# Variations in the superior capsuloligamentous complex and description of a new ligament

Nicole Pouliart, MD, PhD,[a,b,c] Katia Somers, MD,[d] Said Eid, MD,[e] and Olivier Gagey, MD, PhD,[c,d,e] *Brussels, Belgium, and Paris, and Le Kremlin-Bicêtre, France*

*Although the rotator cuff interval and the adjacent ligaments are gaining interest because of their importance for glenohumeral instability and adhesive capsulitis, there seems to be some confusion about their anatomy. This study reinvestigates the superior capsular structures in 110 cadaveric shoulders by a combination of arthroscopy, dissection, histology, and functional analysis. The structure of the superior capsule was found to be more complex than suspected until now. The coracohumeral, coracoglenoid, and superior glenohumeral ligaments joined with a circular transverse band to form the anterior limb of a suspension sling. This was 9 to 26 mm wide at its midportion. In 90% of the specimens, there also was a posterior limb composed of a broad fibrous sheet, 6 to 26 mm wide at its midportion. This hitherto unrecognized posterosuperior glenohumeral ligament joined posterolaterally with the circular transverse band. Four types of configuration for the superior complex could be identified. The suspension sling formed by the superior complex functions in the same way as the hammock formed by the inferior glenohumeral ligament complex. The posterior limb seems to restrict internal rotation, like the anterior limb restricts external rotation. The expanded knowledge of the superior capsular complex increases the understanding of the pathology involved in anterosuperior and posterosuperior impingement, as well as articular-sided rotator cuff tears. It also has clinical implications for rotator cuff interval and biceps pulley lesions, because these areas are bordered by the anterior limb of the superior complex, as well as for adhesive capsulitis, where we can now understand why internal rotation is limited and why the release needs to be extended posterosuperiorly. (J Shoulder Elbow Surg 2007;16:821–836.)*

From the Departments of [a]Orthopaedics and Traumatology, [d]Pathologic Anatomy, Universitair Ziekenhuis Brussel, and [b]Department of Human Anatomy, Vrije Universiteit Brussel, Brussels, [c]Institut d'Anatomie Paris; and [e]Service d'Orthopédie, Hôpital Bicêtre, Université Paris-Sud, Le Kremlin-Bicêtre.

Reprint requests: Nicole Pouliart, Academic Hospital, Vrije Universiteit Brussel, Laarbeeklaan 101, 1090 Brussels, Belgium (E-mail: *izvptn@az.vub.ac.be*).



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Recently, the anterosuperior part of the glenohumeral capsule has gained interest, because it forms the fibrous bottom of the rotator cuff interval. This area has been attributed a role in subtle forms of shoulder instability, such as anterosuperior impingement, and in adhesive capsulitis, where it needs to be released when surgery is decided upon. When one reviews the literature regarding the superior part of the glenohumeral capsule, there appears to be some disagreement. Some authors believe that the coracohumeral ligament is the only substantial structure in this area.[20,58,97] Others think that the coracohumeral ligament is rather indistinct and that the superior glenohumeral ligament is the most important structure.[11,12,30] Most authors are of the opinion that both ligaments coexist in variable configurations, although there is some confusion in terminology.[16,21,46,49,58,82] A few authors also mention a coracoglenoid ligament.[15,46,80,96] On the lateral side of the capsule, Clark et al[9,10] and Burkhart et al[6,7] have described a transverse band or rotator cable, which seems to link the capsular ligaments with the overlying rotator cuff tendons.

In another study, we showed that the anteroinferior and posteroinferior capsular folds seen during arthroscopy correspond with, but are not equal to, the anterior and posterior band of the inferior glenohumeral ligament.[74] During this study, we noticed that similar folds could be seen in the anterosuperior and posterosuperior aspects of the capsule when rotating the humerus. The anterosuperior fold probably corresponds with the superior glenohumeral ligament, but we did not find a reference to a similar posterosuperior substrate. It is our hypothesis that a balancing superior sling, comparable to the inferior hammock described by O'Brien et al,[63] exists.

This study reinvestigates the composition of the anterosuperior part of the shoulder capsule and the rotator cuff interval in an effort to resolve the confusion. The second purpose was to verify our superior-sling hypothesis by defining the anterosuperior and posterosuperior capsular folds with a combination of arthroscopy, macroscopic dissection, and histology.

## MATERIALS AND METHODS

One hundred ten non-embalmed shoulders from donated cadavers (age range, 50-95 years) were studied (Figure 1). All specimens were dissected, but 50 shoulders also underwent arthroscopy.

Twenty-five specimens were inspected arthroscopically through a standard posterior portal before any dissection. This was done to be sure that removal of the superficial layers did not alter the capsular folds. After the superior capsular folds and the rotator cable were visually identified, the scope was removed, and dissection was carried out.

In all shoulders, all soft tissues surrounding the rotator cuff were removed, including the deltoid muscle that was detached at its origin along the clavicle and scapula and then reflected laterally. The subdeltoid bursa was reflected from the underlying rotator cuff. After careful identification of the coracoacromial and coracohumeral ligaments, the conjoined tendon was detached together with the tip of the coracoid process and reflected downward. In most shoulders, the acromion and the distal clavicle were removed as well.

The superior structures were marked arthroscopically in 50 specimens. Again, the functional anatomy was studied by inspection of the intra-articular side of the capsule during motion. A standard posterior portal through the interval between the infraspinatus and teres minor was used. The anterosuperior and posterosuperior structures were inspected in 0° to 90° of abduction and in maximal internal and external rotation at intervals of 30° of abduction. The clearly visible anterosuperior and posterosuperior folds that developed in varying arm positions, as well as the rotator crescent, were then marked with arthroscopically placed suture loops. In 20 specimens, the fold representing the middle glenohumeral ligament was marked as well.

Then, we proceeded with dissection in all specimens. The subscapularis, supraspinatus, infraspinatus, and teres minor muscles were detached from their scapular origin and carefully separated from the underlying capsule, without damaging the latter, as far laterally as possible until the point at which the capsule and tendon could not be separated from each other except by sharp dissection. In the arthroscopic group, care was taken not to cut the suture marker loops when dissecting the rotator cuff muscles free (Figure 2). Next, the humerus was moved from adduction to abduction and through its full range of rotation at intervals of 30° of abduction. In each of these positions, the appearance of folds on the outside of the capsule was noted. For the arthroscopically investigated specimens, the correspondence of these folds and the marker loops was verified. In the other specimens, the site of these folds was marked for further reference. Subsequently, the loose connective tissue and synovium around the fibrous structure of the capsule were carefully removed under loupe magnification by a combination of blunt and sharp dissection, starting at Weitbrecht's foramen between the middle and the superior glenohumeral ligament. The humerus was then again moved through its range of motion to verify that the folds still appeared at the same site as when the capsular ligaments were not yet dissected free.

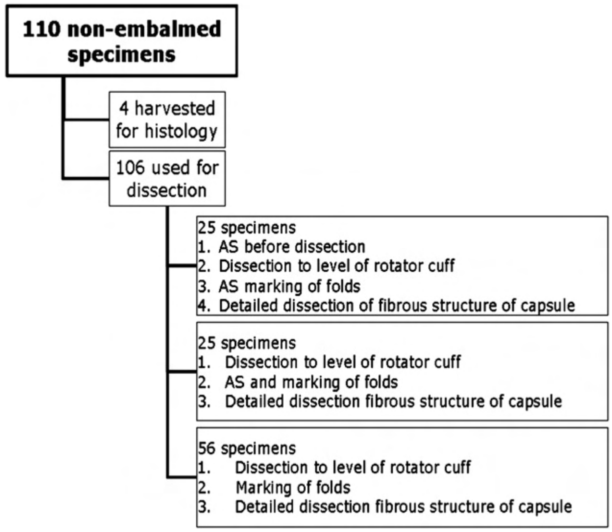


**Figure 1** Flow chart summarizing distribution of specimens in methodologic groups. AS, arthroscopy.

Finally, the glenohumeral capsular ligaments were detached along the glenoid rim, keeping the labrum attached to the ligaments. Before cutting of the remnant of the rotator cuff together with the attached capsule along their humeral insertion, the ligaments were reflected laterally to study their continuity with the anterior capsular structures. Thereafter, the capsule was laid out flat to study the configuration of the various fibrous parts of the capsule and to see whether any folds remained in this situation.

In 4 specimens in the non-arthroscopic group, the capsule was harvested. In 2, the rotator cuff muscles were not fully removed, but they were removed up to their junction with the capsule in the other 2. No detailed dissection of the fibrous structure of the capsule was done in these 4 specimens. These harvested capsules were used for histologic examination to verify whether the folds and the dissected fibrous structure correspond with microscopic examination of the capsule. The harvested pieces were cut into segments of approximately 5 mm in width and 30 mm in length. In 2 specimens, the orientation of the segments was radial, and in the other 2, the orientation was circular. The capsular segments were embedded in paraffin, and from each paraffin block, 3 sequential sections of 4 $\mu$m were made. The sequential slides from each block were stained with hematoxylin-eosin staining, with the Masson trichrome technique, and with van Gieson staining. The slides were examined with a light microscope at 40×, 100×, and 200× magnification. Orientation of the collagen fibers was documented, and relevant areas were digitally photographed.

## RESULTS

### *Arthroscopy*

With the arm hanging down and in neutral rotation, no distinct capsular folds could be observed in

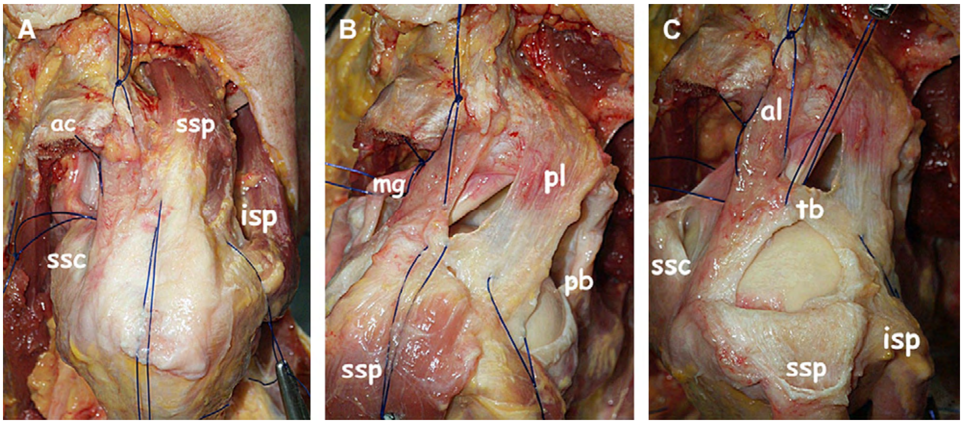


**Figure 2** Progressive dissection around suture markers, with 4 markers: on the middle glenohumeral ligament, on the anterosuperior fold, on the posterosuperior fold, and on the transverse band. **A**, Rotator cuff tendons dissected out. **B**, Rotator cuff tendons dissected off capsule and reflected. **C**, Rotator crescent dissected free and reflected. *ac*, Acromion; *ssp*, supraspinatus; *ssc*, subscapularis; *isp*, infraspinatus; *mg*, middle glenohumeral ligament; *pl*, posterior limb of superior complex; *pb*, posterior band of inferior glenohumeral ligament; *al*, anterior limb of superior complex; *tb*, transverse band.

the superior part of the glenohumeral capsule in the majority of specimens (Figure 3). In about 60% of shoulders, the anterior edge of the superior glenohumeral ligament could be derived from the superior border of a relatively deep Weitbrecht's foramen, giving access to the subscapular recess. If this opening was not clearly present, no landmarks in the capsule were available to identify the superior glenohumeral ligament unequivocally in the resting position of the arm. In 20 of these cases, we decided to mark the middle glenohumeral ligament too. In about half of these aging specimens, a distinct rotator cable surrounding a distinct rotator crescent was seen. The rotator cable spanned from anterolateral above the biceps groove to posterolateral. In about a quarter of shoulders, the rotator cable was less obvious but could be identified by adding traction to the arm or by rotating the humerus. In these shoulders, the rotator crescent was not visible. In the remaining quarter of specimens, the rotator cable and crescent could not be discerned despite manipulations. In these shoulders, the rotator crescent, therefore, could not be marked. In adduction and external rotation, a longitudinal anterosuperior capsular fold with a distinct anterior leading edge developed in all cases. The leading edge corresponded with the superior border of Weitbrecht's foramen. This capsular fold ran from the anterosuperior glenoid rim, adjacent to the tendon of the long head of the biceps, to the biceps pulley. In those specimens in which a rotator cable was observed, the capsular fold joined the anterior leg of the cable. In adduction and internal rotation, the longitudinal anterior capsular fold was no longer seen, although the anterosuperior part of the capsule had a tendency to fold up in the transverse direction. This phenomenon was even more obvious when the humerus was in abduction and internal rotation. With reversed rotation, the same observation of transverse folding, unfolding, and longitudinal folding was observed for the posterosuperior part of the capsule. In adduction and internal rotation, the posterosuperior capsule became so tight that the arthroscope was squeezed downward and out. The longitudinal posterosuperior fold appeared just superior to the arthroscopic posterior portal and ran from the posterosuperior glenoid rim, medial and posterior to the origin of the long tendon of the biceps and the glenoid labrum, to the posterior part of the greater tubercle. Here it merged with the posterior leg of the rotator cable when the latter was visible. Because both longitudinal superior folds were always seen during either external or internal rotation, they could be marked in all cases.

### Dissection

In the interval between the supraspinatus and subscapularis, the deep layer of the subacromial bursa enveloped the free margins of both tendons. In the depth of the interval, it was adherent to the external layer of the glenohumeral capsule. Medial to the level of the glenoid rim and surrounding the neck of the coracoid process, the anterior rotator cuff interval was filled with fatty tissue, and it may actually continue into the subcoracoid bursa. Laterally, the tendons of the supraspinatus and the subscapularis intermingled very little, close to their insertion, and there the interval was no longer definable. The posterior rotator cuff interval between the supraspinatus and the infraspinatus was less clear but still easily distinguishable. Laterally, however, both tendons were seen to cross each other and intermingle over about 2 to 3 cm from their insertion. In 3 specimens, a large rotator cuff tear was observed.

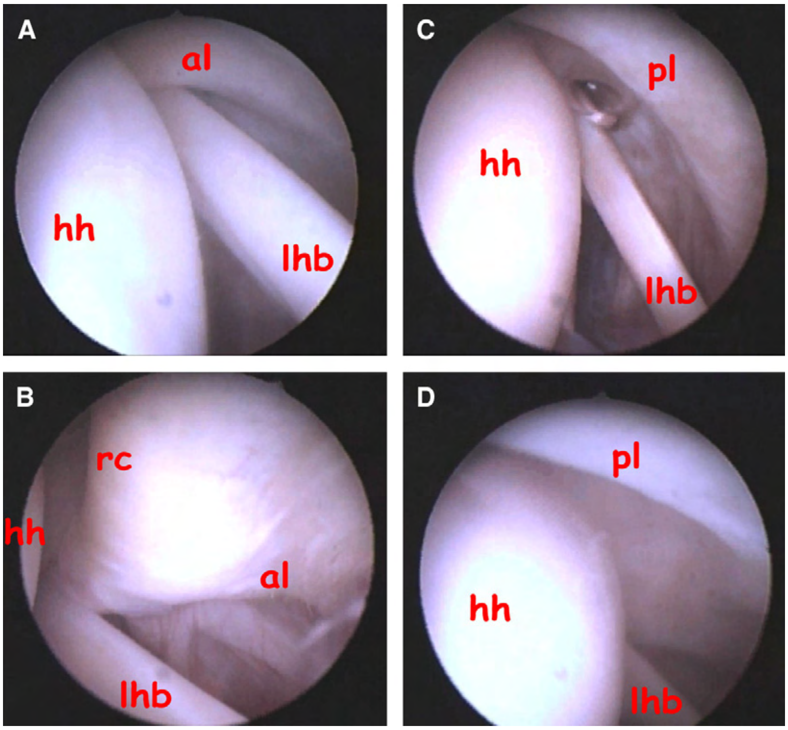


**Figure 3** Arthroscopic view of folds in superior part of glenohumeral capsule. **A**, The anterior limb of the superior complex forms a distinct fold visible in internal rotation. **B**, In this specimen, the anterior limb (*al*) and a distinct rotator cable (*rc*) are visible in internal rotation. The anterior limb (*al*) can be recognized as it courses laterally to the biceps pulley surrounding the long head of the biceps tendon (*lhb*). **C** and **D**, The posterior limb (*pl*) becomes visible as a fold close to the entry point of the arthroscope through the posterior portal during external rotation. The posterior limb is attached more posteriorly on the humeral head (*hh*) and merges with the posterior part of the rotator cable.

Through both rotator cuff intervals, the superior rotator cuff tendons could easily be separated from the underlying glenohumeral capsule up to 14 to 26 mm from the most lateral point of their insertion. At this point, the tendons and the capsule macroscopically intermingled over the entire width of the supraspinatus and the infraspinatus tendons. When the rotator cuff muscles were reflected laterally, the coracohumeral ligament stood out prominently, as it caused the capsule to tent over it, especially with traction exerted on the hanging arm. After all loose connective and synovial tissue was carefully removed while the fibrous structures were retained, a complex superior capsular system emerged (Tables I and II).

