



# The role of bone in glenohumeral stability

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- Shoulder stability depends on several factors, either anatomical or functional. Anatomical factors can be further subclassified under soft tissue (shoulder capsule, glenoid rim, glenohumeral ligaments etc) and bony structures (glenoid cavity and humeral head).
- Normal glenohumeral stability is maintained through factors mostly pertaining to the scapular side: glenoid version, depth and inclination, along with scapular dynamic positioning, can potentially cause decreased stability depending on the direction of said variables in the different planes. No significant factors in normal humeral anatomy seem to play a tangible role in affecting glenohumeral stability.
- When the glenohumeral joint suffers an episode of acute dislocation, either anterior (more frequent) or posterior, bony lesions often develop on both sides: a compression fracture of the humeral head (or Hill–Sachs lesion) and a bone loss of the glenoid rim. Interaction of such lesions can determine ‘re-engagement’ and recurrence.
- The concept of ‘glenoid track’ can help quantify an increased risk of recurrence: when the Hill–Sachs lesion engages the anterior glenoid rim, it is defined as ‘off-track’; if it does not, it is an ‘on-track’ lesion. The position of the Hill–Sachs lesion and the percentage of glenoid bone loss are critical factors in determining the likelihood of recurrent instability and in managing treatment.
- In terms of posterior glenohumeral instability, the ‘gamma angle concept’ can help ascertain which lesions are prone to recurrence based on the sum of specific angles and millimetres of posterior glenoid bone loss, in a similar fashion to what happens in anterior shoulder instability.

**Keywords:** glenoid track; instability; shoulder

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## Anatomy and biomechanics

Glenohumeral stability is a multifactorial process whose balance is guaranteed by several structures, of which bone

is one. The inherent discrepancy between the size of the humeral head and the scapular glenoid fossa<sup>1</sup> allows for a wide range of motion (ROM), but demands effective stabilizers in order to avoid dislocation of the humeral head from its natural position in the glenoid cavity. Said stability is dependent on several anatomical and biomechanical factors: the relationship between the humeral head and the scapula in the different positions of the arm; the integrity of the bony structures and soft tissues; the static and dynamic neuromuscular balance of the muscles surrounding the joint.

Bone and soft tissue both interact to provide adequate stability at different ROMs. With the arm at rest, the weight of the arm hanging loose generates a negative static intra-articular pressure of around –30 mm Hg, which acts as the principal stabilizer of the glenohumeral joint, mainly preventing inferior dislocation.<sup>2,3</sup> The long head of the biceps tendon,<sup>4</sup> the rotator interval capsule and the coracohumeral ligament,<sup>5</sup> located in an antero-superior position relative to the humeral head, add stability: the former by acting as a ‘rein’ to prevent humeral head migration in different directions (mainly antero-inferiorly), the latter by maintaining negative intra-articular pressure.

As the arm moves along the different planes, more factors come into play depending on the considered ROM, namely the mid-range and the end-range. Itoi et al<sup>6</sup> defined the ‘end-range’ as the ROM performed when the arm comes to the limit of shoulder movement. By doing this, a large circle forms around the shoulder joint. The area surrounded by this circle is called the ‘mid-range’ of movement. In this area, the role of the negative intra-articular pressure subsides,<sup>7</sup> the active compression of the rotator cuff muscles (specifically, supraspinatus, subscapularis, and infraspinatus) and the middle portion of the deltoid (partially)<sup>8</sup> push the humeral head against the centre of the glenoid. This mechanism is defined as concavity-compression<sup>9</sup> and is an important factor which further stabilizes the shoulder at the mid-range of movement.

As the shoulder joint approaches the end-range, the glenohumeral capsule and ligaments (superior, middle and inferior) act as the main stabilizers of the shoulder

joint.<sup>10–13</sup> With the arm in abduction and external rotation, the anterior portion of the capsule and the inferior glenohumeral ligament (IGHL), namely its anterior band, become taut and play a major role in preventing the humeral head from dislocating anteriorly.<sup>14</sup> The middle glenohumeral ligament (MGHL) serves a similar function by reaching its highest tension at 30–45 degrees of abduction and external rotation,<sup>15</sup> while the superior glenohumeral ligament (SGHL) becomes tight in adduction and external rotation, thus functioning as an inferior stabilizer.<sup>16</sup> Finally, the posterior band of the IGHL prevents posterior dislocation with the arm in flexion and internal rotation.<sup>14</sup>

### Glenohumeral stability: the ‘bony factors’

When focusing solely on the bone, it is useful to look at the two individual ends of the shoulder joint (the glenoid cavity and the humeral head) and the relative structural variables which can influence stability, and then analyse how these factors interact under normal and pathological circumstances. This way, a systematic approach can be used when evaluating the unstable shoulder in clinical practice, by understanding the role of bone abnormalities (either acquired or congenital) and treating them accordingly, if deemed necessary.

