Critical glenoid defect size leading to persistent anterior shoulder instability despite Bankart repair

Abstract:

Objectives: Anterior glenoid osseous defects are commonly associated with recurrent anterior shoulder instability. Once the defect reaches a critical size, joint stability is restored through treatment with bone-grafting. Prior work has quantified the critical defect size under non-physiological loading conditions to be 20% of the glenoid length. As the stability of the shoulder is load dependent, with higher joint forces leading to a loss in stability, the aim of this study was to determine the critical size of an anterior glenoid osseous defect that leads to persistent anterior shoulder instability under physiological joint loading despite Bankart repair.

Methods: Two Finite Element (FE) models were utilised to determine the risk of shoulder dislocation during 30 functional activities for the intact glenoid and after creating anterior osseous defects of increasing magnitudes. A Bankart repair was simulated for each osseous defect size, and the shoulder was tested under loading conditions that replicate in-vivo shoulder forces during these functional tasks. The critical lesion size was defined as the smallest osseous defect size that leads to shoulder dislocation.

Results: The FE models demonstrate a high risk of shoulder dislocation during functional activities post Bankart repair for anterior osseous defects corresponding to 16% of the glenoid length.

Conclusions: This computational study suggests that an anterior glenoid osseous defect of 16% of the glenoid length or greater may be considered for bone grafting rather than solely a soft tissue stabilisation procedure, in order to optimise joint stability through concavity compression.

Keywords: Glenoid bone loss, shoulder stability, bone grafting, Bankart repair, finite element analysis

Word count: 3986 words

Introduction:

Anterior glenoid osseous defects are commonly associated with recurrent anterior shoulder dislocation with the prevalence of fracture or erosion of the glenoid rim being reported to range from 8 (18 of 226) to 73 percent (116 of 158) of shoulders with recurrent anterior instability[1,2]. As osseous defects may result in a loss of glenoid concavity, the shoulder becomes unstable in the mid-range of motion where stability is achieved through compression of the humeral head into the glenoid labral socket through rotator cuff muscle co-contraction[3]. Biomechanical studies have demonstrated an inverse relationship between the size of the defect and shoulder stability: the larger the osseous defect, the less stable the shoulder[3–5].

The outcomes of surgical treatment of recurrent anterior shoulder instability with arthroscopic Bankart repair in patients with large glenoid osseous defects are inferior to patients with bone-grafting in terms of recurrent instability[6,7]. The rationale for not performing bone grafts on all patients with glenoid defects include concerns related to complication rates, long term sequelae and surgical learning curves[8]. Therefore, defining the critical bone lesion size that leads to persistent anterior shoulder instability under loading conditions expected by the patient during functional activities will assist clinical decision making[9].

Currently, only three biomechanical studies from one group have quantified the critical size of an osseous defect that necessitates bone-grafting[6,7]. These studies found that for glenoid osseous defects that correspond to 19-21% of the glenoid length, a Bankart repair is insufficient to maintain shoulder stability and that joint stability through concavity compression is restored after reconstruction of the glenoid concavity by bone-grafting. Clinical guidelines have taken these data as recommendation for bone-grafting[10].

55 The results above are derived from physical cadaveric experiments and suffer from
56 limitations, including non-physiological loading conditions involving a maximum joint load
57 of 50 N, where *in-vivo* data from instrumented shoulder implant measurements has reported
58 151% body weight loading during activities of daily living[11]. As the stability of the shoulder
59 is load dependent, with higher joint forces leading to a loss in stability[12,13], the use of
60 physiological loading conditions is an essential requirement for the assessment of shoulder
61 stability.

62 Finite Element modelling represents an efficient method to overcome this limitation of
63 cadaveric studies and to simulate experimental testing under *in-vivo* loading conditions.
64 Therefore, the aim of this study was to determine the critical size of an anterior glenoid
65 osseous defect that leads to persistent anterior shoulder instability under physiological
66 loading conditions despite Bankart repair. This has the potential to assist surgical decision-
67 making for patients with recurrent anterior shoulder instabilities.