The coracohumeral ligament originated from the posterior side of the coracoid process, between the 2 branches of the coracoacromial ligament and, in most instances, also from the base of the coracoid process. In about a third of specimens, the coracohumeral ligament was so broad that its insertion spanned onto the anterior margin of the supraglenoid tubercle. At its midportion, it varied in size from 9 to 26 mm, although it was sometimes confluent with the other fibrous structures in the roof of the joint. Laterally, the coracohumeral ligament inserted partially on the posterior margin of the bicipital groove, where it enveloped the tendon by merging with the transverse humeral ligament. The posterior part of the insertion of the coracohumeral ligament was not directly onto bone but instead merged with a distinct fibrous structure with

**Table I** Variations of coracoglenohumeral ligament in 106 specimens: Coracohumeral ligament, superior glenohumeral ligament, and coracoglenoid ligament

| Variation | No. of specimens | % |
|---|---|---|
| Conjoined CHL-SGHL | 11 | 10.4 |
| Medial merging of CHL-SGHL | 44 | 41.5 |
| Merging of CHL-SGHL in midportion | 22 | 20.8 |
| Lateral merging of CHL-SGHL | 27 | 25.5 |
| No merging of CHL-SGHL | 12 | 11.3 |
| Absent CGHL | 1 | 0.9 |
| Distinct CGL | 59 | 55.7 |
| CGL not identifiable | 14 | 13.2 |

*CHL*, Coracohumeral ligament; *SGHL*, superior glenohumeral ligament; *CGHL*, coracoglenohumeral ligament; *CGL*, coracoglenoid ligament.

**Table II** Variation of superior complex in 106 specimens: Posterosuperior glenohumeral ligament, coracoglenohumeral ligament, and gap between posterosuperior glenohumeral ligament and coracoglenohumeral ligament

| Variation | No. of specimens | % |
|---|---|---|
| Absent PSGHL | 11 | 10.4 |
| Broad gap between PSGHL and CGHL | 45 | 42.5 |
| Small gap between PSGHL and CGHL | 21 | 19.8 |
| Very small gap or confluent PSGHL and CGHL | 29 | 27.4 |

*PSGHL*, Posterosuperior glenohumeral ligament; *CGHL*, coracoglenohumeral ligament.

a circular orientation. The superior glenohumeral ligament was also consistently found, although it often only was a thin fibrous strip. It originated immediately lateral and anterior to the base of the coracoid process and from the anterolateral part of the supraglenoid tubercle. The coracohumeral ligament had to be detached from its origin to be able to see the origin of the superior and middle glenohumeral ligaments fully. In about 10% of specimens, the origins of these latter 2 ligaments were conjoined over up to 1 cm and were not separable there except by sharp dissection. In all specimens, a distinct space between the superior and middle glenohumeral ligaments was observed. This space, Weitbrecht's foramen or the rotator cuff interval, was variable in size, ranging in height at the glenoid border from 2 to 8 mm and at its lateral margin from 13 to 25 mm. The observed size also depended on the position of the humerus. In internal rotation, the interval was almost obliterated, but it was at its maximum height in external rotation. In 2 shoulders, both ligaments had a supplementary connection at their midportion that was 6 mm wide. The superior glenohumeral ligament merged with the coracohumeral ligament medially, within 2 cm of its origin, in 41% of specimens. In 23%, these 2 ligaments joined at their midportion, and in 25%, they did so laterally within 2 cm of the biceps pulley. In 11%, the superior glenohumeral ligament did not merge with the coracohumeral ligament but, instead, inserted on the anterior margin of the biceps groove, contributing to the transverse humeral ligament, or merged with the fasciculus obliquus and the tendon of the subscapularis close to or together with the middle glenohumeral ligament. The superior glenohumeral ligament varied in width from 6 to 12 mm, and the middle glenohumeral ligament varied from 6 to 20 mm. In all specimens, the fibers of the circular band continued further anteriorly and inferiorly into those of the fasciculus obliquus. This fasciculus obliquus was found at the convergence of the anterior capsule and the tendon of the subscapularis. A distinct coracoglenoid ligament from the posterior aspect of the tip of the coracoid process and the coracohumeral ligament to the supraglenoid tubercle was observed in 56% of specimens. In 13% of shoulders, no coracoglenoid ligament could be identified. In the remainder of specimens, there was a broad confluence of the coracohumeral and coracoglenoid ligaments, which resulted in both ligaments not being separable.

Unexpectedly, we also found a distinct longitudinal fibrous structure, mainly parallel to the coracohumeral ligament, in the posterosuperior part of the capsule in most specimens. This structure originated from a ridge on the posterosuperior aspect of the glenoid neck, medial to the glenoid labrum, as well as medial and posterior to the origin of the biceps tendon. Laterally, these fibers fanned out and merged with the circular fibrous structure described earlier, and a small part inserted posteriorly onto the greater tuberosity together with the tendon of the infraspinatus. This posterior fibrous structure formed a complex superior fibrous network together with the coracohumeral ligament, the circular band, the coracoglenoid, and the superior glenohumeral ligaments. There was some variation in the form of the superior complex that could be subdivided into 4 main types. In 10% of specimens, the posterior fibrous structure was not retrieved, either because there were too few fiber bundles to form a fibrous sheet or because this area was too degenerative and no organized fiber bundles could be discerned macroscopically. In 43% of shoulders, there was a distinct posterior fibrous structure and a distinct coracohumeral ligament with a broad gap between them (Figure 4). The biceps tendon was visible through this gap, which was 1.5 to 2 times as wide as the biceps tendon. In 20% of specimens, the gap was smaller but still distinct and about the width of the biceps tendon. In the remaining 27% of shoulders, the gap was very small or nonexistent, resulting in a confluent superior complex. In cases with a small gap, fibers of the posterior structure and the coracohumeral ligament tended to cross over and mingle. When the posterior structure was separate, it ranged in width from 6 to 26 mm at its midportion. When the superior complex was confluent, it ranged in width from 34 to 46 mm. In the specimens with supraspinatus tears, the entire superior complex was still present in 2 cases, whereas the anterior limb of the complex formed by the coracohumeral and superior glenohumeral ligament was destroyed in the other shoulder. The retracted supraspinatus tendon and muscle were adherent to the remainder of the superior complex (Figure 5).

Histologic examination confirmed the presence of well-organized fibrous structures with a longitudinal orientation corresponding with the superior glenohumeral and coracohumeral ligaments, as well as with the macroscopic posterosuperior fibrous structure.

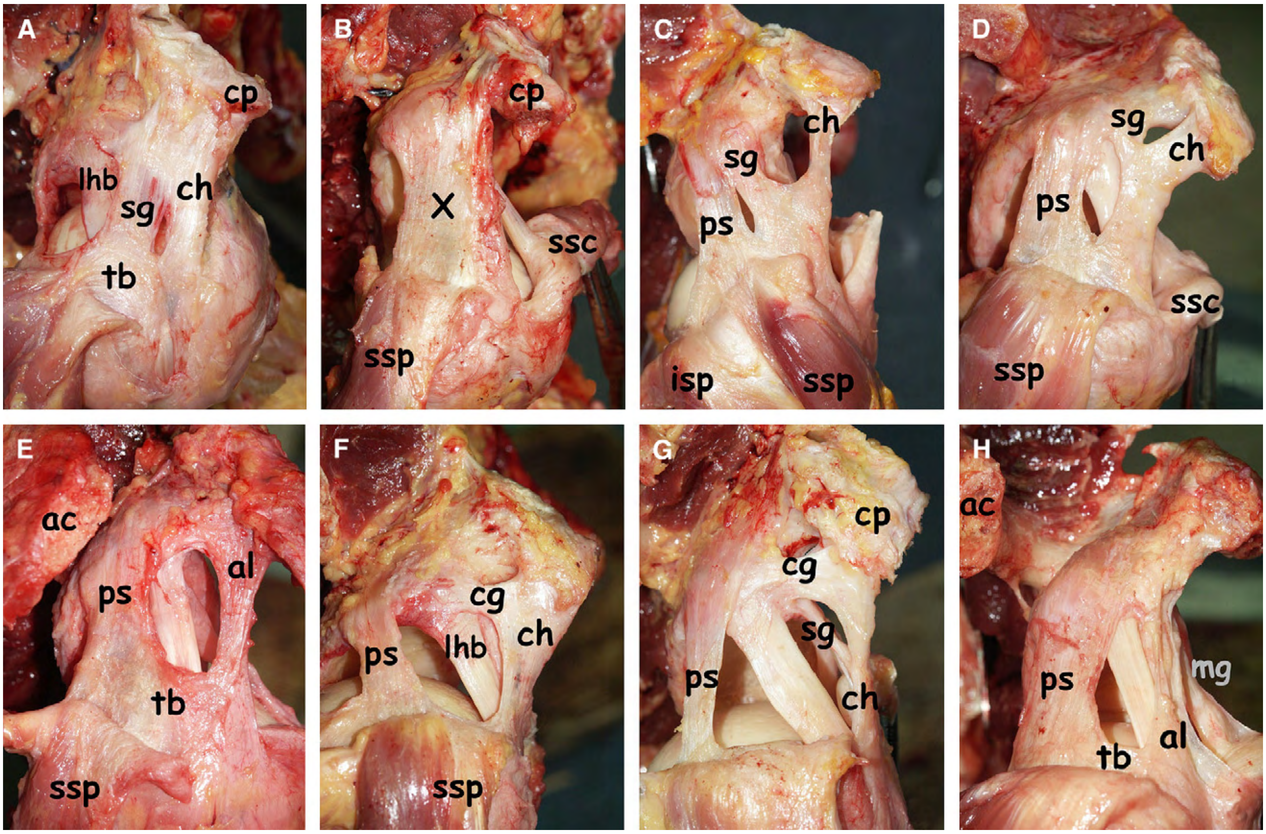


**Figure 4** Various types of superior complex. **A**, Absent posterosuperior glenohumeral ligament (*ps*). **B**, Broad and confluent superior complex (*X*). **C**, Superior complex with small gap between posterosuperior glenohumeral ligament (*ps*) and anterior limb of complex (*al*). The superior glenohumeral ligament (*sg*) and the coracohumeral ligament (*ch*) merge in the middle third, and there is no coracoglenoid ligament (*cg*). **D**, Superior complex with medium-sized gap between posterosuperior glenohumeral ligament (*ps*) and anterior limb of complex (*al*). The superior glenohumeral ligament (*sg*) and the coracohumeral ligament (*ch*) merge in the medial third, and there is no coracoglenoid ligament (*cg*). **E**, Superior complex with medium-sized gap between posterosuperior glenohumeral ligament (*ps*) and anterior limb of complex (*al*). The superior glenohumeral ligament (*sg*), the coracohumeral ligament (*ch*), and the coracoglenoid ligament (*cg*) cannot be separated. **F**, Superior complex with broad gap between posterosuperior glenohumeral ligament (*ps*) and anterior limb of complex (*al*). The superior glenohumeral ligament (*sg*) and the coracohumeral ligament (*ch*) cannot be separated, and there is a distinct coracoglenoid ligament (*cg*). **G**, Superior complex with a very broad gap between posterosuperior glenohumeral ligament (*ps*) and anterior limb of complex (*al*). The superior glenohumeral ligament (*sg*) and the coracohumeral ligament (*ch*) merge in the lateral third, and there is a distinct coracoglenoid ligament (*cg*). **H**, Superior complex with a very broad posterosuperior glenohumeral ligament (*ps*) but thin coracohumeral (*ch*) and superior glenohumeral ligaments (*sg*). The coracoglenoid ligament (*cg*) merges into the posterosuperior glenohumeral ligament (*ps*). *cp*, Coracoid process; *lhb*, tendon of long head of biceps; *tb*, transverse band; *ssc*, subscapularis; *ssp*, supraspinatus; *isp*, infraspinatus; *ac*, acromion; *mg*, middle glenohumeral ligament.

The circular band was also retrieved on microscopic and histologic examination (Figures 6 and 7).

The circular fibrous structure corresponded with the arthroscopically marked rotator cable. The superior glenohumeral and coracohumeral ligaments were found at the site of the marker loop through the anterosuperior capsular fold. The posterior fibrous structure was pierced by the marker loop on the posterosuperior capsular fold (Figure 2).

The functional anatomy of the retrieved fibrous structures and ligaments was similar to that of the capsular folds seen during arthroscopy (Table III). The coracohumeral and superior glenohumeral ligaments were taut in adduction with external rotation, slack in neutral rotation, and folded in internal rotation. In abduction, both ligaments were folded to slack, although they still became taut in extreme external rotation. The posterior fibrous structure was taut in internal rotation, slack in neutral rotation, and folded in external rotation. More internal rotation was required in abduction than in adduction before the posterior fibrous structure became taut. All structures were also

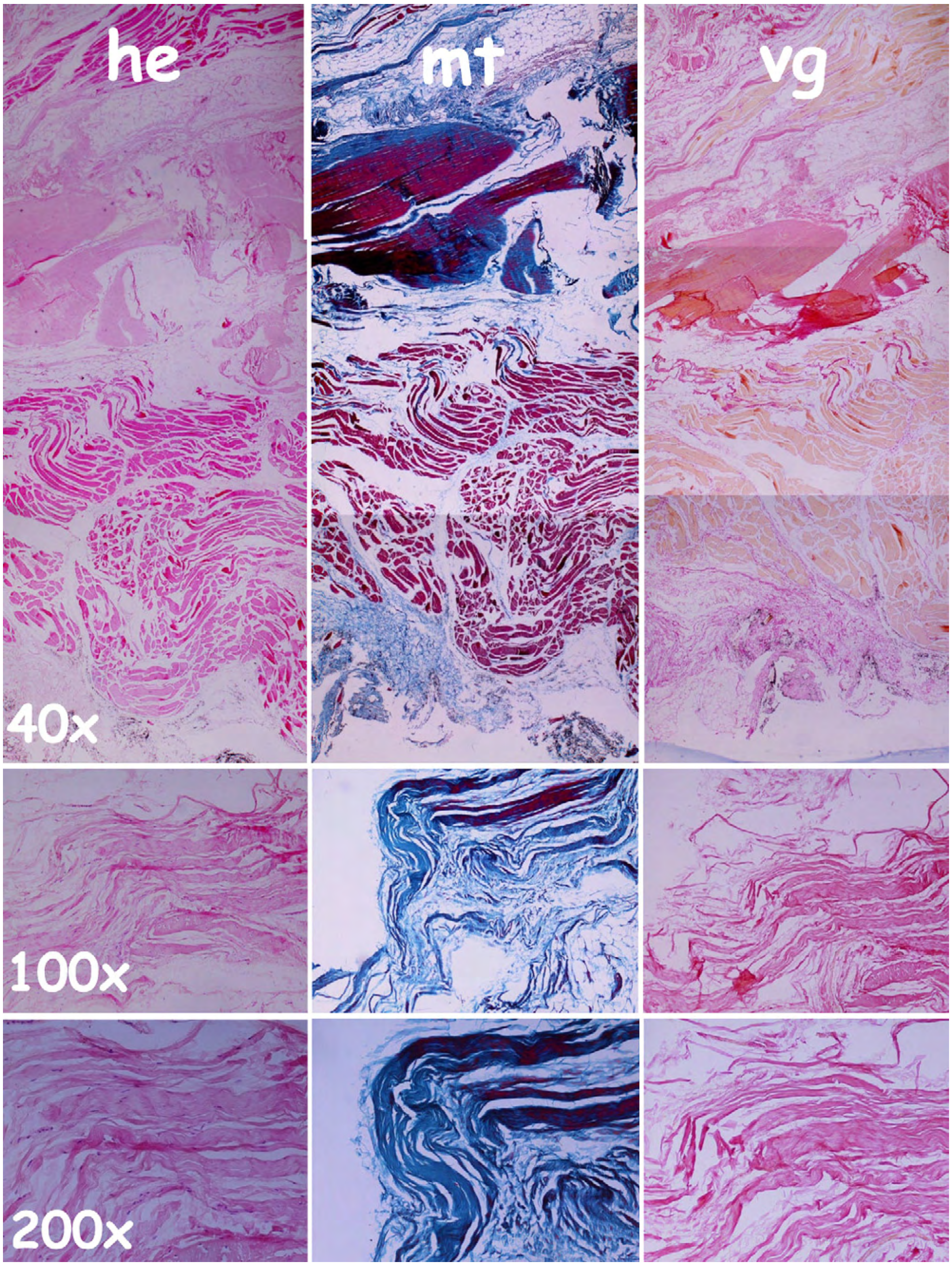


**Figure 5** Digital photographs of light microscopic examination of anterior limb of superior glenohumeral ligament complex: hematoxylin-eosin stain (*he*) (*left*), Masson trichrome stain (*mt*) (*middle*), and van Gieson stain (*vg*) (*right*) with 40× (top), 100× (*middle*), and 200× (*bottom*) magnification. Several layers of thick parallel bundles of collagen fibers with a radial orientation parallel to the cut alternate with parallel bundles of collagen fibers with a transverse orientation perpendicular to the cut. At higher magnification, the fiber bundles are seen to have a wavy appearance, probably because the cut sections were not fixated under tension.

under tension when traction was applied on the humerus, and this effect was again more pronounced in adduction than in abduction. Part of the tension generated in either the anterosuperior ligaments or the posterior fibrous structure seemed to be transmitted to the other structure through the circular band. Figures 7, 8, and 9 illustrate the functional anatomy for 3 types of superior complexes.