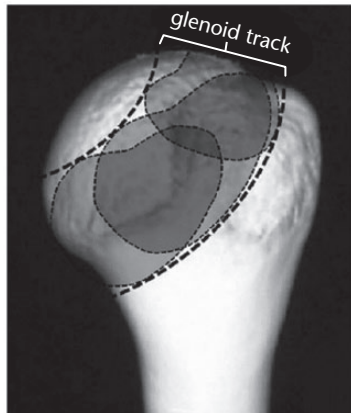
The first factor that needs to be taken into account is the shape of the glenoid and its morphology. As previously mentioned, the socket is inherently small in size when compared to the humeral head; therefore, several factors come into play in order to maintain stability. Concavity-compression is the predominant mechanism by which the humeral head is centred in the glenoid cavity at the mid-range.<sup>17</sup> As the head is pushed against the glenoid bone, its depth, width and version become relevant. The glenoid socket is twice as deep in the superior-inferior direction as in the anteroposterior (AP) direction;<sup>18</sup> therefore, different amounts of displacing forces are needed to dislocate the humeral head in different directions. Lippitt et al<sup>19</sup> quantified the amount of force needed to overcome the compressive force in different directions as suggested by Fukuda,<sup>20</sup> introducing the concept of stability ratio as the translation force divided by the compressive force in different directions. They found that the stability ratio for the superior and inferior directions was about twice as great when compared to the anterior and posterior directions (64% versus 33–35%, respectively). They also concluded that this was related to the greater effective depth of the glenoid in these planes (4.8 mm versus 2.2 mm, respectively). In fact, a linear relationship exists between the effective depth of the glenoid concavity and the stability ratio. The clinical implications of a pathological loss of bone concavity were further studied by Moroder et al<sup>21,22</sup> and Peltz et al,<sup>23</sup> whose studies established a correlation

between the loss of glenoid concavity and instability: in fact, both in traumatic and atraumatic shoulder instability, the glenoid displayed a flatter morphology and a higher radius of curvature (ROC) when compared to healthy volunteers with no history of shoulder pathology. In addition, ROC in the AP direction generally appeared greater than ROC in the supero-inferior direction, confirming previously published data on glenoid morphology,<sup>24</sup> which could explain why a higher degree of instability is present in the AP direction compared to all others.

Glenoid version, defined as the orientation of the articular surface relative to the axis of the scapular body, is another variable which can influence stability, particularly in the posterior direction. It is best measured using advanced imaging techniques (i.e. computed tomography: CT)<sup>25–28</sup> and normally displays few degrees of retroversion with respect to the plane of the scapula (usually 1–7°, although quite some variation is reported in the literature in terms of range and average value).<sup>29,30</sup> When the glenoid version is altered (i.e. due to dysplasia), it can affect stability in the AP plane.<sup>31–35</sup> This proves especially true when glenoid version approaches +10° of anteversion and –15° of retroversion,<sup>36</sup> resulting in increased anterior and posterior instability, respectively. Edelson<sup>37</sup> and Weishaupt et al<sup>38</sup> have provided qualitative descriptions of glenoid dysplasia based on the morphology of the congenital bone deficit, by describing a spectrum of three different anatomic forms of the posterior glenoid rim at the base of the glenoid: pointed form (without bony deficiency), rounded glenoid deficiency (‘lazy J’ form), and triangular bony deficiency (‘delta’ form). In their analysis they also measured glenoid retroversion, which appeared to be significantly increased in posterior shoulder instability when compared to patients with anterior instability, later confirmed by the findings of Inui et al.<sup>39</sup>

Decreased retroversion and inferior inclination also seem to play a role in anterior shoulder stability. Hohmann and Tetsworth<sup>40</sup> found a difference in glenoid version and inclination in patients who had sustained a prior anterior shoulder dislocation compared with the patients in the matched control group who underwent shoulder MRI for other causes: when compared to a healthy control group, the anterior dislocation group displayed –1.7°±4.5 (range: 0.9° to 2.5°) of retroversion and 1.6°±5.9 (range: 0.6° to 2.6°) of inferior inclination, as compared to values of –5.8°±4.6 (range: 5.0° to 6.8°) and 4.0°±6.8 (range: 2.8° to 5.2°) of retroversion and superior inclination, respectively.