68

Methods:

Development of an Intact Finite Element Model

Detailed information about the development and validation of two subject-specific finite element (FE) models of the shoulder is provided in a previous study[13]. Briefly, two right shoulder geometries were obtained from high-resolution physical slices of the male and female Visible Human datasets[14]. The glenoid fossa at the lateral angle of the scapula, the proximal humerus, the articulating cartilage surfaces as well as the glenoid labrum were manually segmented using Mimics (Mimics Research 17.0, Materialise, Leuven, Belgium). The segmented structures were converted to triangular surface meshes to form a 3-dimensional model of the intact shoulder. Local coordinate systems were assigned to the articulating structures to position and orient the humerus with respect to the glenoid fossa[15]. The surface meshes were imported into Finite Element (FE) analysis software (Marc Mentat 2015.0.0, MSC.Software, Palo Alto, USA). The articulating bones, the articular cartilage surfaces and the glenoid labrum were modelled as tetrahedral solid elements. The glenoid labrum was divided into eight sections in order to obtain an anatomically accurate representation of the tissue[16]. Each labral section was assigned a coordinate frame to define the local fibre orientation[17].

Baseline material properties for each structure of the shoulder were assigned based on literature values (Table 1)[16–18]. Due to relatively small deformations compared to other soft tissues, the articulating bones were modelled as rigid materials. The articular cartilage surfaces were assigned linear elastic isotropic properties, while the labrum was modelled as a transversely isotropic hyperelastic material due to the modulus difference in the transverse plane and the circumferential direction[16]. The material coefficients for the hyperelastic model

were obtained by applying the neo-Hookean constitutive equation to experimentally derive material properties for each labral section[19].

[Table 1 near here]

The interfaces between the articulating cartilage surfaces and between the humeral cartilage and the labrum were modelled using frictionless, surface-to-surface contact due to the low friction coefficient in synovial joints[17]. The interfaces between the articulating bones and the corresponding cartilage surfaces were modelled using tied contact.

In a previous study, the FE models of the shoulder were validated against *in-vitro* measurements of shoulder stability for multiple loading conditions and joint positions as reported by two different cadaveric studies[12,13].

Development of Glenoid Osseous Defects

Glenoid osseous defects of 4%, 8%, 12%, 16%, 20% and 24% of the glenoid length were created separately at the anterior glenoid rim. These chronic osseous defect models were created in agreement with cadaveric testing as reported in the literature (Figure 1)[3–5]. The anterior osteotomy lines were drawn parallel to the line passing through superior and inferior contact points of the circumcircle that fits the superoinferior diameter of the glenoid, with the 0% osteotomy line being tangential to the anterior rim of the glenoid[4,5]. The glenoid osseous defects were introduced into the FE models by removing finite elements of the glenoid fossa and the glenoid articular cartilage that correspond to each osteotomy line.

A soft tissue Bankart repair was simulated for each glenoid defect size. This was achieved through the re-attachment of the labrum to the osteotomy line, in order to re-establish the level of the glenoid concavity. This was modelled by displacing the medial surface nodes of

115 the labrum towards the osteotomy line until the labrum was fully aligned and lifted to the
116 level of the osteotomy line. The medial nodes of the glenoid labrum were fixed in that
117 position during further simulations assessing joint stability, in order to simulate labral healing
118 to the osteotomy line as it can be observed *in-vivo* following successful shoulder
119 rehabilitation.

120 The other parts of the intact FE models were not changed. Hill-Sachs lesions were not
121 modelled in this study.

122 [Figure 1 near here]

123

124 **Validation of a Finite Element Model with Glenoid Osseous Defect**

125 The FE models of the shoulder with glenoid osseous defects were validated against *in-vitro*
126 measurements of anterior shoulder stability[4,5]. These cadaveric measurements represent the
127 only data available in the literature that assess the loss in anterior joint stability with anterior
128 glenoid osseous defects of different magnitudes[4,5]. For the validation study, the humerus was
129 positioned in the shoulder dislocation position with 45° of abduction and 35° of external
130 rotation in order to replicate these cadaveric experiments[4,5]. In the starting position, the
131 humeral head was in contact with and centred on the glenoid socket (0° of glenoid version
132 and inclination). With the glenoid surface being fixed in all degrees of freedom during the
133 simulation, the boundary conditions involved the application of a 50 N compressive force
134 through the centre of the humeral head, perpendicular to the plane of the glenoid articulating
135 surface, in order to simulate the joint loading as applied in the cadaveric studies. Under
136 permanent joint compression, the humeral head was subsequently translated towards the
137 anterior glenoid rim until dislocation occurred. The maximum force required to dislocate the
138 shoulder was defined as the dislocation force. The dislocation forces were determined for the