In the specimens with rotator cuff tears, the presence of the superior complex appeared to be able to limit superior displacement of the humeral head. In rotation, the superior complex even seemed to be

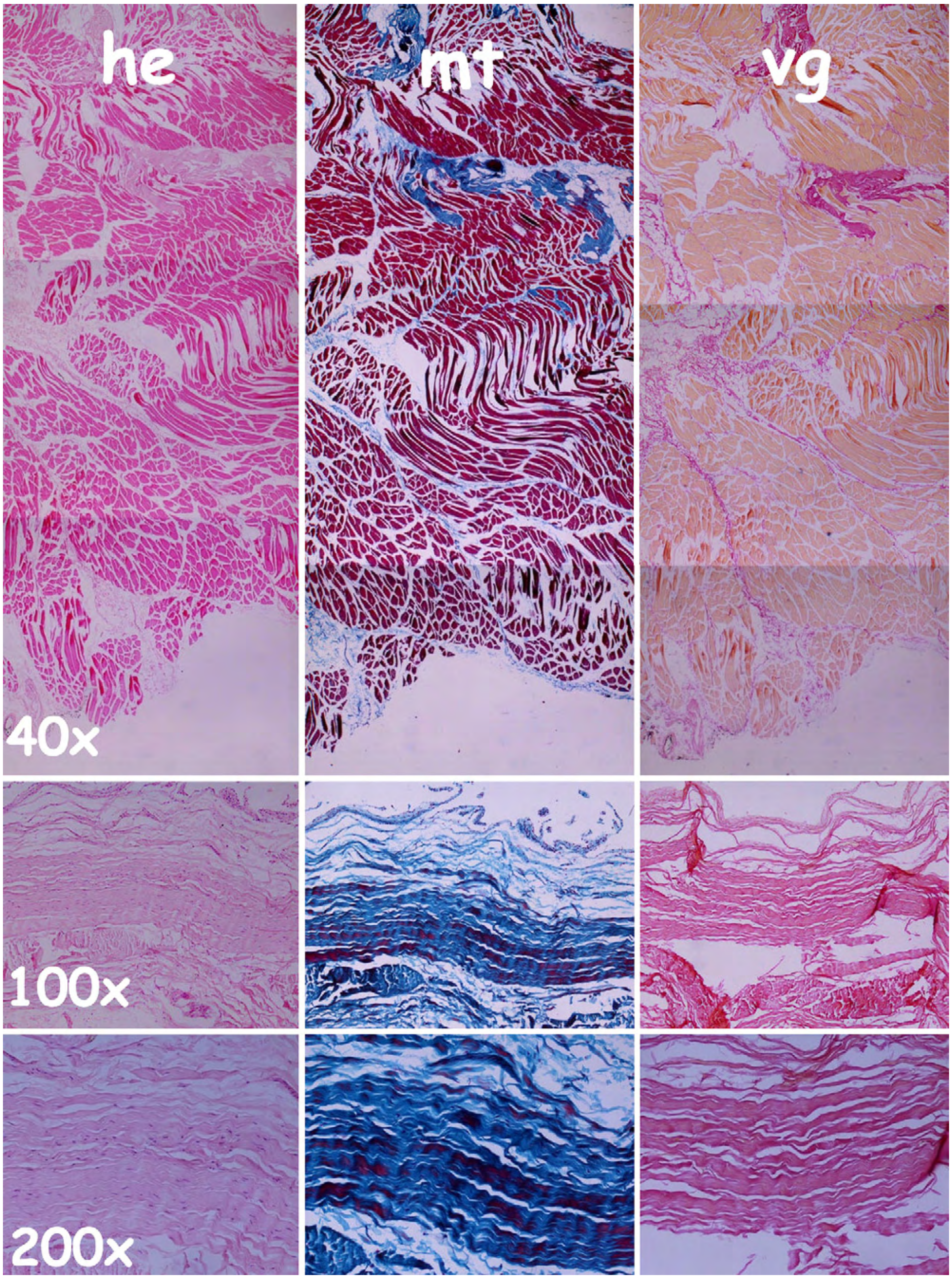


**Figure 6** Digital photographs of light microscopic examination of posterior limb of superior glenohumeral ligament complex; hematoxylin-eosin stain (*he*) (*left*), Masson trichrome stain (*mt*) (*middle*), and van Gieson stain (*vg*) (*right*) with 40× (*top*), 100× (*middle*), and 200× (*bottom*) magnification. Several layers of thick parallel bundles of collagen fibers with a radial orientation parallel to the cut alternate with parallel bundles of collagen fibers with a transverse orientation perpendicular to the cut. At higher magnification, the fiber bundles are seen to have a wavy appearance, probably because the cut sections were not fixated under tension.

able to exert a centering effect on the humeral head. In addition, the intermingling of the supraspinatus and infraspinatus tendons with the superior complex seemed to be responsible for limiting retraction of the torn rotator cuff tendon (Figure 10).

## DISCUSSION

Schlemm[82] originally described the coracohumeral ligament as having 2 roots. He observed a superior, stronger part from the lateral border of the coracoid process to the posterior margin of the bicipital groove

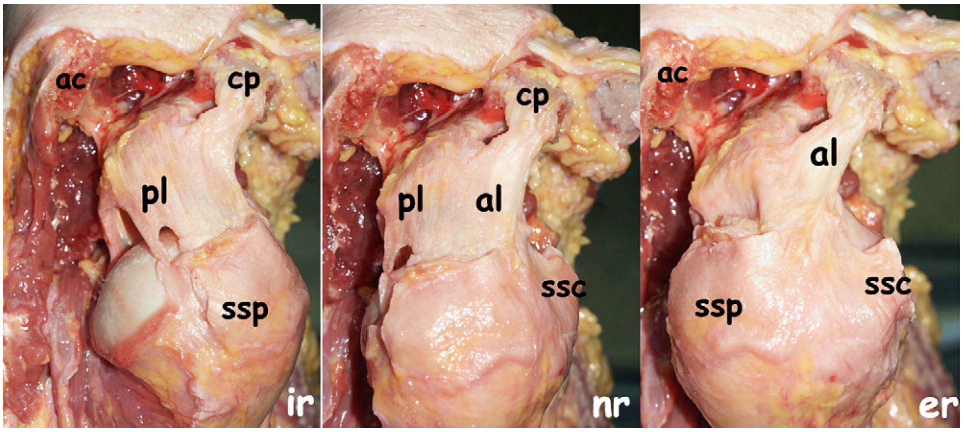


**Figure 7** Functional anatomy of a broad confluent superior complex. In internal rotation (*ir*), the anterior limb (*al*) is folded and the posterior limb is taut; in neutral rotation (*nr*), both limbs are unfolded and taut when the humeral head is allowed to subluxate inferiorly; in external rotation (*er*), the anterior limb (*al*) is taut and the posterior limb (*pl*) is folded. *ac*, Acromion; *cp*, coracoid process; *ssp*, supraspinatus; *ssc*, subscapularis.

and an inferior, weaker part from the glenoid labrum and rim close to the origin of the biceps tendon to the anterior margin of the biceps groove. This description was shared by several authors.[22,35,89,97] Others only mentioned a coracoid origin for the coracohumeral ligament.[16,37] Sappey[80] and Debierre[15] described an anterosuperior structure in 2 parts as well. These authors discerned a superficial coracohumeral ligament, running from the coracoid process and laterally merging with circular fibers, and a deep part from the coracoid process over the supraglenoid tubercle along the biceps tendon to the greater tuberosity. They called the latter part the coracoglenoid ligament, although it continued to the humerus. The coracoglenoid ligament was also studied by Weinstabl et al[96] and Kolts et al.[46,48] In recent literature, Edelson et al[20] and Cooper et al[11,12] found that the coracohumeral ligament appeared as a substantial macroscopic structure but did not correspond with a histologic substrate of discretely organized collagen fiber bundles. Kolts et al[46] found 2 distinct parts closely corresponding with the 2 parts of Debierre and Sappey but laterally inserting on a broad semicircular band spanning the humeral head from the anterior border of the supraspinatus tendon to the posterior border of the infraspinatus tendon, not inserting directly onto the bone. We believe that the semicircular bands of Kolts et al, Sappey, and Debierre; the transverse band described by Clark et al[9,10]; the rotator cable described by Burkhart et al[6,7]; and the circular fiber system described by Gohlke et al[30] are all one and the same. The superior glenohumeral ligament as such was first described by Flood,[25] and Welcker[97] also mentioned a fifth glenohumeral ligament, the "ligamentum interarticulare seu teres humeri." Delorme[16] was convinced that both described the same structure.

**Table III** Functional anatomy of superior complex: Coracoglenohumeral ligament and posterosuperior glenohumeral ligament

| Ligament | Rotation | Adduction (0° and 30°) | Abduction (60° and 90°) |
|---|---|---|---|
| CGHL | Internal | Folded and slack, arthroscopically visible band | Folded and slack |
| | Neutral | Unfolded | Unfolded at 60°, folded at 90° |
| | External | Taut, arthroscopically smooth appearance | Taut only in extreme external rotation |
| PSGHL | Internal | Taut, arthroscopically smooth appearance | Taut only in extreme external rotation |
| | Neutral | Unfolded | Unfolded at 60°, folded at 90° |
| | External | Folded and slack, arthroscopically visible band | Folded and slack |

*CGHL*, Coracoglenohumeral ligament; *PSGHL*, posterosuperior glenohumeral ligament.

The superior glenohumeral ligament seems to be a relative constant in anatomic[13,22,47,86] and orthopaedic literature.[17,57,84,88] Some authors only recognize the coracohumeral ligament and omit the superior glenohumeral ligament from their description, either because they do not observe it or because they consider both ligaments as 2 parts of the coracohumeral ligament.[45,49] Kocher[45] actually described a Y-formed coracohumeral ligament. The superior root had an origin that was consistent with that of the superior glenohumeral ligament but an insertion corresponding with that of the coracohumeral ligament.

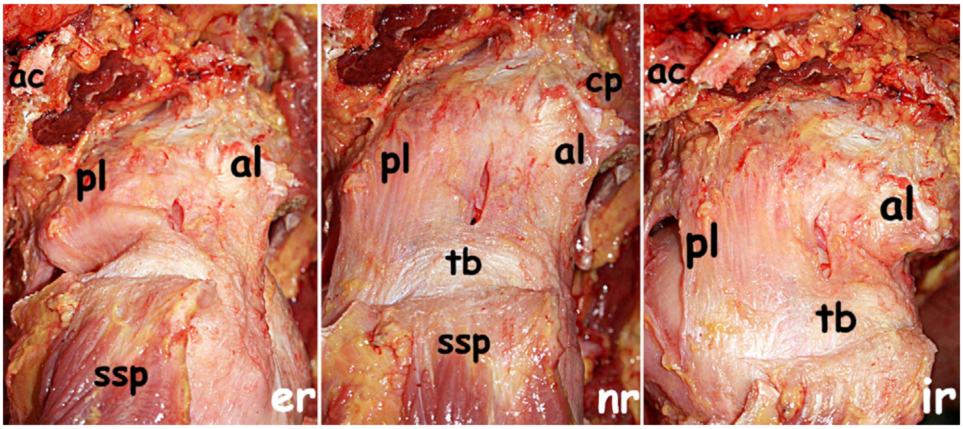


**Figure 8** Functional anatomy of a broad superior complex with a small gap between anterior limb (*al*) and posterior limb (*pl*). In internal rotation (*ir*), the anterior limb (*al*) is folded and the posterior limb is taut; in neutral rotation (*nr*), both limbs are unfolded and taut when the humeral head is allowed to subluxate inferiorly; in external rotation (*er*), the anterior limb (*al*) is taut and the posterior limb (*pl*) is folded. The transverse band (*tb*) does not change in appearance during rotation. *ac*, Acromion; *ssp*, supraspinatus; *cp*, coracoid process.

The anterior root of Kocher's coracohumeral ligament actually corresponded with the usual description of the superior glenohumeral ligament. More recently, both ligaments have again been described as 2 separate entities of variable configuration and with a variable extent of fusion.[2,5,18,21,43,70,77,86] In texts on arthroscopic anatomy, of course, only the superior glenohumeral ligament is described.[4,8,55]

From our own dissections, we can conclude that the coracohumeral, coracoglenoid, and superior glenohumeral ligaments do exist as separable entities in most specimens. However, they are thin and broad sheetlike structures, rather than cordlike. Although only 4 capsules were studied histologically, the coracohumeral and superior glenohumeral ligaments do correspond with longitudinally organized collagen fiber bundles. As the extent of merging, fusion, or even confluence of these 3 ligaments showed considerable variations, we think that it might be better to consider them together as 1 ligamentous structure with variable parts. This also makes sense from a functional point of view, as will be discussed later. This combined structure might then more aptly be labeled as the coracoglenohumeral ligament. In 90% of specimens, we were able to dissect an additional fibrous sheet that was histologically confirmed as being composed of parallel collagen fiber bundles in the posterosuperior area of the glenohumeral capsule. In the remaining specimens, we failed to identify this structure, which we would propose to call the posterosuperior glenohumeral ligament. In these cases, the coracohumeral–superior glenohumeral ligament complex was usually very broad. This configuration may correspond with the rare variant described by Pradhan et al,[75] in which a thickened superior glenohumeral ligament crossed over the biceps tendon to insert on the glenoid neck and labrum posteriorly. Otherwise, although we consulted many anatomic texts and textbooks of the 19th and 20th centuries, no reference to our posterosuperior structure was found. Further dissection of the posterosuperior part of the glenoid neck and inspection of dry bone scapulae showed that there are bony irregularities at the site of insertion of this posterosuperior glenohumeral ligament (Figure 11). This corroborates the hypothesis that the attached ligament is a substantial fibrous structure subjected to tension and stress.

A few biomechanical studies have been devoted to the coracoglenohumeral ligament.[5,36,50,64,67,68,94] In addition, it has received more attention because of its importance for rotator cuff interval lesions[23,24,32,61] and for adhesive capsulitis.*

Boardman et al[5] determined the tensile properties of the coracohumeral and superior glenohumeral ligaments and unequivocally proved that both can be considered ligaments because they are able to withstand tensile forces. The coracohumeral ligament has tensile properties that are about 1.5 times those of the inferior glenohumeral ligament, whereas those of the superior glenohumeral ligament are similar to those of the inferior glenohumeral ligament. We share the opinion of Cooper et al[11] that the coracohumeral and superior glenohumeral ligaments were probably cut and evaluated together in most biomechanical studies. The coracoglenohumeral ligament limits external rotation in adduction[16,21,32,34,58,88] and aids in restraining the humeral head against anterior displacement, probably by causing an obligate posterior translation in this position. Delorme[18] believed that the

*References 3, 33, 38, 39, 53, 58-60, 62, 65, 66, 69, 71-73, 83, 91-93, 99.