Scapular positioning can also affect stability. Warner et al<sup>41</sup> found that almost two thirds of instability patients showed alteration in scapulothoracic motion, probably due to muscle inhibition and poor dynamic control of the scapula itself.<sup>42</sup> A systematic review by Struyf et al<sup>43</sup> confirmed the alterations in scapulothoracic muscle activity in instability patients when compared to healthy controls,



**Fig. 1** During arm motion towards the end-range, the glenoid moves along the contact zone on the posterior margin of the humeral articular surface, shifting from inferomedial to posterolateral. Reproduced with permission from Itoi E. 'On-track' and 'off-track' shoulder lesions. *EFORT Open Rev* 2017;2:343-351.

although no clear changes in activation patterns were identified. A cadaveric study by Kikuchi et al<sup>44</sup> showed how posterior and inferior stability increased with an anterior tilt of more than 5° and with a superior tilt of 10°, respectively, whereas on the other hand the anterior and posterior stability decreased with an anterior tilt of 5° and with a posterior tilt of 15°, respectively.

Anatomical factors seem to play a far less important role, if any, on the humeral side. Several authors, while confirming the role of anatomical variations in the glenoid of unstable shoulders, have shown how these variations had no role on the humeral side. Early roentgenographic evaluations by Cyprien et al<sup>45</sup> suggested no influence of humeral head torsion variation in normal and unstable shoulder. Further CT studies<sup>33,46</sup> found no significant differences in glenohumeral index, humeral retrotorsion or variation in radius or width of the humeral head between patients with recurrent anterior shoulder instability and control patients.

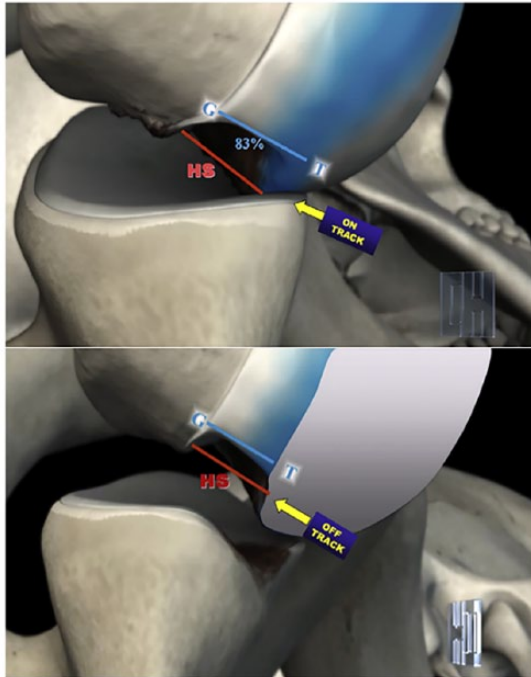
### Bone loss size and location: clinical relevance and management strategies

In most cases, forceful abduction and external rotation force the humeral head out of the glenoid in an anterior-inferior direction, although other mechanisms of injury have been recently suggested.<sup>47,48</sup> After a traumatic episode, there is a high probability of bony lesions on both the humeral head and the glenoid socket. A posterolateral humeral head compression fracture, known as Hill–Sachs lesion (HSL), is therefore caused by the impact with the glenoid and is present in 65% to 67% of dislocations after the first episode and in 84% to 93% in recurrent dislocations.<sup>49,50</sup> Posterior dislocation

occurs much less frequently and is usually a result of direct trauma or seizure.<sup>51</sup> In this case, an anterior-superior impaction fracture ('reverse Hill–Sachs lesion' or RHSL, first described by McLaughlin) is created by the postero-inferior glenoid rim and can be present in about 86% of first-time posterior dislocations.<sup>52</sup>