intact glenoid and after introducing anterior glenoid osseous defects of 8%, 16% and 24% of the glenoid length to validate modelling results against *in-vitro* measurements as reported in the literature[5].

Quantifying the Critical Size of a Glenoid Osseous Defect

The critical size of an anterior glenoid osseous defect was investigated during 30 functional activities by determining the dislocation forces of the intact glenoid and after creating anterior glenoid osseous defects of 4%, 8%, 12%, 16%, 20% and 24% of the glenoid length respectively. For each functional activity that was simulated, the dislocation forces were determined in the joint position most susceptible to instability as quantified in a previous study[20], where the ratio of shear to joint compression force was maximum. Furthermore, the dislocation forces for each functional daily activity were determined under *in-vivo* loading conditions that were quantified in a previous study, where *in-vivo* shoulder contact forces were analysed during 26 functional daily tasks[20]. For the purpose of this study, 4 throwing sport activities were added using the same experimental approach as presented in Klemt et al. (2018)[20].

In order to quantify the critical lesion size of an anterior glenoid osseous defect that leads to persistent anterior shoulder instability despite Bankart repair, the shoulder dislocation forces for each functional daily activity as obtained by the FE models were compared to *in-vivo* shoulder shear forces during these functional daily activities (ADL) as quantified by the UK National Shoulder Model (UK NSM) in a previous study (Figure 2)[20]. Based on the comparison of *in-vivo* shear force with shoulder dislocation forces for each functional task, each ADL was classified for each glenoid defect size as stable (dislocation force of the glenoid is larger than the *in-vivo* shear force of the ADL) or unstable (dislocation force of the

glenoid is smaller than the *in-vivo* shear force of the ADL). Finally, the critical size of an anterior osseous defect was quantified as the smallest glenoid osseous defect size that leads to anterior shoulder dislocation across all functional daily activities.

[Figure 2 near here]

Results:

In agreement with cadaveric experiments, the dislocation forces determined by the male and female FE models demonstrate a decrease in shoulder stability with increasing size of the glenoid osseous defect[4]. The dislocation forces quantified by the FE models compare well to those experimentally measured with model predictions being within one standard deviation of experimental values (Figure 3).

[Figure 3 near here]

The classification of all ADLs with respect to joint stability for each anterior glenoid osseous defect size is shown in Table 2, with the corresponding dislocation forces and joint positions that yielded this classification being demonstrated in Table 3 and 4. As can be seen in Table 2, the critical size of an anterior glenoid defect that leads to persistent anterior shoulder instability under physiological joint loading despite Bankart repair was predicted by the FE models to be 16% of the glenoid length.

There are 4 loading conditions with high anterior shear forces such as a quarterback throw, lineout throw as well as reaching across the body and cleaning the upper back that demonstrate a high likelihood of instability for the glenoid with an anterior osseous defect of 16% of the glenoid length (Table 2). This is due to *in-vivo* shear forces for these two functional tasks that exceed the anterior dislocation forces of the glenoid with an anterior osseous defect corresponding to 16% of the glenoid length (Table 3). There are four functional daily activities with large abduction and external rotation ranges of motion such as abduction (slow/fast), reaching the back of the head and a volleyball serve that show a great risk of instability for the glenoid with an anterior osseous defect of 20% of the glenoid length (Table 2, 3). The majority of functional daily tasks such as eating, drinking, reaching opposite axilla, flexion, driving and a basketball throw affect joint stability only for anterior osseous

defects of 24% of the glenoid length or greater (Table 2). These functional activities involve smaller abduction and external rotation ranges of motion as well as loading conditions, with anterior *in-vivo* shear forces for these tasks not exceeding the anterior dislocation forces for anterior osseous defects of 24% of the glenoid length or greater (Table 2, 3, 4).