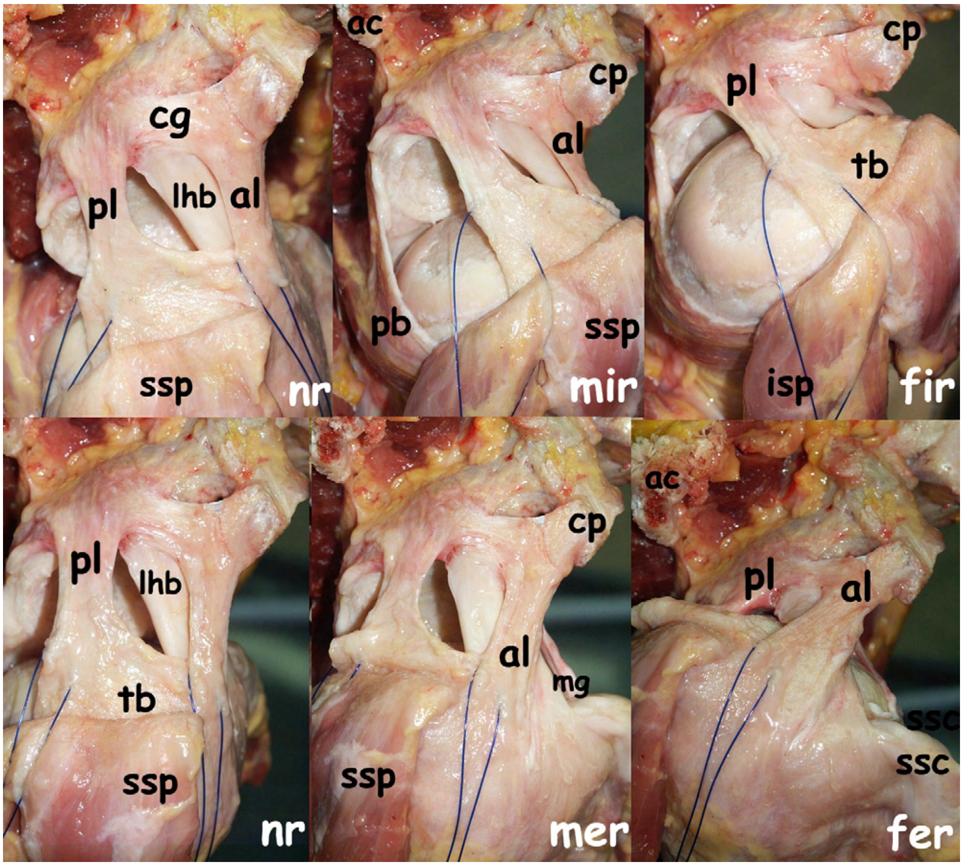


**Figure 9** Functional anatomy of a superior complex with a broad gap between anterior limb (*al*) and posterior limb (*pl*) and with a distinct coracoglenoid ligament (*cg*). In neutral rotation (*nr*), both limbs are unfolded and taut when the humeral head is allowed to subluxate inferiorly; in mid internal rotation (*mir*), the anterior limb (*al*) is folded and the posterior limb is taut; in full internal rotation (*fir*), the anterior limb (*al*) is almost doubled up whereas the posterior limb (*pl*) limits further internal rotation; in mid external rotation (*mer*), the anterior limb (*al*) is taut and the posterior limb (*pl*) is folded; in full external rotation (*fer*), the anterior limb (*al*) limits further external rotation with the posterior band being doubled up. The transverse band (*tb*) and the separate coracoglenoid ligament (*cg*) do not change in appearance during rotation. *lhb*, Tendon of long head of biceps; *ssp*, supraspinatus; *ac*, acromion; *cp*, coracoid process; *pb*, posterior band of inferior glenohumeral ligament; *isp*, infraspinatus; *mg*, middle glenohumeral ligament; *ssc*, subscapularis.

coracohumeral ligament reached its maximal limiting function when the humerus was externally rotated, adducted, and retroflexed. Most authors agreed that the coracohumeral ligament limited external rotation in the lower ranges of abduction (up to 60°) and did not play a role in internal rotation.[21,58] Gagey et al[26,27] discovered that the coracohumeral ligament limited flexion of the humerus to, on average, 75° when the humerus was in neutral rotation. In external rotation, the coracohumeral ligament became tight at an earlier degree of flexion, and in internal rotation, the coracohumeral ligament was under tension at a later level of flexion. Lee et al[52] stated that the anterior band of the coracohumeral ligament should become taut with increasing external rotation whereas the posterior band should tighten with increasing internal rotation. Their posterior band may correspond with our posterosuperior glenohumeral ligament. In the biomechanical study of Kuhn et al,[50] cutting the coracohumeral ligament had the same effect of increasing external rotation in the abducted position as cutting the entire inferior glenohumeral ligament. Either or both parts of the coracoglenohumeral ligament have been attributed a role in preventing inferior displacement of the humeral head.[20,21,32,34,36,51,67,95] Helmig et al[36] noted that no inferior humeral head displacement occurred in internal rotation when the coracohumeral ligament, the anterosuperior glenohumeral capsule, and the subscapularis were cut as long as the posterosuperior capsule was intact. As soon as this area, which corresponds with our posterosuperior glenohumeral ligament, was transected too, inferior displacement was no longer restrained in internal rotation. This study underlines the importance of the

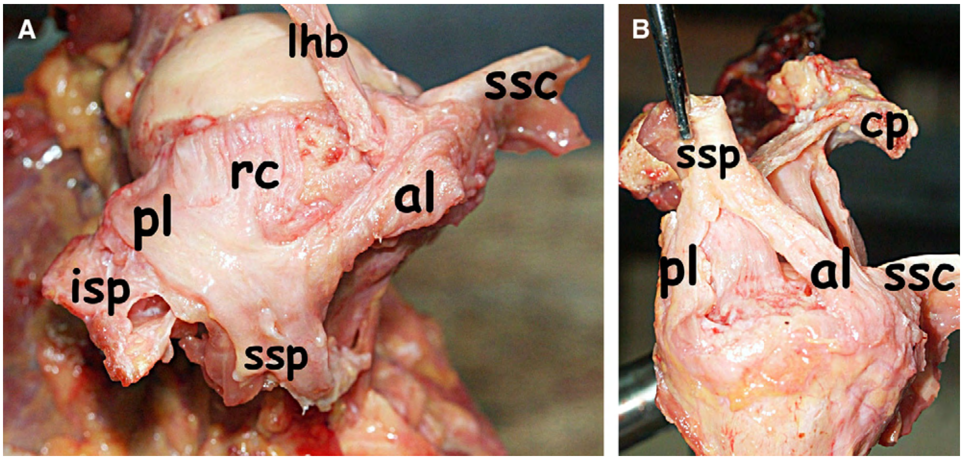


**Figure 10** Specimen with a massive supraspinatus (*ssp*) tendon tear spanning entire area of rotator crescent (*rc*): top view on articular side (**A**) and top view on extra-articular side (**B**). The stump of the supraspinatus muscle was adherent to the superior complex whose transverse band, with the anterior limb (*al*) and the posterior limb (*pl*), was still firmly anchored into the humeral head. The joint was still well centered, and there were no overt signs of rotator cuff arthropathy. The tendon of the long head of the biceps (*lhb)* was also intact. *ssc*, Subscapularis; *isp*, infraspinatus; *cp*, coracoid process.

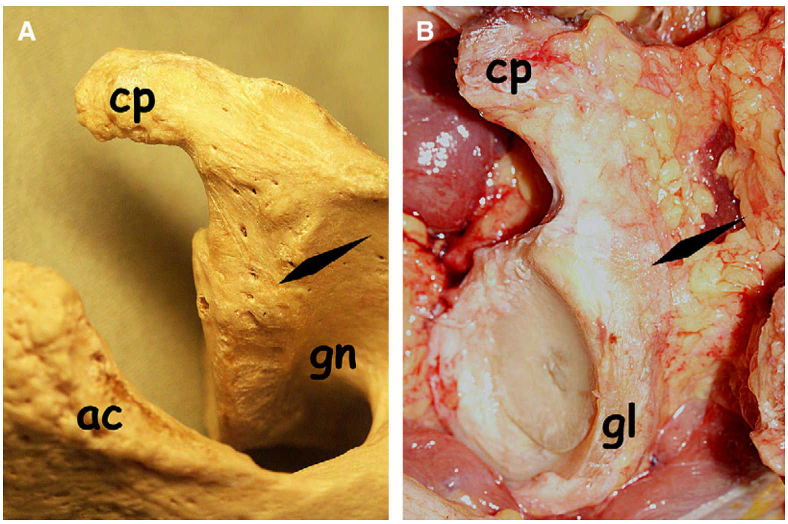


**Figure 11** Photographs of posterosuperior aspect of glenoid neck (*gn*) of a dry bone scapula (**A**) and of a completely dissected specimen (**B**). The *arrows* indicate the site of attachment of the posterosuperior glenohumeral ligament that corresponds with the presence of bony irregularities. *cp*, Coracoid process; *ac*, acromion; *gl*, glenoid labrum.

posterosuperior glenohumeral ligament in internal rotation, even up to 60° of abduction. External rotation is limited when the coracoglenohumeral ligament is shortened by imbrication[21,34,61] or by closing the rotator cuff interval[23,32] or is clinically contracted, as is the case in adhesive capsulitis.[†] In contrast, cutting the coracoglenohumeral ligament results in increased external rotation, just as release of this contracted area results in restoration of external rotation in patients with frozen shoulder. Open release[33,53,58-60,66,69] or arthroscopic release[‡] of the rotator cuff interval, including sectioning of the coracohumeral (and superior glenohumeral) ligament, has been reported as a successful procedure in case of recalcitrant stiffness in patients with adhesive capsulitis. Not all authors

[†]References 3, 33, 38, 39, 53, 58-62, 65, 66, 69, 71-73, 83, 91-93, 99.

[‡]References 3, 33, 38, 39, 65, 71-73, 83, 91-93, 99.

extend their release to the coracohumeral ligament.[65,73,99] Although internal rotation is also limited in patients with adhesive capsulitis, it is rarely addressed surgically. Some authors have suggested that the posterior capsule should be released when limitation of internal rotation and flexion is present.[24,28,91,93] The group of Bunker and colleagues[3,66] specifically extended their release to the posterosuperior aspect of the glenohumeral capsule up to the 9-o'clock position, because they think that this accelerates recuperation of internal rotation, although they do not report its range after release. From our study, we know that they actually release the posterosuperior glenohumeral ligament. As this ligament restricted internal rotation in our specimens, it seems logical that release of contraction in this area would result in a gain in internal rotation. Segmuller et al[83] only released the inferior capsule and noted a marked increase in external rotation but no gain in internal rotation in half of their patients.

Lesions of the rotator cuff interval have mainly been described in patients with subtle instability or anterosuperior impingement, although they can be associated with other capsuloligamentous lesions and more overt instability as well.[1,2,23,24,57,61,78,79] This lesion has been defined as a small to large opening between the superior and middle glenohumeral ligament. We would prefer to caution the surgeon in interpreting any opening between these 2 ligaments as a rotator interval lesion, as our study has shown that the normal interval or Weitbrecht's foramen can be quite variable in size. Tetro et al[87] quantified the interval between the supraspinatus and the subscapularis at the level of the glenoid rim in normal cadavers at 21.6 mm (range, 17-26 mm). Although they actually measured the intratendinous interval, these values are comparable to our measurements of the intraligamentous gap. Closure of an interval that actually is normal may induce an undesired limitation of external rotation. When closing the interval, external rotation should, therefore, always be checked to avoid this complication. An efficient coracoglenohumeral ligament results in an anterosuperior fold appearing during external rotation, and the middle glenohumeral ligament also becomes unfolded and taut[74] in this position. Possibly, disappearance of this folding-unfolding mechanism may be an indication of pathology, either contracture or excessive laxity. This hypothesis needs to be studied in a clinical series.

On the lateral side, several authors have shown that the coracohumeral ligament–superior glenohumeral ligament complex inserting around the bicipital groove was instrumental in retaining the biceps tendon and in preventing its subluxation.[2,21,98] We believe that the coracoglenohumeral ligament inserts onto both margins of the groove together with the anterior limb of the rotator cable. The tendons of the supraspinatus and of the subscapularis rarely cover the groove. Werner et al[98] reported that the fasciculus obliquus makes an important contribution to the biceps pulley together with the superior glenohumeral ligament. Lesions of the so-called biceps pulley may be destabilizing for 2 reasons: either because the long tendon of the biceps may subluxate or because the head-depressing effect of the rotator cable is diminished. Anterosuperior internal impingement has been associated with these biceps pulley lesions, as well as anterior articular-sided partial-thickness rotator cuff tears and anterosuperior labral lesions.[19,29,31,41,42,44,76,85] Posterosuperior internal impingement has been associated with posterior articular-sided partial-thickness rotator cuff tears and posterosuperior labral lesions.[14,40-42,54,56,81,90] We believe that truly superficial articular-sided rotator cuff tears actually represent damage to the superior complex rather than to the rotator cuff tendons themselves. This damage compromises the head-depressing and -centering effect that the superior complex normally has. The findings in the small number of specimens with rotator cuff tears in our series suggest that, when the superior complex remains intact or is only partially damaged, it may limit retraction of the rotator cuff tendon. This effect was already shown in the studies of Burkhart et al[6,7] by proving that the rotator cable was pivotal in maintaining normal kinematics in the presence of massive rotator cuff tears. Burkhart et al, Clark et al,[9,10] and Kolts et al[48] failed to recognize that the coracoglenohumeral and posterosuperior glenohumeral ligaments merge into their rotator cable or transverse band. We are convinced that both ligaments provide the essential medial anchoring for the function of the rotator cable. Without this medial origin, the function of the rotator cable would only be dependent on the insertion of its anterior and posterior limbs on the humerus and the mingling of its fibers with those of the rotator cuff tendons. With the additional medial anchoring, the depressing and centering effect of the superior complex can probably be maintained as long as one of the medial points of bony attachment and one of the lateral points are preserved. Because of its head-centering effect, the reciprocal tightening of its anterior and posterior limbs with rotation, and its 4-point anchorage, we believe that the superior complex functions in the same way as the hammock formed by the inferior glenohumeral ligament complex. The humeral head is suspended on the superior complex and cradled by the inferior complex. The superior complex appears to be more efficient in adduction, although it may serve as a secondary restraint in abduction. The inferior complex is more efficient in abduction and may act as a secondary restraint in adduction. Furthermore, the superior and inferior complexes may be linked to each other medially, through the glenoid labrum, as well as laterally, through the fasciculus obliquus. Werner et al[98] already reported that the fasciculus obliquus makes an important contribution to the biceps pulley together with the superior glenohumeral ligament. The potential implications of the superior complex and the linkage between both complexes for glenohumeral stability require further study.

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# Arthroscopic glenohumeral folds and microscopic glenohumeral ligaments: The fasciculus obliquus is the missing link

Nicole Pouliart, MD, PhD,[a,b,c] Katia Somers, MD,[d] and Olivier Gagey, MD, PhD,[c,d,e] *Brussels, Belgium; and Paris and Kremlin-Bicêtre, France*

*This study tested the hypotheses that the folds in the inferior glenohumeral capsule appear at the borders and crossings of the underlying capsular ligaments and that embalming may result in misinterpretation of these folds as ligaments. The inferior capsular structures in 80 unembalmed cadaver shoulders were compared with 24 embalmed shoulders. During arthroscopy and dissection, an anteroinferior fold was more prominently seen in internal rotation and was almost obliterated in external rotation. A posteroinferior fold appeared in external rotation and almost disappeared in internal rotation. During dissection, the anteroinferior fold developed at the border of the anterior band of the inferior glenohumeral ligament (ABIGHL) and where this ligament crossed with the fasciculus obliquus (FO). Several patterns of crossing of the ABIGHL and the FO were seen that determined the folding-unfolding mechanism of the anteroinferior fold and the appearance of possible synovial recesses. The axillary part of the IGHL is formed by the FO on the glenoid side and by the ABIGHL on the humeral side. The posteroinferior fold was determined by the posterior band of the IGHL. The folds in the embalmed specimens did not necessarily correspond with the underlying fibrous structure of the capsule. The folds and recesses observed during arthroscopy indicate the underlying capsular ligaments but are not the ligaments themselves. The IGHL complex is formed by its anterior and posterior bands and also by the FO. Both findings are important during shoulder instability procedures because the ligaments need to be restored to their appropriate anatomy and tension. Because the FO may also be involved, Bankart-type surgery may have to reach far inferiorly. Midsubstance capsular shift procedures also need to incorporate this ligament.*
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From the Departments of [a]Orthopaedics and Traumatologie and [d]Pathologic Anatomy, Universitair Ziekenhuis Brussel; [b]Department of Human Anatomy, Vrije Universiteit Brussel; [c]Institut d'Anatomie, and [e]Service d'orthopédie, Hôpital Bicêtre, Université Paris-Sud.

Reprint requests: Nicole Pouliart, Universitair Ziekenhuis Brussel, Laarbeeklaan 101, 1090 Brussels, Belgium (E-mail: *nicole.pouliart@uzbrussel.be*).



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A previous study[19] demonstrated that the folds of the anterior glenohumeral capsule, usually observed during arthroscopy, are dependent on the position of the humeral head in relation with the glenoid. In certain positions, as well as when flattening out the harvested capsule, the folds disappear altogether. We believe that these folds arise at the borders of the glenohumeral ligaments, as described by O'Brien et al[17] and Turkel et al,[26] and where the fibers of these ligaments cross each other, as demonstrated by Gohlke et al.[7]

The aim of the present work was to try to link the macroscopically visible folds with an anatomic substrate, because this was precluded by the experimental setup of the initial study. A combination of dissection, arthroscopy, and histologic examination was used to verify our hypothesis. In addition, the morphology of the glenohumeral ligaments in embalmed specimens was compared with that in unembalmed shoulders to study the hypothesis that embalming may result in misinterpretation of folds as ligaments.

## MATERIAL AND METHODS

Eighty unembalmed cadaver shoulders (aged 50 to 90 years) and 24 embalmed cadaver shoulders (age unknown) were studied. Thirty-six specimens were first inspected arthroscopically through a standard posterior portal, and the folds, representing the middle and the inferior glenohumeral ligaments, were visualized. The folding-unfolding mechanism was verified to be certain that removal of the overlying soft tissue envelope did not change its appearance. The anterior capsule and the inferior glenohumeral complex were inspected with the arm in 0° to 90° of abduction and in maximal internal and external rotation at intervals of 30° of abduction. All soft tissues superficial to the rotator cuff were removed in all specimens.