The glenoid socket bone can also be affected depending on the direction of the dislocation. In first-time unilateral anterior dislocation, glenoid bone loss (GBL) can occur in about 41% of cases. This percentage may rise to 86% in patients with recurrent unilateral anterior dislocation.<sup>53</sup> This confirms the findings of Sugaya et al,<sup>54</sup> who found bony lesions in the anterior-inferior portion of the glenoid in about 90% of cases, and further classified them into fragment-type and erosion/compression-type, a probable consequence of a strong or weak capsular tissue, respectively.<sup>55</sup> Similar percentages of posterior-inferior GBL are found in the rarer occurrence of posterior instability.<sup>38,52</sup>

In the setting of shoulder instability, regardless of the direction, precise bone loss evaluation through three-dimensional CT scans is key.<sup>55,56,57</sup> This happens when the glenoid and humeral bone loss interact in such a way that favours further dislocation, hence the HSL 'engages' the anterior glenoid defect and dislocates the humeral head from the socket.<sup>58</sup> Therefore, HSL and glenoid bone deficits must be considered together. In the setting of anterior instability, it is important to determine how these affect the zone of contact between the glenoid and the humeral head, defined as 'glenoid track' (GT)<sup>59</sup> (Fig. 1), which has been measured to correspond to about 83% of glenoid width in live shoulders<sup>60</sup> and represents a key factor in maintaining joint stability. Quantitative and qualitative bone loss analysis is therefore crucial in determining the pathogenesis of recurrent instability and choosing an effective treatment strategy (i.e. 'bone-block' surgery versus capsulo-labral repair). Di Giacomo et al<sup>61</sup> developed the concept of 'on-track' and 'off-track' shoulder lesions: when the HSL engages the anterior glenoid rim, it is defined as 'off-track'; if it does not, it is an 'on-track' lesion. The surgeon must then understand which factors in glenoid and humeral bone loss can cause further instability, whether anterior or posterior. In other words, what features of the HSL and glenoid lesion drive the shoulder 'off-track' (Fig. 2). It is important to point out that this classification differs from the 'engaging'-vs-'non-engaging' type in its very nature: the 'on-track'-vs-'off-track' concept is evaluated through CT scan with the arm at rest, thus maintaining a fixed instantaneous center of rotation (ICR) of the humeral head. This is different from the 'engaging'-vs-'non-engaging' classification, which is a clinical evaluation performed preoperatively under general anesthesia, where the humeral head dislocates due to the unrepaired Bankart lesion. In this case, the ICR migrates



**Fig. 2** The “on-track”/“off-track” concept in anterior shoulder instability from Di Giacomo et al.<sup>61</sup> If HSL falls within the medial margin of the GT, there is still glenoid track support for bone stability (“on-track” HSL) and the HSL will not engage (above). If the HSL extends medial to the medial margin of the GT and there is concomitant loss of bone support at the anterior glenoid rim, the HSL will engage (“off-track HSL”)(below). Reproduced with permission from Elsevier.

anteriorly. This leads to a reduction of the GT, therefore impairing evaluation. In our distinction, the ICR remains fixed and the GT is unaffected, thus leading to a more precise evaluation of the type of lesion and of the GT itself.

In terms of anterior-inferior GBL, several CT-based methods are available in order to achieve precise measurements,<sup>62–67</sup> usually expressed as a percentage of the total glenoid surface. Cadaveric studies by Yamamoto et al<sup>68</sup> and Itoi et al<sup>69</sup> proved how a GBL of 20–25% (equivalent to about 6 mm) can compromise the result of an isolated soft-tissue repair (i.e. Bankart repair with or without capsuloplasty), although this value might be as low as 13.5%, particularly in high-demand patients.<sup>70</sup> To corroborate

this, a subsequent cadaveric study by Arciero et al<sup>71</sup> showed that, when an HSL is present, this deficit can be as small as 2 mm in order to compromise glenohumeral stability. This is further proof of the interdependence of the two lesions and reinforces the concept of the bipolar nature of anterior shoulder instability, which constitutes the majority of cases.<sup>53</sup> In terms of HSL morphological features, its size, depth, width and orientation have all been thought to be parameters which could estimate the risk of engagement.<sup>72–76</sup> However, their evaluation alone is not sufficient to evaluate the risk of recurrent instability. In fact, it is also critical to evaluate the position of the HSL. This is because with an increase in arm elevation, the contact area between the glenoid and the humeral head moves from the inferomedial to the superolateral portion of posterior articular surface of the humeral head; therefore, the larger the medial portion of the HSL that falls outside the GT, the higher the probability of recurrence,<sup>59</sup> thus becoming an ‘off-track’ lesion with potential for re-engagement, even after capsulo-labral repair surgery. Categorizing HSLs as such can thus help the surgeon predict the chance of failure of arthroscopic stabilization (versus bone-block surgery) better than solely quantifying glenoid osseous defect.<sup>77</sup> In addition, the presence of a GBL increases the likelihood of recurrence. This happens because the width of the glenoid is the only factor which influences the width of the GT.<sup>78</sup> Therefore, an anterior rim deficit directly affects the width of the GT: the smaller the glenoid track, the higher the probability that the medial margin of the HSL falls outside its boundaries, creating an ‘insufficient’ GT. The new GT will therefore be equal to 83% of the diameter of the inferior glenoid minus the width of the anterior GBL. It can then be calculated using CT scans and superimposed onto the HSL in order to classify it as either ‘on-track’ or ‘off-track’ and treat it accordingly.