[Table 2 near here]

[Table 3 near here]

[Table 4 near here]

Discussion:

In this study, two FE models of the shoulder with anterior glenoid osseous defects of different magnitudes were validated against *in-vitro* measurements of anterior shoulder stability[4]. The anterior dislocation forces predicted by the FE models were within one standard deviation of experimentally measured values. The validated FE models quantified the critical size of an anterior glenoid osseous defect that leads to persistent anterior shoulder instability under physiological loading, despite Bankart repair, to be 16% of the glenoid width. Therefore, this modelling study suggests that if there is an osseous defect of such size, bone-grafting may be considered rather than solely a soft tissue stabilisation procedure to optimise joint stability through concavity compression.

Previous biomechanical studies showed that slightly larger anterior glenoid osseous defects of 19-21% of the glenoid length were required to produce a significant loss in anterior shoulder stability[3–5]. These results have clinically been used as an indication for bone-grafting to restore joint stability[10], despite these *in-vitro* experiments being conducted under low non-physiological loading conditions. As the stability of the shoulder is load dependent, with higher joint forces leading to a loss in stability[12,13], the results of the FE models here strongly suggest that slightly smaller anterior glenoid osseous defects of 16% of the glenoid length lead to persistent anterior shoulder instability under physiological joint loading despite Bankart repair. Similar findings have recently been presented in the literature[21,22], with these *in-vitro* studies demonstrating significant changes in joint functionality following creation of anterior glenoid osseous defects corresponding to 13.5 to 15% of the glenoid length. When considering the clinical relevance of this study, it should be noted that recurrent instabilities are commonly observed in clinical practice during sport participation rather than functional activities of daily living[23,24]. While this study includes basic throwing sports to demonstrate the critical size of an anterior glenoid osseous defect that leads to persistent anterior shoulder

instability under physiological loading despite Bankart repair, we acknowledge that further inclusion of contact and impact sports has the potential to decrease the critical lesion size due to the excessive shoulder loading during these sporting events. However, such data are not available due to the inherent difficulty in obtaining shoulder loading during those sporting incidents, with existing studies in the literature not providing details of anterior shoulder shear forces during tackles in contact sports[25]. When considering the clinical importance of this study, it should be noted that bone-grafting yields better clinical outcomes in terms of recurrent instability[6,7], when compared to a Bankart repair, therefore performing bone-grafting on smaller osseous defect sizes will decreases rates of recurrent anterior shoulder instability. However, bone-grafting procedures are associated with an increased surgical invasiveness, surgical complication rates and longer rehabilitation periods[8,26], necessitating a treatment selection on a patient-specific basis. As the study findings demonstrated a high likelihood of instability for the glenoid with an anterior osseous defect of 16% of the glenoid length during activities with large anterior shear forces at large abduction and external rotation ranges of motion, slightly smaller anterior glenoid osseous defects may be considered for bone-grafting in patients with great functional demands. Glenoid bone loss can precisely be determined from Computed Tomography with an accuracy of below 3% of the glenoid length[27], thus this study finding has the potential to support clinical decision-making by suggesting that slightly smaller anterior osseous defects of 16% of the glenoid length should be reconstructed to restore joint stability. As Hill-Sachs lesions were not investigated in this study, the findings are only representative for anterior glenoid osseous defects in the absence of engaging Hill-Sachs lesions.

A novel factor in this study is the use of joint loading in terms of *in-vivo* shoulder contact forces (compressive and shear) that have been quantified by musculoskeletal modelling for a wide range of activities of daily living[20,28]. The comparison of *in-vivo* shear forces with the

predicted glenoid dislocation forces allows for a mechanical assessment of joint stability through classification of each ADL as stable (dislocation force of the glenoid is larger than the *in-vivo* shear force of the ADL) or unstable (dislocation force of the glenoid is smaller than the *in-vivo* shear force of the ADL) for each for each glenoid defect size. This mechanical assessment of joint stability to quantify the critical glenoid defect size that leads to persistent anterior shoulder instability despite Bankart repair represents the first approach of its kind, with previous *in-vitro* studies[3–5] utilising statistical significant changes in joint stability as predictor for the critical lesion size of a glenoid osseous defect.