In the 36 arthroscopic specimens, the arthroscope was reinserted through the same portal, and the functional testing

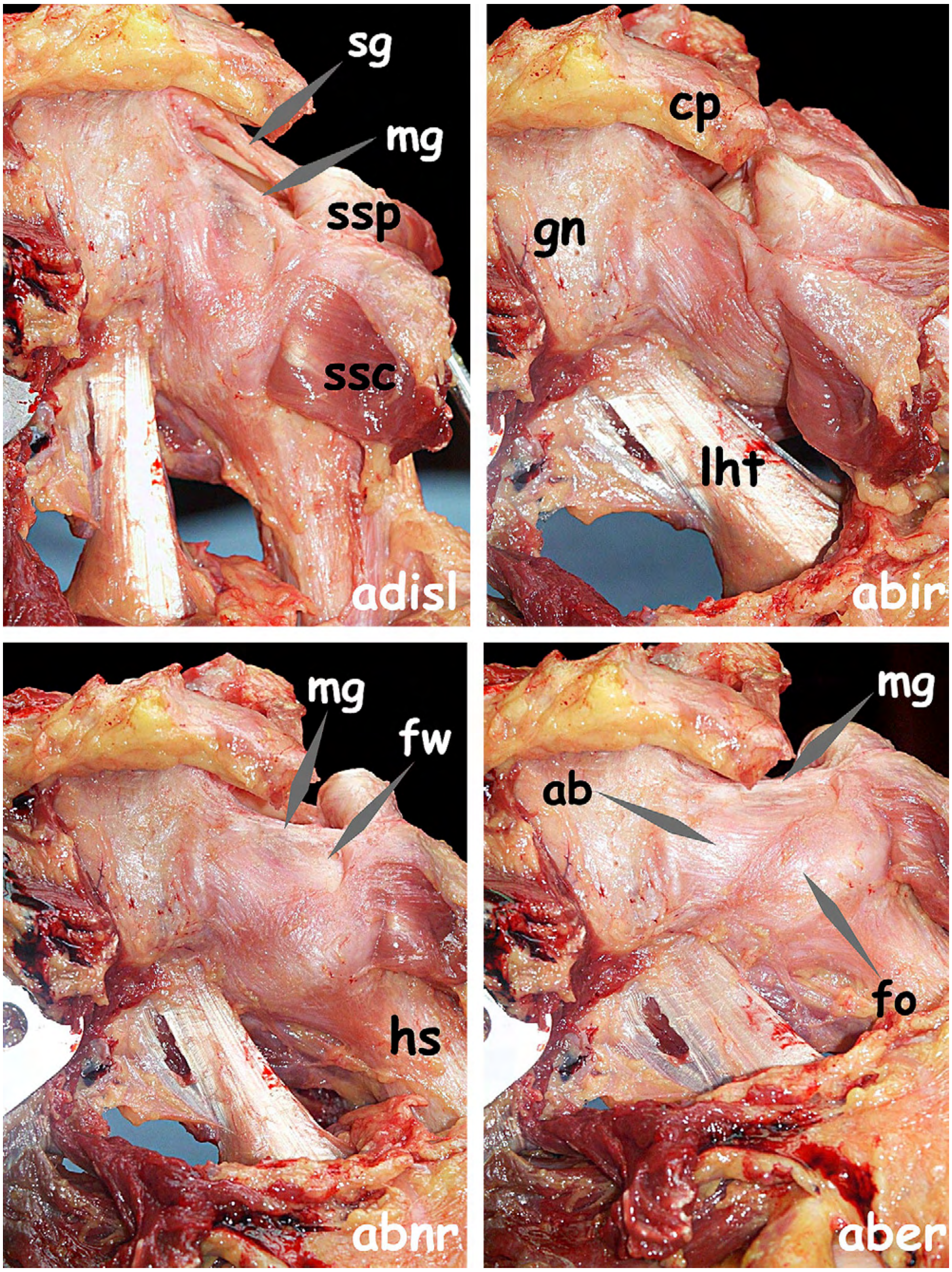


**Figure 1** Appearance of the anterior glenohumeral capsule before dissecting out the fibrous structure. **Top left,** The humerus is in adduction and inferiorly subluxated *(adisl)* due to gravity. In the other 3 images, the humerus is in **(top right)** 60° of abduction and internal rotation *(abir)*, **(bottom left)** neutral rotation *(abnr)* or **(bottom right)** external rotation *(aber)*. *fo,* Fasciculus obliquus; *ab,* anterior band of the inferior glenohumeral ligament; *mg*, middle glenohumeral ligament; *sg,* superior glenohumeral ligament; *fw,* foramen of Weitbrecht; *hs,* humeral shaft;*cp,* coracoid process; *gn,* glenoid neck; *lht,* tendon of the long head of the triceps; *ssc,* subscapularis; *ssp,* supraspinatus.

procedure was repeated. The clearly visible folds that developed in varying arm positions, representing the anterior and posterior parts of the inferior glenohumeral ligament complex and the middle glenohumeral ligament, were marked with arthroscopically placed suture loops. The subscapularis, supraspinatus, infraspinatus, and teres minor were transected medially and detached from their scapular origin. The rotator cuff muscles and tendons were carefully separated from the underlying capsule up to the point where the tendons and capsule merge. Great care was taken not to damage the capsule or the suture marker loops.

The humerus was moved from adduction to abduction and through its full range of rotation at intervals of 30° of abduction. In each of these positions, the appearance of folds on the outside of the capsule was noted, and the site of these folds was related to the marker loops or, in the nonarthroscopic group, marked for further reference. Finally, the loose connective tissue around the fibrous structure of the capsule was carefully removed under loupe magnification by a combination of blunt and sharp dissection. The humerus was again moved through its range of motion to verify that the folds still appeared at the same site as when the capsular ligaments were not yet dissected free. In 4 shoulders, 2 each from the arthroscopic and nonarthroscopic groups, the rotator cuff and the underlying capsule were harvested in toto for microscopic evaluation.

In the embalmed specimens, the surrounding soft tissues and the rotator cuff were removed in the same manner as described for the fresh shoulders. Functional anatomy could, of course, not be studied because of the stiffness of the soft tissues. The interior side of the capsule was first inspected through the foramen of Weitbrecht; thereafter, the posterosuperior capsule was removed as well as the humeral head, as described by Schlemm.[21] The folds visible in the embalmed specimens were compared with those in the fresh specimens.

For the histologic study, the 4 harvested pieces were cut into thin strips approximately 5 mm wide and 30 mm long. In 2 specimens, these strips were oriented parallel to the glenoid circumference, and in the other 2, the strips were oriented perpendicular to the glenoid. The capsular segments were embedded in paraffin, and 3 sequential 4-$\mu$m sections were made from each paraffin block. The sequential slides from each block were stained using hematoxylin and eosin, Masson trichrome, and van Gieson stains. The slides were examined using a light microscope at original magnification $\times 40$, $\times 100$, and $\times 200$. Orientation of the collagen fibers was documented, and relevant areas were digitally photographed.

## RESULTS

### *Dissection and functional anatomy*

After removal of the surrounding muscles, but before removal of the loose connective tissue, the anterior and anteroinferior capsule from the outside looked like a smooth, homogenous cuff. The next section describes the macroscopic appearance of the capsule with the arm in neutral rotation and 0° abduction. This position is relevant for describing the orientation of the various components. At the level of the coracoid process, the foramen of Weitbrecht was always observed, although it varied considerably in size and extent. It had a maximum width of 13 to 25 mm at its lateral border. Medially, this foramen expanded into the subcoracoid or subscapular bursa.

After meticulous dissection of the fibrous structure of the capsule and removal of the bursa, the limits of the foramen could be discerned more clearly. Laterally, it tapered and was limited by the tendon of the subscapularis and the middle glenohumeral ligament inferiorly, by the tendon of the supraspinatus and the superior glenohumeral ligament superiorly, and by the fasciculus obliquus and the middle glenohumeral ligament laterally. In this series of 100 specimens, only 4 had a foramen of Rouvière that appeared to be located between the middle and inferior glenohumeral ligaments. A small inferior subscapular bursa was noticed in 2 of these 4 specimens (Figure 1).

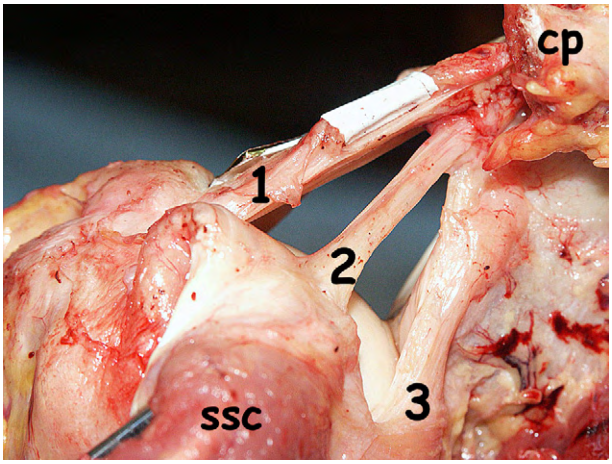


**Figure 2** The Z-like structure of the anterior glenohumeral capsule formed by the superior glenohumeral ligament with the coracohumeral ligament *(1)*, the middle glenohumeral ligament *(2)*, and the inferior glenohumeral ligament *(3)*. The coracohumeral ligament and the superior glenohumeral ligament merged in their middle third and are separated by the white marker paper. *cp,* Coracoid process, *ssc,* subscapularis.

In 5 specimens, the middle glenohumeral ligament was very thin and nearly vestigial. In all other shoulders, this ligament could always be dissected as a distinct fibrous structure of somewhat variable size; at its midportion, the width ranged from 6 to 12 mm. On the glenoid side, it arose from the glenoid neck directly medial to the labrum and was attached to the labrum in most cases. The origin on the glenoid neck was between the 2- and 12-o'clock position for a right shoulder. Between the 12- and 1-o'clock position, there usually was some overlap with the superior glenohumeral ligament, which arose more medially on the glenoid neck. In 10% of cases, the origins of the middle and the superior glenohumeral ligaments could only be separated by sharp dissection. Laterally, the middle glenohumeral ligament inserted on the lesser tuberosity of the humerus together with the superior part of the subscapularis tendon. Over the lateral 2 cm, it was impossible to separate the ligament from the tendon.

In addition, the lateral part of the fibrous structure of the middle glenohumeral ligament intermingled with the superolateral continuation of the fasciculus obliquus to the anterior ridge of the bicipital groove. Here, it seemed to be continuous with the transverse humeral ligament of the bicipital groove. Some intermingling with fibers from the transverse band (rotator cable) and the superior glenohumeral and the coracohumeral ligaments seemed to occur. Figure 2 illustrates the classic Z-like pattern of the anterior glenohumeral capsule.

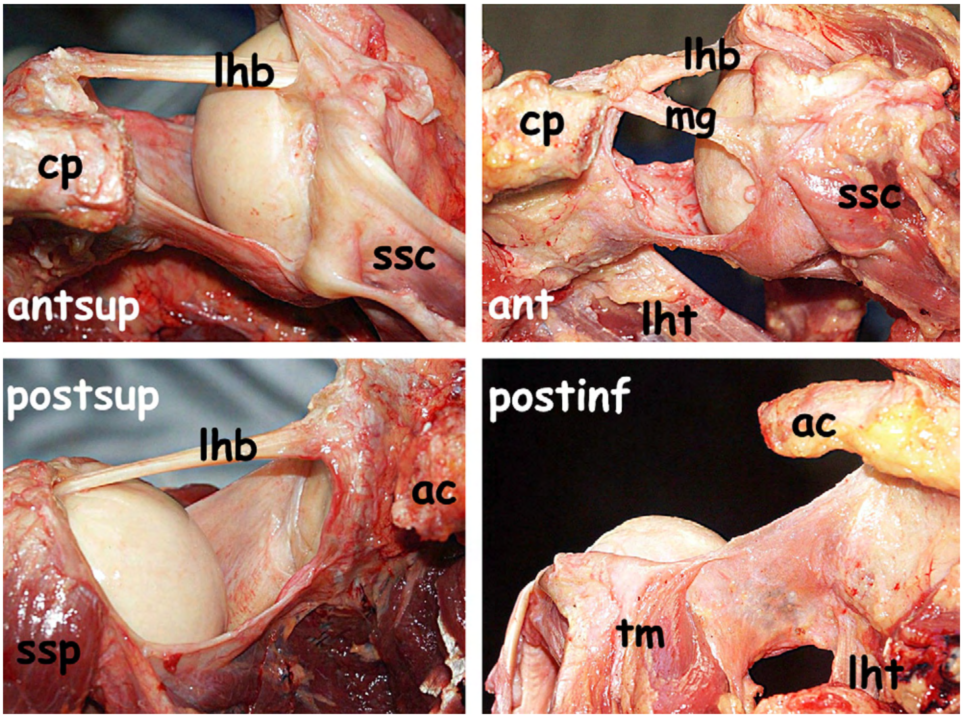


**Figure 3** The hammock-like inferior glenohumeral ligament-complex cradles the humeral head. **Top left,** Anterosuperior *(antsup)*, **(top right)** posterosuperior *(postsup)*, **(bottom left)** anterior *(ant)* and **(bottom right)** posteroinferior *(postinf)* views. *mg,* Middle glenohumeral ligament; *ssc,* subscapularis; *ssp,* supraspinatus; *isp,* infraspinatus; *lhb,* tendon of the long head of the biceps; *lht,* tendon of the long head of the triceps; *tm,* teres minor; *cp,* coracoid process; *ac,* acromion.

The fasciculus obliquus also inserted on the humeral neck to form the exterior part of the anterior limb of the classic V-like insertion of the inferior capsule between 9 and 6 o'clock for a right shoulder. The fasciculus obliquus arose from the glenoid labrum, from which it could not be separated, but it also had a broad origin from the tendon of the long head of the triceps. This tendinous connection may be up to 1.5 cm wide in the medial-to-lateral direction.

The origin of this part of the inferior capsule on the glenoid side therefore had a somewhat triangular form, with the base attached to the glenoid labrum between 4 to 5 and 7 o'clock for a right shoulder. In the neutral position, it adopted a broad, trapezoidal shape running diagonally from inferomedially to superolaterally, as well as inferolaterally. Here, it lay anterior to, and crossed over, the anterior part of the inferior glenohumeral ligament (the superior band of Turkel et al[26] and the anterior band of O'Brien et al[17]).

The anterior band of the inferior glenohumeral ligament arose from the glenoid labrum between 6 to 5 and 3 o'clock for a right shoulder. It coursed slightly diagonally downward to its humeral insertion together with the fasciculus obliquus, where it formed the interior part of the anterior limb of the V-shaped insertion. Because of the crossing of both structures, the anterior band of the inferior glenohumeral ligament appeared triangular in shape when viewed from the outside in neutral rotation. The anterior band of the inferior glenohumeral ligament and the fasciculus obliquus could not be separated from each other in the region where they crossed.

The posterior leg of the V-shaped inferior humeral insertion was formed by the posterior part of the inferior glenohumeral ligament. On the medial side, this part arose from the glenoid labrum between 6 and 8 to 9 o'clock for a right shoulder. Inferomedially, there seemed to be some intermingling and crossing of fibers of the posterior part of the inferior glenohumeral ligament and fibers of the fasciculus obliquus. The posterior part of the inferior glenohumeral ligament became progressively thinner superiorly, although loupe magnification and meticulous dissection still allowed discernment of its superior border adequately. Figure 3 illustrates the hammock-like appearance of the inferior glenohumeral ligament complex.

The relationship between the fasciculus obliquus and the anterior band of the inferior glenohumeral ligament could be divided into 6 patterns, where the superolateral border of the anterior band crossed the superior border of the fasciculus obliquus (Figure 4):

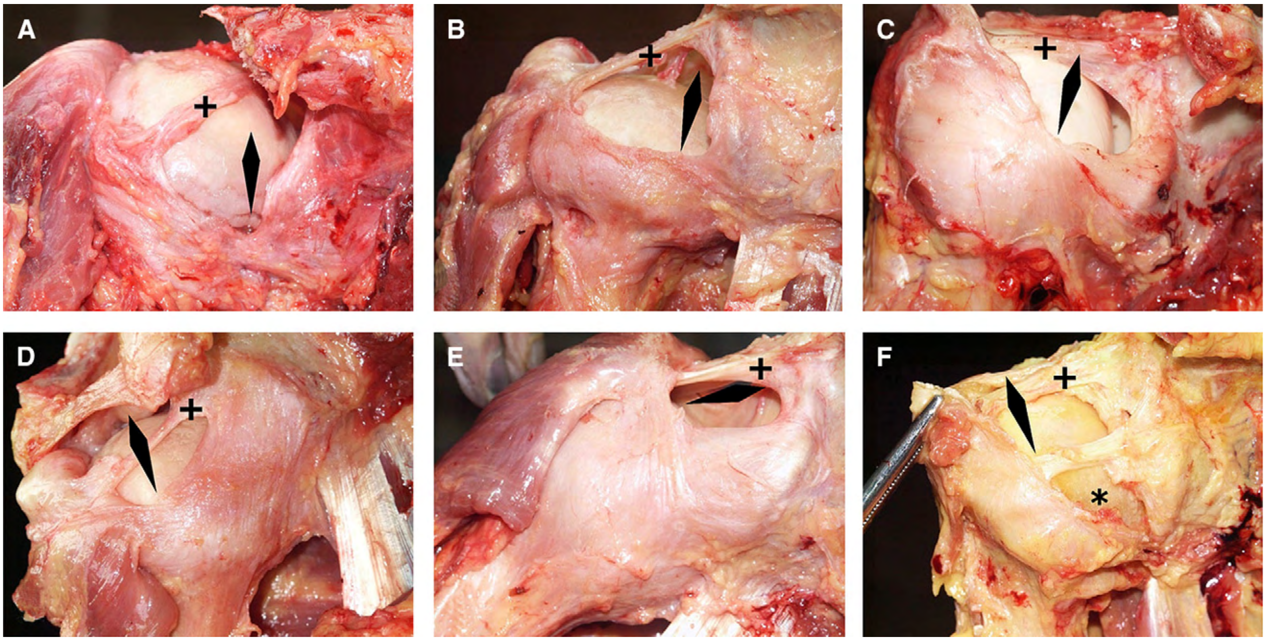


**Figure 4** Types of inferior glenohumeral ligament-complexes: **(A)** no anterior band of the inferior glenohumeral ligament (ABIGHL) visible, possibly due to degeneration, relatively thin middle glenohumeral ligament (MG, indicated with **+**), with its origin far superiorly away from the fasciculus obliquus (FO); **(B)** very small triangle of the ABIGHL visible, ABIGHL and MG are far apart; **(C)** thin strip of the ABIGHL is visible, with a relatively broad origin for the MG nearing that of the ABIGHL; **(D)** relatively broad ABIGHL that crosses the FO in the middle third; **(E)** broad ABIGHL that crosses (almost) the entire length of the FO; and **(F)** thin ABIGHL that crosses the FO in the middle third and with a gap between both. The gap *(*)* corresponds with a foramen of Rouvière. The *black arrow* indicates the area where the ABIGHL and the FO cross.