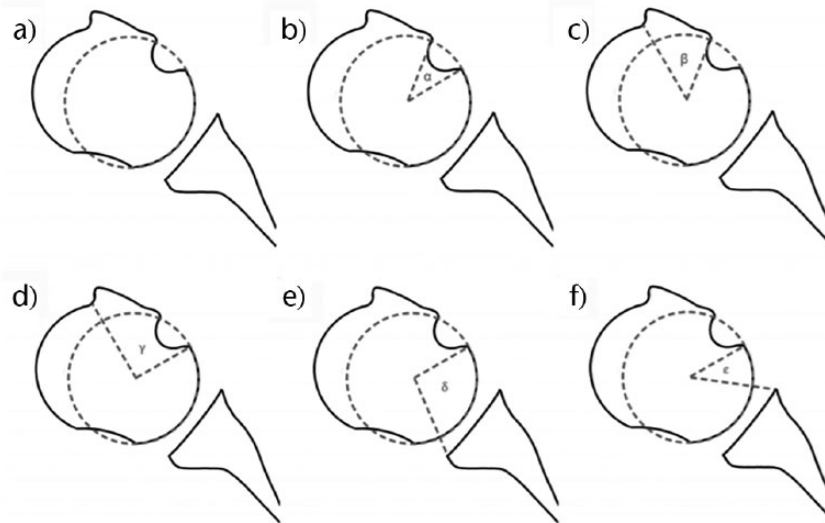
We can therefore summarize treatment options based on the percentage of GBL and type of HSL (Table 1). In general, we can adopt a cut-off value of 25% of GBL, beyond which bone-block surgery is mandatory, regardless of the type of HSL. Below 25% GBL, it could be said that a ‘grey area’ of treatment exists. Nevertheless, a further cut-off value of 13.5% GBL should be adopted:<sup>70</sup> between 0% and 13.5%, an arthroscopic Bankart repair can be safely chosen as a treatment option when the lesion is ‘on-track’;

**Table 1.** Antero-inferior shoulder instability treatment algorithm based on different combinations of GBL and type of HSL

	‘On-track’ HSL	‘Off-track’ HSL
<b>0–13.5% GBL</b>	Arthroscopic Bankart repair	<ul style="list-style-type: none"> <li>Arthroscopic Bankart repair + remplissage</li> <li>Open inferior capsular shift</li> <li>Bone-block surgery (i.e. Bristow–Latarjet)</li> </ul>
<b>13.5–25% GBL (‘grey area’)</b>	<ul style="list-style-type: none"> <li>Arthroscopic Bankart repair</li> <li>Open inferior capsular shift</li> <li>Bone-block surgery (i.e. Bristow–Latarjet)</li> </ul>	<ul style="list-style-type: none"> <li>Arthroscopic Bankart repair + remplissage</li> <li>Open inferior capsular shift</li> <li>Bone-block surgery (i.e. Bristow–Latarjet)</li> </ul>
<b>&gt; 25% GBL</b>	Bone-block surgery (i.e. Bristow–Latarjet)	Bone-block surgery (i.e. Bristow–Latarjet)

Note. GBL, glenoid bone loss; HSL, Hill–Sachs lesion.





