral movement, BOBFs and the GH contact area and for the distribution of contact pressure on the GH cartilages. In addition, we designed a novel stability index for critical analysis of the integrated GH stability variation based on the results above.

Superior migration of the humeral head is typically seen in patients with RC tears [12], [19]–[21]. The simulation results of humeral head migration in this study predicted this phenomenon, which supports our hypothesis that the humeral head migrates in a superior direction as the size of the RC tear increases. These results were also found to agree with those of previous studies in which similar RC tear conditions were simulated (see Figure 6). In addition, the tear range when this translation became significant (i.e., SUP + 1/2INF to SUP + INF) is also consistent with clinical observations which stated that tears extended into the infraspinatus tendon resulted in greater humeral migration [6]. Another recent *in-vitro* experiment also determined that the critical tear size for significant changes in the kinematics of the humeral head is a tear of the entire supraspinatus tendon and half of the infraspinatus tendon [16]. In this study eight cadaver shoulders were used in a custom testing system to determine the

TABLE 4. Individual stability ratio and integrative stability index results.

Tear size	S_D	S_N	S_F	S_C	S
INTACT	100.00%	100.00%	100.00%	100.00%	100.00%
1/2SUP	105.00%	100.69%	96.69%	101.32%	103.57%
SUP	101.75%	99.98%	93.21%	101.78%	96.51%
SUP + 1/2INF	100.89%	98.60%	92.32%	100.70%	92.48%
SUP + INF	102.02%	87.66%	54.90%	96.43%	47.35%
SUP + INF + 1/2TMIN	88.95%	87.32%	27.46%	95.87%	20.45%
SUP + INF + 23/24TMIN	71.20%	76.22%	14.35%	89.75%	6.99%
SUP + INF + 95/96TMIN	59.91%	70.12%	11.28%	80.76%	3.83%
FULL TEAR	37.43%	27.75%	15.79%	25.16%	0.41%

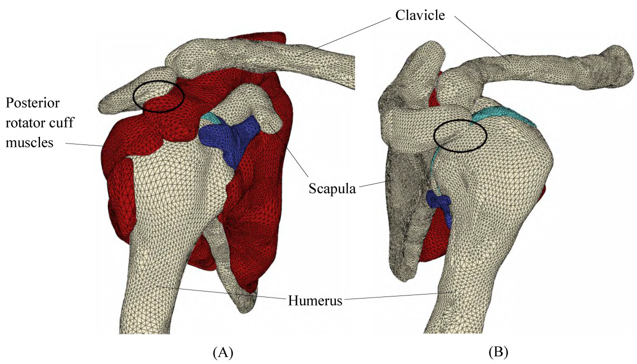

FIGURE 8. Sub-acromion impingement in (A) SUP + INF and (B) FULL TEAR (posterior RC hidden to show bone impingement).

existence of such a critical RC tear size and reach the same finding as it is in this study. In addition, the humeral head was found to migrate in both anterior and superior directions. This anterior migration was also consistent with the findings of a recent *in-vivo* ultrasonographic measurement study that stated that the posterior cuff stabilises the humeral head via tethering the anterior migration of the humeral head [40]. Tearing of the posterior cuff as in this study would result in anterior migration of the humeral head. Finally, sub-acromion impingement, which is a common shoulder disorder associated with RC tears [41], was also observed in massive RC tear conditions. As shown in Figure 8, starting from SUP + INF, the supraspinatus tendon was found to be pressed by the decreased sub-acromion space till full tear when the humerus showed a slight collision with the acromion. This shows that this model might have potential in the investigation of sub-acromion impingement, although it is beyond the scope of this study.

A quantitative analysis of the variations in GH stability during the propagation of RC tears was successfully conducted with the integrative stability index designed in this study. The results support our hypothesis that the stability of the GH joint generally decreases as the size of the tear increases (see Figure 7). The integrative stability index was found to be dominated by S_F, the ratio that quantifies the effects of shear force between articular surfaces on joint stability, which indicates that the mechanism by which RC tear propagation affects GH joint stability is via variation of the resultant BOBF direction. This finding does not support a previous opinion that the proximal humeral migration is caused by the increased deltoid upward muscle force [42], [43]. In this study, the deltoid muscle force remained unchanged, but the humeral head was still found to migrate in a posterior direction. Therefore, it is believed that variation in the BOBF direction can also cause this migration.