1. anterior band absent as a fibrous sheet, perhaps because of degeneration of the capsule (3 of 76 specimens, Figure 4, *a*);
2. small strip of anterior band visible from the exterior, crossing borders in the medial third (11 of 76 specimens, Figure 4, *b*);
3. intermediate anterior band, crossing borders in the middle third (33 of 76 specimens, Figure 4, *c*);
4. broad anterior band, crossing borders in the lateral third (19 of 76 specimens, Figure 4, *d*);
5. very broad anterior band, crossing with the fasciculus obliquus over its entire length (3 of 76 specimens, Figure 4, *e*); and
6. anterior band crossing the fasciculus obliquus in the lateral third, but with a gap between them; the distinct foramen of Rouvière in these shoulders corresponded with this gap, rather than with the gap between the AB and the middle glenohumeral ligament (4 of 76 specimens, Figure 4, *f*).

Microscopic evaluation confirmed that the macroscopically observed ligamentous structures corresponded with discrete areas of organized collagen fiber bundles (Figure 5).

The positioning of the glenohumeral ligaments and their folding-unfolding mechanism during functional testing is summarized in Table I. Figures 6 to 8 illustrate this mechanism from the anterior side for 3 different types of the anteroinferior complex and Figure 9 from the posterior side, for the posterior band of the inferior glenohumeral ligament.

### *Arthroscopy*

By moving the arm from adduction to abduction and varying the degree of external or internal rotation, the limits of the middle glenohumeral ligament and of the posterior and anterior parts of the inferior glenohumeral ligament could always be discerned. When the humerus was positioned in maximal internal rotation and about 30° of abduction, delineation and subsequent suture loop marking of the middle glenohumeral ligament and the anterior band of the inferior glenohumeral ligament were facilitated. For easier delineation and marking of the posterior part of the inferior glenohumeral ligament, the humerus was best positioned in about 30° of abduction and maximal external rotation. As described previously, the folds indicating the underlying ligamentous fibrous components of the glenohumeral capsule disappeared when the humerus was positioned in the opposite direction; that is, maximal external rotation in abduction for the

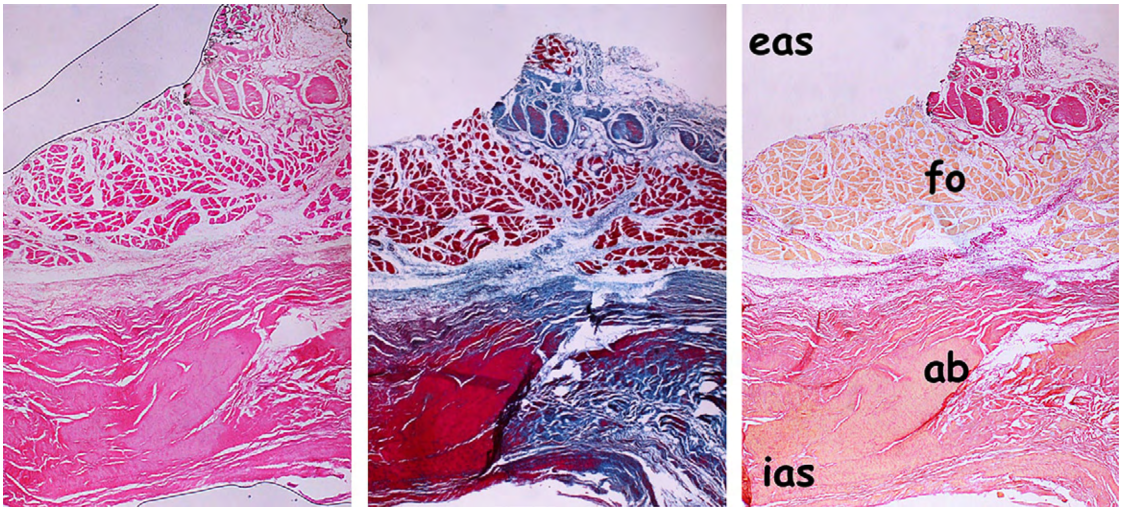


**Figure 5** Digital photographs of light microscopic examination of the inferior glenohumeral ligament-complex where the fasciculus obliquus and the anterior band of the inferior glenohumeral ligament cross each other. Images stained with **(left)** hematoxylin-eosin **(middle)** Masson-trichrome, and **(right),** von Giesson stain are shown at original magnification ×40. On the intraarticular side *(ias)*, thick parallel bundles of collagen fibers with a radial orientation are running parallel with the cut. These correspond with the anterior band of the inferior glenohumeral ligament *(ab)*. On the exterior extraarticular side *(eas)*, thick parallel bundles of collagen fibres with a transverse orientation are perpendicular to the cut. These correspond with the fasciculus obliquus *(fo)*.

**Table I** Functional anatomy of the anterior and inferior glenohumeral ligaments

| Ligament | Rotation | Adduction | Abduction | |
|---|---|---|---|---|
| MGHL | Internal | Folded | Slack | But tight, almost vertical when >90° of abduction |
| | Neutral | Slack | Taut | Folded when >90° of abduction |
| | External | Taut, over upper part of HH | Restricting external rotation, almost horizontal | Folded, over top of HH when >90° of abduction |
| ABIGHL | Internal | Folded, sharply oblique downwards | Folded, almost vertical | In full abduction, MGHL-ABIGHL and FO almost vertical and parallel |
| | Neutral | Unfolded, oblique downwards | Unfolded, horizontal | In full abduction, ABIGHL at 45° to FO |
| | External | Taut, horizontal slightly upwards | Taut, horizontal slightly upwards | In full abduction, ABIGHL and FO horizontal and parallel |
| FO | Internal | Folded, almost vertical | Folded, curved obliquely upwards | |
| | Neutral | Unfolded, almost Horizontal under HH | Unfolded, oblique upwards | |
| | External | Taut, horizontal | Taut, horizontal | |
| PBIGHL | Internal | Slack, more vertical | Taut | |
| | Neutral | Slack, obliquely downwards | Slack | |
| | External | Folded, horizontal under HH | Slack | |

*ABIGHL,* Anterior band inferior glenohumeral ligament; *FO,* fasciculus obliquus; *HH,* humeral head; *MGHL,* middle glenohumeral ligament; *PBIGHL,* posterior band inferior glenohumeral ligament

middle glenohumeral ligament and the anterior band of the inferior glenohumeral ligament, and maximal internal rotation in abduction for the posterior part of the inferior glenohumeral ligament. Because of the more or less pronounced presence of the foramen of Weitbrecht, the upper border of the middle glenohumeral ligament never completely disappeared, regardless of arm positioning. The folds that were marked corresponded with the macroscopically identified ligamentous structures. The anteroinferior fold actually was at

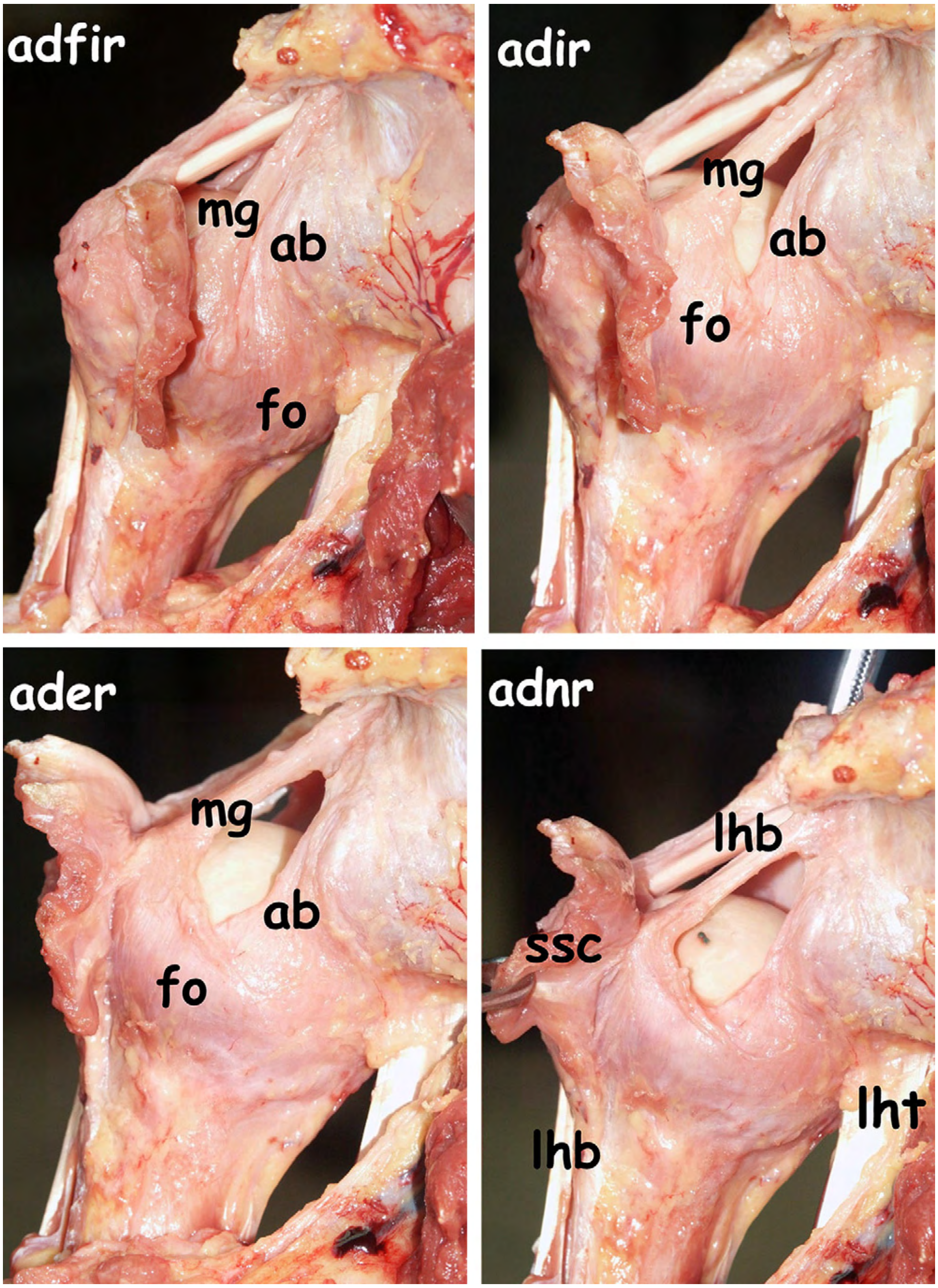


**Figure 6** The folding-unfolding mechanism of the inferior glenohumeral ligament-complex in adduction, with its anterior band (AB) and the fasciculus obliquus (FO) crossing in the middle third, in combination with that of the middle glenohumeral ligament (MG). **Top left,** In full internal rotation *(adfir)*, the 3 ligamentous structures are collapsed onto each other, with the AB and the MG lying parallel and vertical and the fasciculus obliquus folded up and almost horizontal. **Top right,** In internal rotation *(adir)* a small gap appears between the MG and the AB, but both are still vertical and parallel, and the FO is more diagonal. **Bottom left,** In external rotation *(ader)*, the AB is more diagonal, the MG is more diagonal and over the top of the humeral head and the FO is taut and almost vertical. **Bottom right,** In neutral rotation *(adnr)*, the AB is vertical, the MG is diagonal downwards and the FO diagonal upwards. *ab,* Anterior band of the inferior glenohumeral ligament; *fo,* fasciculus obliquus; *mg,* middle glenohumeral ligament; *ssc,* subscapularis; *lhb,* tendon of the long head of the biceps; *lht,* tendon of the long head of the triceps.

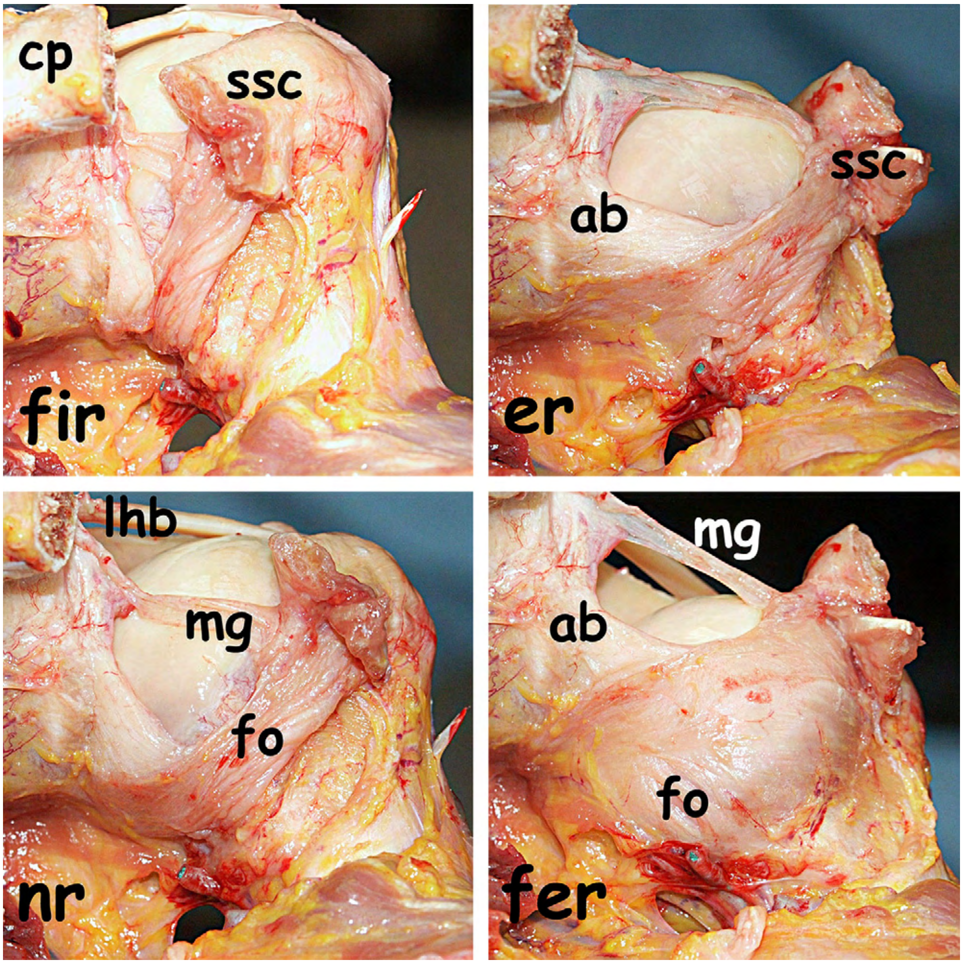


**Figure 7** The folding-unfolding mechanism for an inferior glenohumeral ligament-complex with its anterior band (AB) and the fasciculus obliquus (FO) crossing in the medial third, in combination with that of a thin middle glenohumeral ligament (MG). Anterior view in 30° of abduction. **Top left,** In full internal rotation *(fir)*, the AB and the FO cross at an acute angle and are both almost vertical, and the vestigial MG horizontally closes the triangle. **Top right,** In neutral rotation *(nr)*, the angle of crossing between the AB and the FO is less acute and both are more diagonal, the MG is still horizontal as the base of the triangle. **Bottom left,** In external rotation *(er)*, the angle between the AB and the FO is obtuse, with the AB lying horizontally and the FO still diagonal, and the MG is now parallel to the AB. **Bottom right,** In full external rotation *(fer)*, the AB is horizontal, the FO is spread out in a trapezoidal shape, and the MG is still parallel with the AB but now lies over the top of the humeral head. *ab,* Anterior band of the inferior glenohumeral ligament; *fo,* fasciculus obliquus; *mg,* middle glenohumeral ligament; *ssc,* subscapularis; *lhb,* tendon of the long head of the biceps; *cp,* coracoid process.

the confluence of the fasciculus obliquus and the anterior band of the inferior glenohumeral ligament.