This study has limitations in that the anatomy of the glenoid osseous defects were idealised to being parallel to the longitudinal axis for anterior osseous defects in order to replicate *in-vitro* experiments. This does not represent all *in-vivo* defects, but it has been demonstrated that the margin of a glenoid defect is linear in most cases, with most of these defects being located at the anterior glenoid rim[29]. Also, the defects were created in increments of 4% of the glenoid length, thus representing a different proportion of loss for scapulae of different sizes[30]. This has been partially taken into account in this study through the use of one large and one small scapula. In addition, the dislocation forces for each functional daily activity were determined in the joint position most susceptible to instability, without considering movement of the humeral head during arm motion. As the humeral head remains mainly centred on the glenoid fossa with humeral head translations of only a few millimetres during functional daily activities, the effect of this simplification is assumed to be small.

The stability provided by the bony articulating structures is only one stabilising mechanism of the shoulder. Other mechanisms such as the capsuloligamentous structures need to be considered in future studies. Therefore, the results presented here are representative only for the mid-range of shoulder motion where the capsuloligamentous structures are lax. Despite these limitations, this is the first study to determine the critical size of an anterior glenoid

277 osseous defect that leads to persistent anterior shoulder instability under physiological
278 loading conditions despite Bankart repair. This study suggests why shoulder instability
279 persists in some patients who have had soft tissue stabilisation only, and proposes bone
280 grafting for anterior glenoid osseous defects of 16% of the glenoid length.

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Supporting data are available on request by contacting the corresponding author.

Conflict of interest:

The authors declare no conflict of interest.

Table 1: Baseline material properties for the finite element model of the glenohumeral joint. E = Young's Modulus, υ = Poisson's Ratio, $C_{1\text{-}8}$ = Hyperelastic Labral Coefficients.

Anatomy	Material Type	Parameter	Value	Reference
Humerus	Rigid	-	-	Terrier et al. (2007) [30]
Humeral Cartilage	Isotropic Elastic	E υ	10 MPa 0.4	Buchler et al. (2002) [7]
Glenoid	Rigid	-	-	Terrier et al. (2007)
Glenoid Cartilage	Isotropic Elastic	E υ	10 MPa 0.4	Buchler et al. (2002)
Labrum	Transversely Isotropic Hyperelastic	C_1 C_2 C_3 C_4 C_5 C_6 C_7 C_8	3.4 5.4 7.0 5.9 5.2 4.8 5.7 4.3	Smith et al. (2008) [28]

399 Table 2: Classification of each anterior glenoid osseous defect size for each ADL as unstable
400 (dark grey) or stable (white). The defect sizes correspond to 4%, 8%, 12%, 16%, 20% and
401 24% of the glenoid length respectively.

	Anterior Glenoid Osseous Defect						
	Intact	4%	8%	12%	16%	20%	24%
Quarterback Throw							
Lineout Throw							
Extreme (Reach Across Body)							
Clean Upper Back							
Volleyball Serve							
Slow Abduction							
Fast Abduction							
Reach Back of Head							
Basketball Free Throw							
Sit to stand							
Pull							
Push							
Lift Block to Head Height							
Lift Block to Shoulder Height							
Reach Opposite Axilla							
Lift Shopping bag from Floor							
Lift Shopping Bag on Lap							
Perineal care							
Reach far Ahead							
Brush Left Side of Head							
Drive Slow Left							
Drive Slow Right							
Drive Fast Left							
Drive Fast Right							

Slow Flexion							
Fast Flexion							
Eat with Hand							
Drink from Mug							

Table 3: Anterior dislocation forces of the intact glenoid and after creating anterior osseous defects of 4%, 8%, 12%, 16%, 20% and 24% of the glenoid length, as well as the in-vivo anterior shear force for each functional task as quantified in a previous study.[26] The dislocation forces were tested in the joint position most susceptible to instability.