**Fig. 3** Illustrations of the measurements performed to determine the defect size and localization in posterior shoulder instability as proposed by Moroder et al.<sup>86</sup> (A) Best-fit circle placed on the remainder of the humeral articular surface to create a reference centre for the measured angles; (B) alpha, defined as the angle between the anterior and posterior defect margin; (C) beta, defined as the angle between the anterior defect margin and the bicipital groove; (D) gamma, defined as the angle between the posterior defect margin and the posterior glenoid rim; (E) delta, defined as the angle between the posterior defect margin and the posterior glenoid rim; and (F) epsilon, defined as the angle between the posterior defect margin and the anterior glenoid rim. Reproduced with permission from SAGE Publications.

between 13.5% and 25%, the same procedure can be effective in eliminating instability, but could result in reduced ROM, particularly in abduction and external rotation, as the repair is performed onto the bone loss, thus restricting capsular space and motion.<sup>69</sup> In these cases, open inferior capsular shift and bone-block surgery are viable options. ‘Off-track’ lesions, on the other hand, have a higher risk of engagement, therefore an arthroscopic Bankart repair with an additional infraspinatus tenodesis onto the HSL (or ‘remplissage’) can transform the HSL from intra- to extra-articular, effectively addressing instability and lowering the recurrence rate.<sup>79</sup> Because loss of external rotation is a well-known side effect of this procedure,<sup>80-81</sup> open capsular shift and the Bristow–Latarjet procedure both represent a valid alternative should there be need for its complete restoration (e.g. in professional throwing athletes).

Similar conclusions have been drawn from studies about posterior instability. Nacca et al<sup>57</sup> recently found the critical posterior GBL to be greater than or equal to 20% of the posterior glenoid width, leading to failure of isolated posterior Bankart repair. This result paves the ground for the decision-making process in posterior bone loss, as previous meta-analyses failed to identify high-quality studies which could indicate an adequate treatment algorithm for such lesions.<sup>82</sup> This is due to the rarer (and therefore easily missed) occurrence of posterior instability in the setting of posterior shoulder dislocation, which is classically

associated with epileptic seizure, electrocution accidents and high-energy trauma.<sup>83-84</sup> Posterior GBL and RHSL are found in 9% and 39% of cases respectively, yet a combination of the two lesions seems to occur in only 2% of cases.<sup>82</sup> Therefore, a high index of suspicion after an acute episode of posterior dislocation is a key step in identifying posterior instability. Once the diagnosis is made, evaluation of bone loss with the aid of CT scans will help assess the feature of bone deficits, similarly to the procedure applied in anterior instability. The proposed treatments available in the literature at this time are mostly solely based on the percentage of humeral head bone loss: if this is lower than 25%, it is most frequently managed with a posterior capsular repair, a closed reduction, or, rarely, an arthroscopic repair. A humeral head bone loss from 25% to 50% is mainly managed with an open reconstruction with bone graft or a subscapularis tendon transfer (McLaughlin technique). Finally, if the humeral head bone loss is > 50%, arthroplasty is the suggested choice.<sup>82</sup>

Moroder et al<sup>85</sup> proposed a different approach to posterior instability by performing a CT best-fit circle measurement of the angle between the posterior RHSL edge and the bicipital sulcus (‘gamma angle’) and the angle between the posterior defect margin and the posterior glenoid rim (‘delta angle’)<sup>86</sup> (Fig. 3). According to this data, a posterior glenoid bone defect can turn a non-engaging RSHL lesion into an engaging one when 2.3° per mm bone loss at the posterior glenoid rim plus the

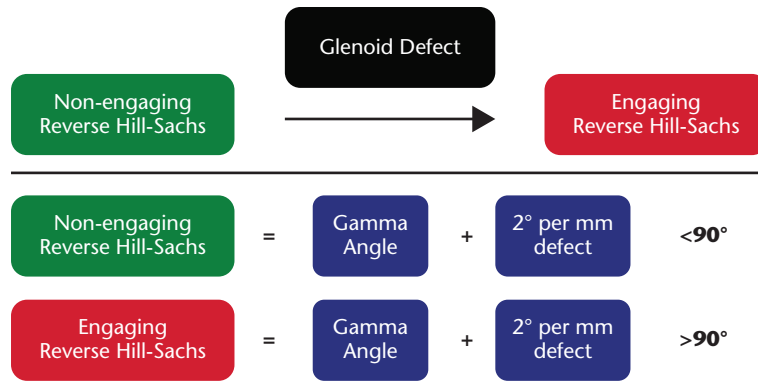


Fig. 4 The gamma angle concept, as proposed by Moroder et al.<sup>85</sup> Reproduced with permission from SAGE Publications.