### *Embalmed specimens*

All embalmed specimens showed at least 2 folds, 1 posterior and 1 anterior. Most shoulders also had a third fold anterosuperiorly, and some even had a fourth fold. These folds tended to smooth out slightly with the limited motion possible in these embalmed specimens. Upon further dissection, however, these folds did not necessarily correspond with the underlying fibrous structure of the capsule.

## DISCUSSION

In a previous study,[19] the folding-unfolding mechanism of the anteroinferior and posteroinferior capsular folds was compared with the functioning of a Chinese finger trap. The comparison of the histologic structure of the glenohumeral capsule with this device was originally made by Gohlke et al.[7] Because the capsule in our original study was harvested without dissection and because no specimens were selected for histologic study, it was not possible to relate the capsular folds with the underlying fibrous structure. The first objective of the present study was to rectify this limitation.

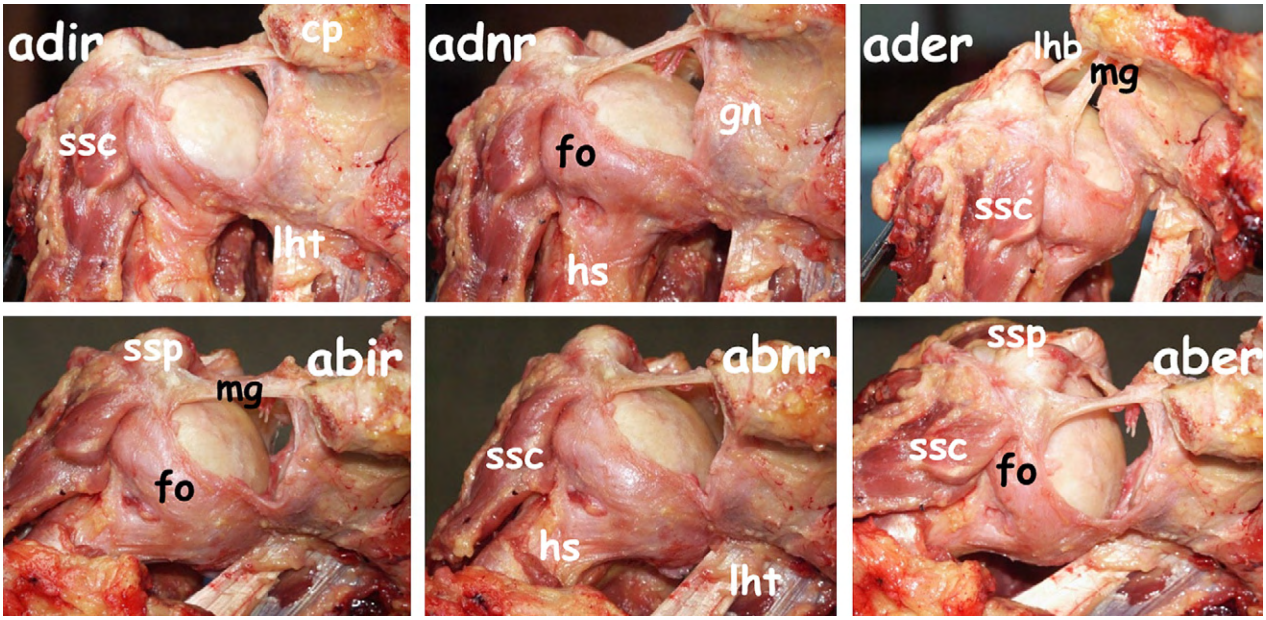


**Figure 8** The folding-unfolding mechanism for an inferior glenohumeral ligament-complex without visible anterior band. **Top left,** Anterior view in adduction with and internal rotation *(adir)*, **(top middle)** neutral rotation *(adnr)*, **(top right)** or external rotation *(ader)*, or **(bottom left)** in 60° of abduction and internal rotation *(abir)*, **(bottom middle)** neutral rotation *(abnr)*, or **(bottom right)** external rotation *(aber)*. *fo;* Fasciculus obliquus; *mg,* middle glenohumeral ligament; *ssc,* subscapularis; *ssp,* supraspinatus; *lhb,* tendon of the long head of the biceps; *lht,* tendon of the long head of the triceps; *cp,* coracoid process; *gn,* glenoid neck; *hs,* humeral shaft.

The second objective was to compare the folds and recesses observed in embalmed specimens with those seen in fresh specimens.

The capsular folds in unembalmed specimens occurred at the borders of the ligamentous reinforcements of the capsule but were not comparable with the capsular ligaments themselves because these were much broader. This confirmed our hypothesis from the original study. Macroscopically, the fasciculus obliquus formed a continuous and confluent sheet of fibrous tissue with the anterior and posterior bands of the inferior glenohumeral ligament in 91% of specimens.

Ferrari[5] examined the glenohumeral capsule from the outside and concluded that the presence or absence of the superior and inferior subscapular bursae determined the observation of the capsular ligaments. DePalma et al[3] and others[7,14,16,17,22] classified the synovial recesses seen on the inside of the anterior capsule. Much variation in the frequency of the types of recesses was observed (Table II). Type I had 1 recess above the middle glenohumeral ligament. Patterns 4 and 5 of the present study would result in the observation of a type I recess. DePalma type II had 1 recess below the middle glenohumeral ligament, and type III had 2 recesses, 1 superior above and 1 inferior below the middle glenohumeral ligament. Patterns 2 and 3 could either result in a type II recess or a type III recess when the interval between the middle and the superior glenohumeral ligament is small. DePalma type IV had 1 large recess above the inferior glenohumeral ligament with an absent middle glenohumeral ligament. Pattern 1 might lead to the observation of a type IV recess. In DePalma type V, the middle glenohumeral ligament existed as 2 small synovial folds; and finally, type VI had no recesses. Pattern 6 would probably result in a type II or a type VI recess.

One must bear in mind, however, that many of the studies that detailed the observation of glenohumeral ligaments or of the synovial recesses were done in embalmed cadavers[7,14,16,22] or in static specimens; that is, the capsule was not observed during motion of the humerus.[3,4] Another study[8] on the appearance of the capsule using Schlemm's approach in embalmed shoulders was recently published.

Although these studies are useful, because they warn against interpretation of normal variants as pathology, they have drawbacks as well. The danger is that they may mislead the unwary into thinking that the visible folds and cords actually are the ligaments themselves. Looking at the folds of the glenohumeral joint is comparable to looking at a pitched tent with the flysheet draped over its tent poles. If the tent poles are not tensioned enough, the flysheet will hang in folds, but it will hang taut when the guy-ropes are maximally tensioned. In both instances, however, the flysheet has the same fabric structure. Likewise, the capsule is draped over poles with tension lines

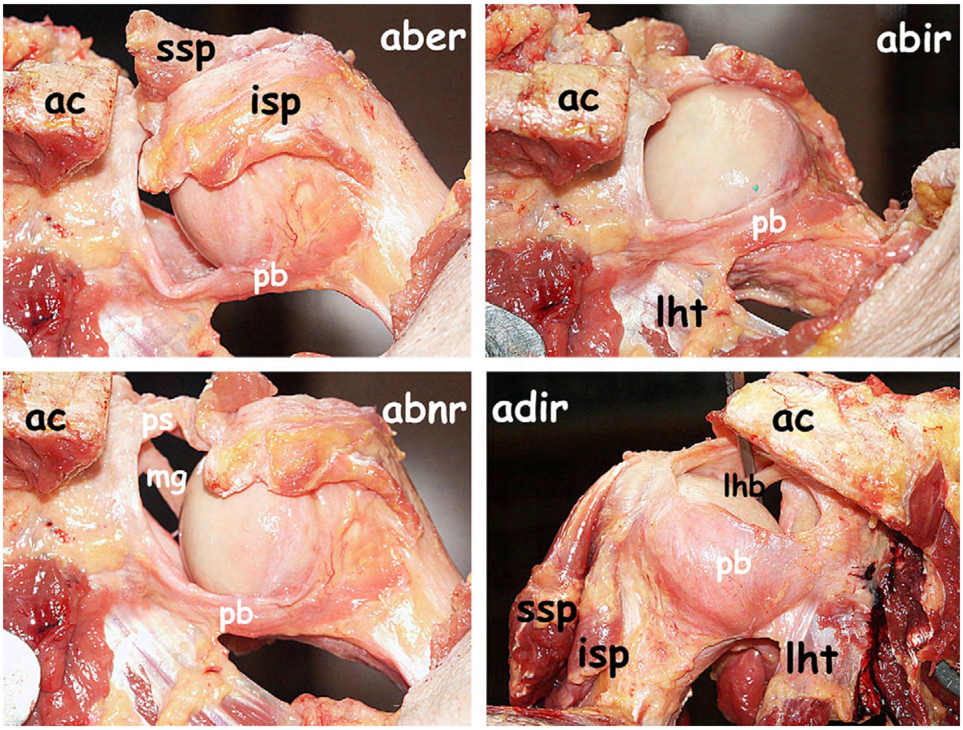


**Figure 9** Posterior view of the folding-unfolding mechanism of the posterior band of the inferior glenohumeral ligament *(pb)*. **Top left,** It is folded in abduction and external rotation *(aber)*, **(top right)** unfolded in abduction and neutral rotation *(abnr)*, **(bottom left)** taut posteroinferiorly under the humeral head in abduction and internal rotation *(abir)* and **(bottom right)** stretched out over the posterior side of the humeral head in adduction and internal rotation *(adir)*. *ps*, Posterosuperior glenohumeral ligament; *mg*, middle glenohumeral ligament; *ssp*, supraspinatus; *isp*, infraspinatus; *lhb*, tendon of the long head of the biceps; *lht*, tendon of the long head of the triceps; *ac*, acromion.

**Table II** Distribution of synovial recesses in the literature according to classification of DePalma[3]

| First author | Total specimens, No. | Type I, % (No.) | Type II, % (No.) | Type III, % (No.) | Type IV, % (No.) | Type V, % (No.) | Type VI, % (No.) |
|---|---|---|---|---|---|---|---|
| DePalma[3] | 96 | 30.2 (29) | 2.04 (2) | 40.6 (40) | 9.03 (9) | 5.1 (5) | 11.4 (11) |
| Moseley[14] | 75 | 6.7 (5) | 2.7 (2) | 89.3 (67) | 1.3 (1) | 0 | 0 |
| O'Brien[17] | 11 | 18.1 (2) | 0 | 36.3 (4) | 18.1 (2) | 9.9 (1) | 18.1 (2) |
| Gohlke[7] | 43 | 28 (12) | 9.0 (4) | 42 (18) | 4.7 (2) | 4.7 (2) | 11.6 (5) |
| Nishida[16] | 15 | 73.3 (11) | 0 | 13.3 (2) | 0 | 0 | 13.3 (2) |
| Steinbeck[22] | 104 | 38.5 (40) | 0 | 46.2 (48) | 5.8 (6) | 0 | 9.6 (10) |

(the ligaments and their borders), but the folds are not equal to the capsular ligaments (the fabric of the fly-sheet). When more or less tension is put on the guy-ropes by rotating the humerus, the folds become more or less prominent. Embalming or looking at the capsule in only 1 position results in a specific configuration of folds, which lets one suspect the position of the supporting poles but gives no idea about the tension and the position of the guy-ropes.

Mistaking folds for ligaments is, in our opinion, one of the major factors explaining the failure of many surgical procedures for instability. For example, when observing a slack ligament with a torn labral origin, it is tempting to reattach only the labral tear and disregard the possible capsular laxity, or even the tear, as one expects a certain amount of folding. This is what was done in the early arthroscopic Bankart repairs. To return to the tent analogy, this only retensioned the guy-rope by reinserting its peg. Only by being aware of the true configuration of the capsular ligaments can anatomically correct techniques be developed.

Nevertheless, the folds and recesses that are seen during arthroscopy can be used to identify the underlying ligamentous reinforcements. In internal rotation, the middle glenohumeral ligament, the anterior band of the inferior glenohumeral ligament, and the fasciculus

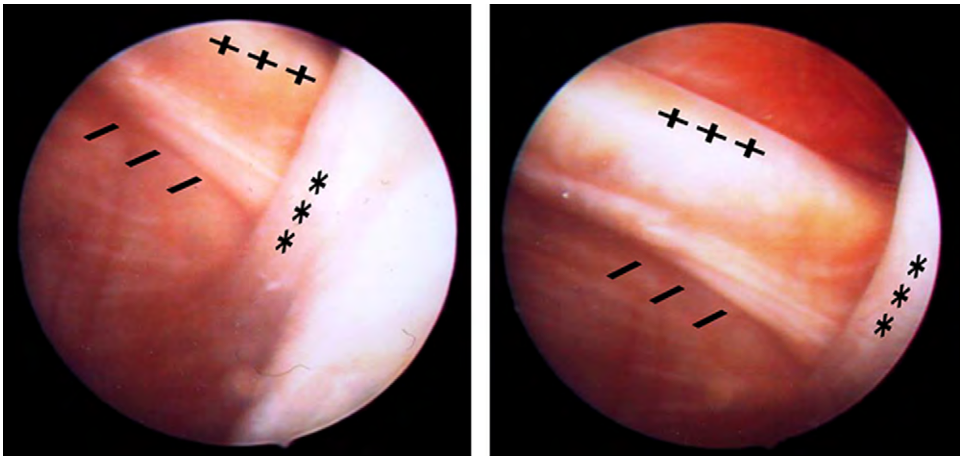

**Figure 10** Arthroscopic image shows the edge of a fasciculus obliquus (/ / /). The view is of the anterior capsule of a left shoulder from a standard posterosuperior portal. The fasciculus obliquus runs parallel with the tendon of the subscapularis (+ + +) and crosses the medially situated anterior band of the inferior glenohumeral ligament (* * *). This shoulder probably has a pattern 2, with a small anterior band that is crossed by the fasciculus obliquus far medially.

obliquus lie almost parallel and are folded up like an accordion. In external rotation, the middle glenohumeral ligament and the anterior band of the inferior glenohumeral ligament–fasciculus obliquus complex are maximally separated, and the thin capsular tissue without fibrous reinforcement in between them is stretched out. In a shoulder with normal capsular tension, the anteroinferior fold disappears in external rotation as the ligaments become taut.

It is important to draw attention to the fasciculus obliquus. This ligamentous reinforcement runs from inferiorly on the glenoid and the long head of the triceps to anterosuperiorly on the humerus, where it mingles with the tendon of the subscapularis as well as with the middle glenohumeral ligament. It can best be seen from the extraarticular side, where it is superficial to the anterior band of the inferior glenohumeral ligament that runs diagonally to it. Because both structures cross each other to a variable extent, the fasciculus obliquus may be difficult to visualize arthroscopically. The part that is not covered by the anterior band of the inferior glenohumeral ligament can be seen laterally, running almost parallel with the intraarticular portion of the subscapularis tendon just before it fuses with the tendon and the middle glenohumeral ligament (Figure 10). Only Turkel et al[26] and Gohlke et al[7] have recognized this structure. Otherwise, it seems utterly neglected, possibly because it was originally described in the German literature,[2] which may not be widely accessible. As recently as 2001, Kolts et al[10,13] believed that they had discovered a new ligament, the ligamentum glenohumeral spirale, when they actually described the fasciculus obliquus.

O'Brien et al[17] investigated the histology of the inferior glenohumeral ligament complex on the glenoid side but failed to recognize that the fasciculus obliquus constituted the middle layer, most prominent in the axillary pouch. Gohlke et al[7] histologically identified 2 distinct layers, too. Their superficial layer, with a predominant circular fiber orientation, corresponded with the fasciculus obliquus, and their articular-sided deeper layer, with a predominant radial orientation, corresponded with the anterior band of the inferior glenohumeral ligament and the middle glenohumeral ligament. In addition, they described 2 types of configuration for the origin of the middle and inferior glenohumeral ligaments. In half of their specimens, they observed that both ligaments had a separate origin, and both had a radial fiber orientation. In the other half, the origin of both ligaments partially overlapped, and the middle glenohumeral ligament was radially oriented, whereas the anterior band of the inferior glenohumeral ligament had more diagonally oriented fibers.

In neutral rotation, our patterns 2 and 3 displayed a more diagonal orientation for the fibers of the anterior band of the inferior glenohumeral ligament. Patterns 4 to 6 resulted in a more horizontal fiber orientation. In the Gohlke et al study,[7] the capsule was harvested and laid out flat for microscopic evaluation, and this resulted in a static representation of the fiber orientation. In the present study, the fiber orientation was viewed dynamically. Both studies are complementary. The fiber orientation in neutral rotation and slight abduction probably compares best with the flattened specimens in the Gohlke et al study.

The fasciculus obliquus completes the inferior hammock to the humeral head as it constitutes the axillary pouch on the glenoid side. In addition, the fasciculus obliquus and the anterior band of the inferior glenohumeral ligament also form a retaining sling anteroinferiorly. This construction, which can be compared to a baby-bundler, allows for reciprocal tightening of both ligaments during increasing external rotation in various degrees of abduction. The fasciculus obliquus appears to contribute most to anterior stability in adduction, together with the middle glenohumeral ligament, as well as anteroinferior stability in adduction, whereas the inferior glenohumeral ligament seems to be more important in abduction. Because both structures not only cross but also adhere to each other, they may also be able to limit the consequences of damage to one of the components. With a typical Bankart lesion, for example, a broad fasciculus obliquus may limit medial retraction of the detached anterior band, although the fasciculus obliquus probably also tends to cause inferior displacement of the anterior band of the inferior glenohumeral ligament. The fasciculus obliquus itself may be involved when capsuloligamentous lesions extend far inferiorly on the glenoid side, when humeral avulsion of the glenohumeral ligaments occurs anteriorly, or due to capsular stretching, especially with repetitive subluxation and dislocation.