	Anterior Dislocation Forces [N]							In-vivo Shear Forces [N]
	Intact	4%	8%	12%	16%	20%	24%	
Quarterback Throw	632	595	557	522	489	397	294	516
Lineout Throw	453	415	393	366	342	274	220	359
Extreme (Reach Across Body)	103	95	89	84	78	64	49	81
Clean Upper Back	98	90	85	81	74	60	46	79
Volleyball Serve	324	294	266	241	210	153	112	201
Slow Abduction	180	162	148	132	119	87	61	112
Fast Abduction	174	161	144	128	114	85	54	108
Reach Back of Head	110	106	102	96	90	74	57	84
Basketball Free Throw	278	254	236	215	196	142	116	132
Sit to stand	437	401	369	313	281	193	120	171
Pull	86	82	78	73	68	53	41	48
Push	85	80	76	72	67	54	41	46
Lift Block to Head Height	204	190	176	159	147	106	89	98
Lift Block to Shoulder Height	197	184	171	155	141	98	83	93
Reach Opposite Axilla	82	77	72	67	63	51	44	47
Lift Shopping bag from Floor	180	171	160	149	137	99	83	80
Lift Shopping Bag on Lap	212	197	183	171	158	127	86	83
Perineal care	108	103	98	91	85	71	58	51
Reach far Ahead	190	175	163	151	139	112	74	66

Brush Left Side of Head	109	105	101	96	90	74	57	52
Drive Slow Left	109	105	100	94	88	71	56	51
Drive Slow Right	111	108	103	96	91	75	60	49
Drive Fast Left	102	97	91	85	79	65	49	43
Drive Fast Right	99	95	89	84	79	65	49	43
Slow Flexion	202	190	178	163	149	118	77	68
Fast Flexion	189	180	167	151	138	107	72	66
Eat with Hand	85	80	76	71	65	52	44	39
Drink from Mug	83	79	75	70	63	51	42	37

443 Table 4: Joint positions that served to determine the anterior dislocation forces. These joint
444 positions were demonstrated to be most susceptible to instability due to the largest ratio of
445 shear to joint compression force. The shoulder angles represent forward flexion (Flex,
446 positive sign indicates forward flexion), abduction (Abd, positive sign indicates abduction)
447 and rotation (Rot, positive sign indicates internal rotation).

448

Functional Activity	Shoulder Position [°]		
	Flex	Abd	Rot
Quarterback Throw	94	71	-39
Lineout Throw	75	-61	-53
Extreme (Reach Across Body)	111	60	-48
Clean Upper Back	-45	49	-41
Volleyball Serve	107	54	-37
Slow Abduction	120	83	-74
Fast Abduction	102	70	-71
Reach Back of Head	67	-44	-56
Basketball Free Throw	86	59	-47
Sit to stand	-12	-14	-4
Pull	12	8	-9
Push	21	3	-12
Lift Block to Head Height	70	27	-32
Lift Block to Shoulder Height	52	22	-24
Reach Opposite Axilla	60	23	23
Lift Shopping bag from Floor	-7	-12	-14
Lift Shopping Bag on Lap	29	-9	-22
Perineal care	-19	-5	8
Reach far Ahead	76	24	-27
Brush Left Side of Head	60	16	19
Drive Slow Left	39	-2	-19
Drive Slow Right	27	2	-14
Drive Fast Left	33	-3	-17
Drive Fast Right	20	-4	-10
Slow Flexion	114	42	-37
Fast Flexion	98	39	-35

Eat with Hand	34	-7	-18
Drink from Mug	24	-18	-22

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453 **List of figure legends:**

454 Figure 1: Simulated anterior osteotomy lines, at 4%, 8%, 12%, 16%, 20% and 24% of the
455 glenoid width respectively were drawn parallel to the longitudinal axis of the glenoid.

456

457 Figure 2: Overview of the study design.

458

459 Figure 3: Dislocation forces of the shoulder for the intact glenoid and after creation of
460 anterior glenoid osseous defects that correspond to 8%, 16% and 24% of the glenoid width.
461 The dislocation forces represent cadaveric experiments (Yamamoto et al, 2009)[4] as well as
462 simulation results obtained in this study (Male and Female Visible Human dataset). The bars
463 represent one standard deviation from the cadaver *in-vitro* values.