The critical tear size is controversial. Factors influencing this appear to be subject-dependent and associated with age, trauma and the critical shoulder angle [44]. Patients with the same tear size could be either asymptomatic or symptomatic [23]. This study shows that in an isolated supraspinatus tear there is no demonstrable glenohumeral joint instability, which is in agreement with previous clinical observations and cadaveric results [9], [45], [46]. The stability analysis shows that the critical tear size for this subject was a tear which involved at least half of the footprint width of the infraspinatus tendon. This critical tear size does correlate with previous clinically observed *in-vivo* kinematics patterns determined in patients with known massive tears, which showed stable GH kinematics in patients with tears of the superior RC and unstable GH kinematics in patients with tears that involved the entire superior and posterior RC [21]. The same critical tear size was determined in an *in-vitro* study [16], although it focused on the use of the kinematics of the humeral head rather than the combined functions to evaluate GH joint stability. Further studies, using our model, could be done in other subjects or different tear configurations, such as the incidence of subscapularis tears, to determine the specific parameters that influence the critical tear size.

The proposed integrative stability index successfully accounted for both the concavity compression and scapulohumeral balance mechanisms, as proposed by Lippitt et al. [33]. Specifically, the combination of S_D and S_N is equivalent to and completely defines the compression concavity mechanism, whilst S_F defines the scapular scapulohumeral balance mechanism. Furthermore, beyond these two mechanisms, the joint conformity mechanism was included by S_C. More importantly, as seen in this study, this stability ratio could be used to examine the fundamental mechanism by which the GH joint stability was influenced in stability analyses and the degree of this influence, thus leading to improved diagnostic or surgical treatment strategies.

This stability index is also flexible and convenient to use. It could also be used in other GH joint stability studies or compound biomechanical studies. Firstly, each ratio could be used independently, even if insufficient data are available to calculate all four ratios. Secondly, the definition of each ratio can be revised according to the specific conditions. For example, if applied to the study of bony Bankart lesions, in which the geometry of the glenoid fossa is altered, the stability ratio S_D could be revised according to the defective geometry of the glenoid fossa. Finally, for practical use in clinical applications, calculation of the ratios for different conditions need not use the healthy condition as the baseline, because such a condition is unlikely to be available for most patients. Instead, the baseline can be varied for each individual ratio based on the relevant available data. For example, the normalisation of S_C can use the total glenoid cartilage area, and that of S_F can use the glenoid normal direction as the baseline. In addition, the computation of this stability index does not require that the simulation or experiment be performed until failure is demonstrated. This flexibility and convenience could make this index suitable for studies to evaluate the GH joint.

This study also has several limitations in addition to those inherent in the FE model. First, this study did not consider the effects of variations of muscle forces, which are likely to change due to pathological or morphological changes of the tendon or alterations in muscle contractions due to pain (neural control). Second, the RC tears were assumed to be detached from the tendon's osseous insertion sites. Therefore, the RC tendons remained connected, so that even if the SUP and/or infraspinatus tendon were completely detached from the humeral head, the relative muscle forces can still be transmitted via the remaining tendons until complete tears occur in all three posterior RC tendons. However, in the presence of a massive longitudinal tear along the orientation of the RC fibres, such as an L-shaped RC tear [47], this force transmission can be disrupted. In addition, due to the limitation in the current computational power and cost, the proposed method in this study may not be suitable to be used in real-time experiments. Finally, the biomechanical investigation was conducted in a static position with a relatively low abduction angle. It remains unknown whether the results above are consistent with the humerus abducted to a higher angle or in combination with humeral rotation.

This study thoroughly investigated the propagation of RC tears using a previously constructed and validated FE shoulder model. An original novel comprehensive stability index was proposed and used to quantify the variation in GH stability during the propagation of RC tears. Further studies could involve the effects of RC tears in combination with subscapularis tears, patient-specific case studies for greater validation and accurate examination of the simulation results.

V. CONCLUSIONS

A biomechanical investigation of the effects of the propagation of RC tears on the stability of the GH joint was conducted based on a previously constructed and well-validated FE model of the shoulder complex. The results of simultaneously determined BOBFs, contact area, pressure distribution and the superior-inferior migration of the humeral centre were found to be comparable with *in-vitro* and/or *in-vivo* measurements. A novel integrative stability index was proposed and used to quantify the effects of RC tear propagation on GH joint stability. It was found that (1) the stability of the GH joint generally decreases as the tear size increases; (2) smaller tears do not significantly affect the joint stability, and the critical tear size that leads to a loss of normal shoulder biomechanics was determined as a half tear of the infraspinatus accompanied by a complete tear of the supraspinatus tendon for this subject; and (3) the main mechanism by which a RC tear destabilises the GH joint is the change of direction of the BOBF. These findings are consistent with previous cadaveric results and clinical observations. This study further validated the model we constructed in first part of this study. The results obtained may lead to a better understanding of the rationale of RC tears and hence may be used to improve diagnostic and therapeutic strategies for clinicians.

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