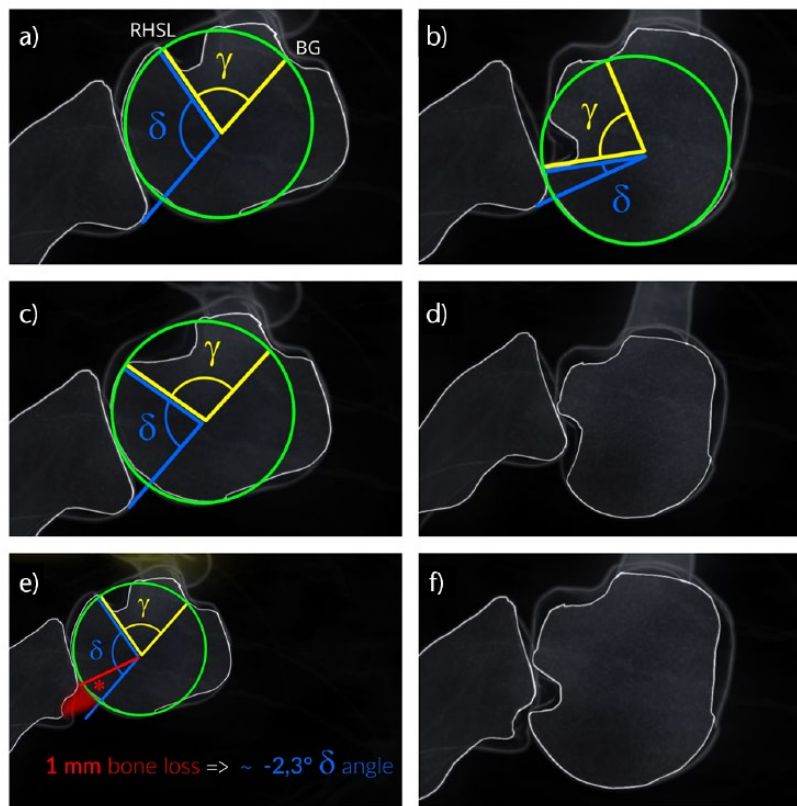


Fig. 5 Schematic representation of the gamma angle concept applied to posterior instability. (A) Gamma angle <90°, (B) internal rotation does not engage the posterior glenoid. (C) Gamma angle >90°, (D) internal rotation engages the posterior glenoid. (E) When posterior GBL is present, about 2.3 degrees per mm of bone loss are lost on the delta angle. (F) In this case, concomitant posterior glenoid defects might lead to the engagement of noncritical RHSLs.

Note. BG: Bicipital Groove.

gamma angle is greater than 90°. They defined this as the ‘gamma angle concept’ (Fig. 4), confirming how posterior instability acts similarly to its anterior counterpart in terms of humeral head defect size and location in the risk of engagement, as the more medial and bigger the RHSL, the greater the gamma angle.<sup>86-87</sup> In general, a gamma

angle greater than 90° warrants surgical stabilization as it leads to a higher risk of recurrence (Fig. 5).

Therefore, when evaluating bone loss in posterior shoulder instability, a cut-off value of 20% of posterior GBL generally warrants posterior bone-block stabilization surgery, achievable mainly through the use of posterior

iliac bone graft<sup>88</sup> as described by Levigne et al,<sup>89-90</sup> while posterior GBL < 20% can be treated with good results with arthroscopic posterior capsulo-labral repair.<sup>91</sup> In addition, the gamma angle concept proposed by Moroder et al can further help to identify those lesions which are prone to engagement and assist the surgeon in the decision-making process, especially in cases of bipolar bone lesion (RHSL with posterior GBL). Nevertheless, further clinical research is needed in order to determine whether the engagement predictions correlate with clinical instability.<sup>85</sup> Until these kind of studies are available, it is reasonable to treat these lesions based on history and clinical evaluation, along with the aid of sagittal and axial CT scans to help evaluate the risk of chronic posterior instability by evaluating humeral and posterior glenoid bone loss and applying the gamma angle concept as previously described.

## Conclusions

Abnormality of the dynamic interplay of bony structures in the setting of shoulder instability, either congenital or acquired, can both favour instability and impair surgical results if not diagnosed correctly. This is especially true in the evaluation of bone loss after an episode of traumatic shoulder dislocation, either in an anterior or posterior direction. Precise assessment of such lesions can be achieved through CT imaging, and an adequate evaluation methodology can guide surgeons in choosing the optimal type of stabilization surgery.

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