The results from the present study allow us to understand better how capsular shift procedures work. In Neer's anterior capsular shift,[15] the entire humeral insertion from inferior to anterosuperior is detached, and the entire anterior band–fasciculus obliquus complex is shifted superolaterally. Sugalski et al[23] recently demonstrated that the humeral insertion of the inferior glenohumeral ligament may occasionally be split into 2 leaves. We believe that their superior internal fold corresponds with the insertion of the anterior band of the inferior glenohumeral ligament and that their inferior external fold corresponds with the inferior insertion of the fasciculus obliquus. They suggested that the release of the humeral insertion for a laterally based capsular shift should be extended inferiorly and posteriorly to include both leaves. The observations of Sugalski et al and our observations from the present and another study[18] also explain the variation of V-shaped and collar-like humeral insertion observed by some authors.[17,24] Multidirectional instability always involves laxity of the inferior capsule and, thus, the fasciculus obliquus, too.

T-shift capsulorrhaphies, either on the humeral side [20,25,27] or on the glenoid side,[1,9,11,12] involve detachment of either the humeral or the glenoid insertion of the anterior band–fasciculus obliquus complex and also tend to separate the anterior band of the inferior glenohumeral ligament from the fasciculus obliquus. Because the fasciculus obliquus is tightened, inferior instability and anterior instability, in adduction, are addressed; whereas, tightening of the anterior band of the inferior glenohumeral ligament addresses anterior instability in abduction. Both flaps are also imbricated over each other, which serves to tighten the crossing of both components of the inferior glenohumeral ligament complex as well. Arthroscopic capsular shift techniques that do not incorporate the fasciculus obliquus may result in failure, especially when dealing with multidirectional instability. From an anatomic point of view, it is probable that the hyperabduction test,[6] which accurately tests for inferior instability, reflects the state of laxity of the fasciculus obliquus.

## CONCLUSION

The present study brings back from oblivion the fasciculus obliquus as an integral and important part of the inferior glenohumeral ligament complex. The fasciculus obliquus and the anterior and posterior bands of the inferior glenohumeral ligament form a complex network of fibrous reinforcement around the lower half of the glenohumeral joint. Being aware of all components of this complex and its folding-unfolding mechanism is important for a better understanding of the pathology involved in instability, whether anterior, inferior, or combined. In patients with multidirectional and anteroinferior instability associated with capsular laxity, it is necessary to shift both the inferior glenohumeral ligament and the fasciculus obliquus to obtain adequate capsular tension.

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# The arthroscopic view of the glenohumeral ligaments compared with anatomy: Fold or fact?

Nicole Pouliart, MD,[a,b] and Olivier J. Gagey, PhD,[c,d] *Brussels, Belgium, and Paris, France*

*In a morphologic cadaveric study with observational arthroscopy in living subjects, we tried to resolve the contradiction in the literature with regard to the nature of the glenohumeral ligaments and the difference in observation of the folds during arthroscopic and open surgery. Observation of morphology and functional anatomy of the glenohumeral capsule was performed in 200 non-embalmed cadavers through open dissection (100 specimens) and by arthroscopy (50 specimens) or both (50 specimens), as well as in 100 living subjects undergoing shoulder arthroscopy. In the resting arm position, folds and bands can be observed on the inside of the anteroinferior capsule. When the arm is moved into full abduction and external rotation, however, all bands progressively disappear from sight. The bands generally observed in the shoulder capsule during arthroscopy appear at the site of histologic reinforcements of the capsule but are not the capsular ligaments themselves, as they seem to disappear in certain positions of the humerus. Arthroscopically, it is, therefore, not possible to discern the exact limits of these ligaments. This may give rise to a certain amount of confusion when comparing clinical with anatomic and physiologic studies. On the other hand, their presence or absence in arthroscopic surgery might be of clinical relevance in evaluating capsular tension. (J Shoulder Elbow Surg 2005;14:324-328.)*

In their original descriptions of the "large ligament of the shoulder," Flood[4] and Schlemm[10] described a unique ligament involving what is called today the inferior glenohumeral ligament (IGHL) and the middle glenohumeral ligament (MGHL). Since then, the literature concerning the glenohumeral ligaments has resulted in a certain amount of confusion.

According to Turkel et al,[12] the IGHL consists of 3 distinct and readily identifiable parts: an anterior band, an intervening axillary pouch, and a less well-defined and less constant posterior band. The histologic study of the IGHL by O'Brien et al[7] clearly stated that the anterior and posterior bands of the IGHL could only be seen after positioning of the joint into various degrees of abduction and internal or external rotation. According to them, the so-called posterior band of the IGHL fans out in 90° of abduction and internal rotation whereas the anterior band fans out in 90° of abduction with external rotation. The histologic part of their study did show the existence of the 2 bands, anterior and posterior, consisting of a clear and well-defined reinforcement of the deep layer formed by thickened bands of well-organized collagen bundles. Despite the histologic evidence of O'Brien et al and older descriptions of the capsule,[1,4,6,10] the description of Turkel et al has been adopted by most authors of experimental as well as clinical studies.

However, in open surgery of the shoulder joint, we have always found a smooth capsule without any evidence of anatomic bands. Observations made during anatomic research led to the same conclusion. During arthroscopy, visible folds in the neutral position seem to disappear when the shoulder is moved. To resolve this apparent paradox, a study was undertaken that combined observation by dissection and by arthroscopy in cadavers, as well as arthroscopy in patients.

## MATERIALS AND METHODS

Five groups were studied. Group A consisted of 200 non-embalmed cadaveric shoulders (aged 51 to 103 years). Specimens with signs of shoulder surgery, limited range of motion, and sequelae of shoulder fracture or rotator cuff rupture were excluded from the study.

Fifty shoulders (group A.1) were dissected through a superior approach. The deltoid and coracobrachialis muscles were removed. Then the subscapularis muscle was carefully separated from the underlying capsule, cut, and resected just lateral to the area of dissection between the muscle and the ligament. Thus, 1 to 2 cm of the distal tendon was left attached to the humerus and to the under-

From the Department of Orthopaedics and Traumatology,[a] Academic Hospital, and Department of Human Anatomy,[b] Vrije Universiteit Brussel, Brussels, Service d'orthopédie,[c] Hôpital Bicêtre, Université Le Kremlin-Bicêtre, and Institut d'Anatomie,[d] Paris.

Reprint requests: Nicole Pouliart, MD, Academic Hospital Vrije Universiteit Brussel, Laarbeeklaan 101, 1090 Brussels, Belgium (E-mail: *izvptn@az.vub.ac.be*).



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lying part of the ligament. Great care was taken to leave the capsule intact. The supraspinatus was cut and removed just proximal to its insertion. The infraspinatus and teres minor muscles could be resected without difficulty and without damage to the posterior capsule despite its thinness. At the end of the dissection, the MGHL and IGHL were completely exposed. Inspection of the joint cavity was done, during functional tests, either through a posterosuperior horizontal incision of the capsule or through the foramen of Weitbrecht after opening of the synovial tissue, closing the rotator interval.

Fifty shoulders (group A.2) were dissected through an inferior approach. The axillary neurovascular bundle was resected, but the surrounding structures were otherwise left intact. The tendons of the latissimus dorsi and teres major were detached and reflected as necessary to expose the humeral insertion of the inferior capsule.

After inspection of the anatomic structures in situ in groups A.1 and A.2, the entire capsule was detached either from the glenoid rim (in the 50 specimens of group A.1) or detached from the humerus (in the 50 specimens of group A.2). For those specimens in which the glenoid attachment was left intact (glenoid-block group), the glenoid and the attached capsule were harvested in 1 piece. For those specimens in which the humeral insertion was preserved (humeral-block group), the humerus was fully dislocated by cutting the restricting soft tissues. Finally, in all 100 specimens, the remaining osseous block was removed as well to expose the entire capsule on a flat surface. As the primary purpose of this study was to compare the view of the intraarticular capsule obtained by arthroscopy and that obtained through dissection, we opted not to remove the synovial lining of the capsule.

In fifty shoulders (group A.3), the anteroinferior capsuloligamentous complex was examined arthroscopically through a standard posterior portal. Fifty shoulders (group A.4) first underwent arthroscopic examination and labeling of the folds with suture wires. Thereafter, these shoulders were dissected to relate the arthroscopically visualized folds with those seen at dissection. In total, 100 shoulders were studied arthroscopically.

In all shoulders of group A, the functional anatomy was studied by observing the anteroinferior capsule during humeral motion from 0° to 90° of glenohumeral abduction and from 90° internal to 90° external rotation at intervals of 30° of glenohumeral abduction. In the arthroscopic subgroup, the maneuver was carried out twice, once with the scope positioned superior to the humeral head and then with the scope positioned inferiorly.

Group B consisted of 100 patients who underwent shoulder arthroscopy for various pathologies but not instability (mostly cuff rupture and impingement syndromes). Functional anatomy was studied as in group A.

## RESULTS

When the capsule was examined from the outside, after removal of the surrounding muscles, the capsule looked like a smooth, homogeneous cuff practically without discernible reinforcements or characteristic folds, except when there was a very well-formed foramen of Rouvière.[8,9] This was seen only in 3 cases. With the arm in the resting position alongside the body, in all shoulders, the anterior side of the capsule showed no particular characteristics except for a plication at the foramina of Weitbrecht and Rouvière.

With the arm in the resting position, observation of the anteroinferior capsule from within the joint through a horizontal posterior capsular incision and arthroscopically demonstrated the presence of the classically described structures[6]: the foramen of Weitbrecht, delineated by the superior glenohumeral ligament superiorly and by the MGHL inferiorly. The upper limit of the MGHL was always clearly seen as well as the band defined by arthroscopists as the anterior band of the IGHL. In 3 cases, the subcoracoid foramen of Rouvière was seen, with the inferior border of the MGHL as its upper limit and the superior border of the IGHL as its lower limit.[2] With increasing external rotation and abduction, the borders between the MGHL and the IGHL progressively disappeared until a single homogeneous structure without visible demarcations was noted (Figure 1). Only a slight indentation at the level of the foramen of Rouvière, if present, remained visible. This observation was made in all specimens. When the capsule was detached inferiorly upward through the axillary approach, it was equally impossible to identify the ligaments unequivocally.

In the glenoid-block group, folds and bands delineating the foramina of Weitbrecht and Rouvière were visible to some extent, depending on the size and depth of these foramina. In the humeral-block group and when the capsule was detached inferiorly, no bands could be seen without manipulation. When pulling on the capsule after unilateral detachment of the capsuloligamentous complex, ridges appeared systematically at the same site. The localization of these ridges corresponded with the localization of the folds seen when the capsule was still in situ. After harvesting of the entire capsuloligamentous complex, this was exposed on a flat surface. In this position, the shape of the capsuloligamentous complex was either a rectangle (in about 70%) or the flattened-out representation of the surface of a cone (in about 30%). In this situation, neither a fold nor thickening of the capsuloligamentous complex was seen. It was impossible to dissociate the capsule from the fold or to individualize the ligaments, unless clearly demarcated foramina of Rouvière and Weitbrecht were present. Therefore, in this series, no correlation between folds and ligamentous reinforcements could be made by comparing observation with a fine-detail dissection under loupe magnification and histologic investigation.

Posteriorly, there is only a progressive thinning of the fibrous structure, without a real border. In the living subjects, the same observations were made

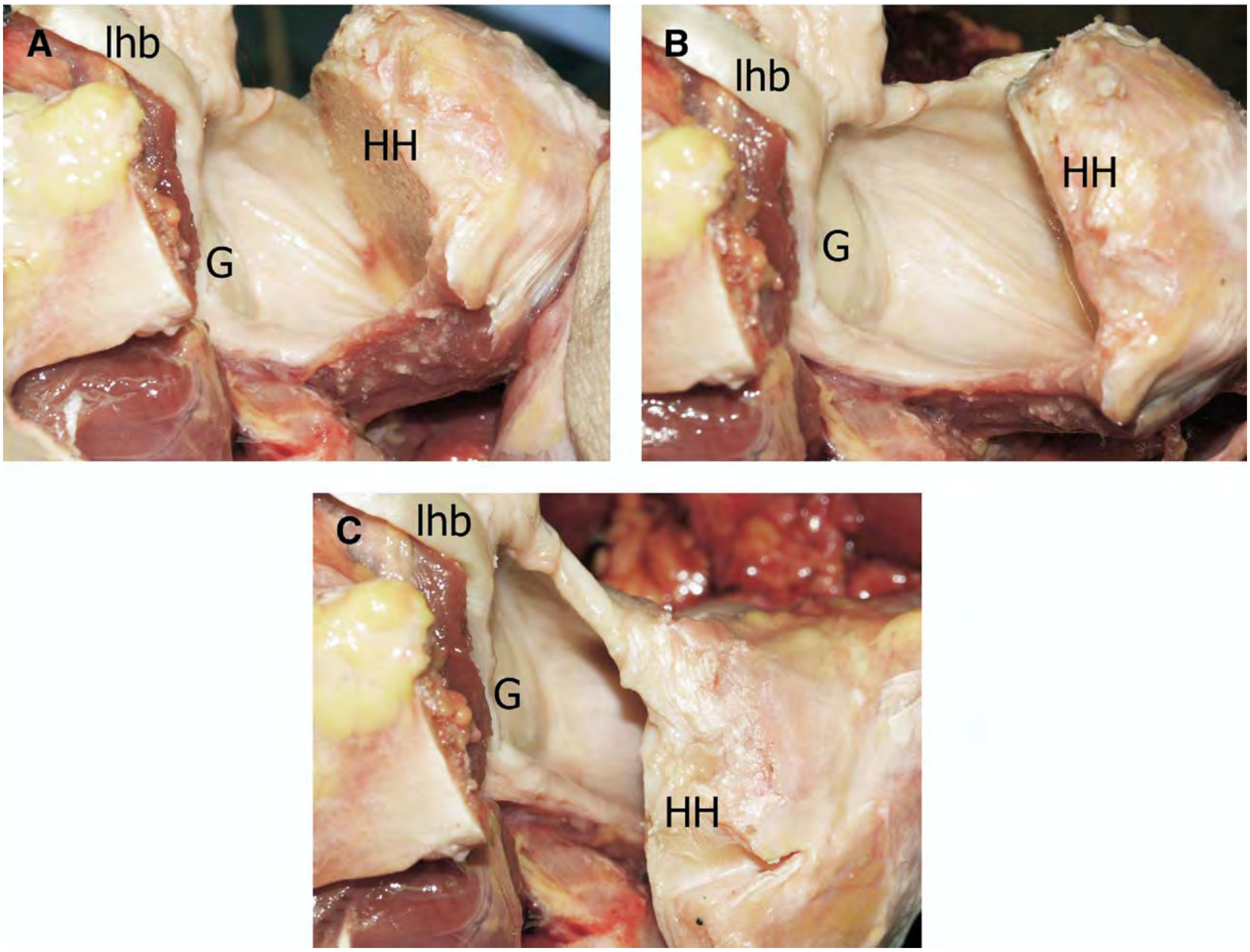


**Figure 1** Disappearing folds: posterior open dissection view from internal (**A**) over neutral (**B**) to external (**C**) rotation with the arm in abduction. In this specimen, the humeral head (*HH*) and posterior capsule were resected so that the anteroinferior capsule could be better shown for this illustration. *G*, Glenoid fossa; *lhb*, tendon of long head of biceps.

during arthroscopy. With the arm in neutral rotation and in lower degrees of abduction, bands corresponding with the IGHL and MGHL could almost always be seen. In 2 cases, there was a well-pronounced foramen of Rouvière. However, when the arm was moved over 90° of abduction and into full external rotation, all anterior bands progressively disappeared from sight. In full internal rotation, the anterior fold becomes more prominent (Figure 2).

## DISCUSSION

Our observations in 300 living and cadaveric shoulders do not leave much room for discussion. The folds that one can see on the anterior side of the glenohumeral capsule, in some positions of the arm, are not the ligaments themselves. The folds progressively disappear from sight when the arm is moved into a different position. In full glenohumeral abduction, especially with external rotation, the capsule always looks smooth and homogeneous, as no folds remain.

Therefore, one must agree with the description of O'Brien et al.[7] The glenohumeral ligaments are no more than a histologically demonstrable reinforcement of the capsule. As capsular ligaments, they form an integral part of the glenohumeral capsule and cannot be separated from the capsule. It is likely that the folds that systematically appear at the same place are the visible reference to these capsular ligaments. The folds may represent either the edges of the ligamentous reinforcements or the areas of maximal tension in the capsule.

The confusion that has arisen, starting from the work of Turkel et al,[12] is a result of positional bias. In part, these authors actually described the ligaments after detaching the capsule from the humerus. There-

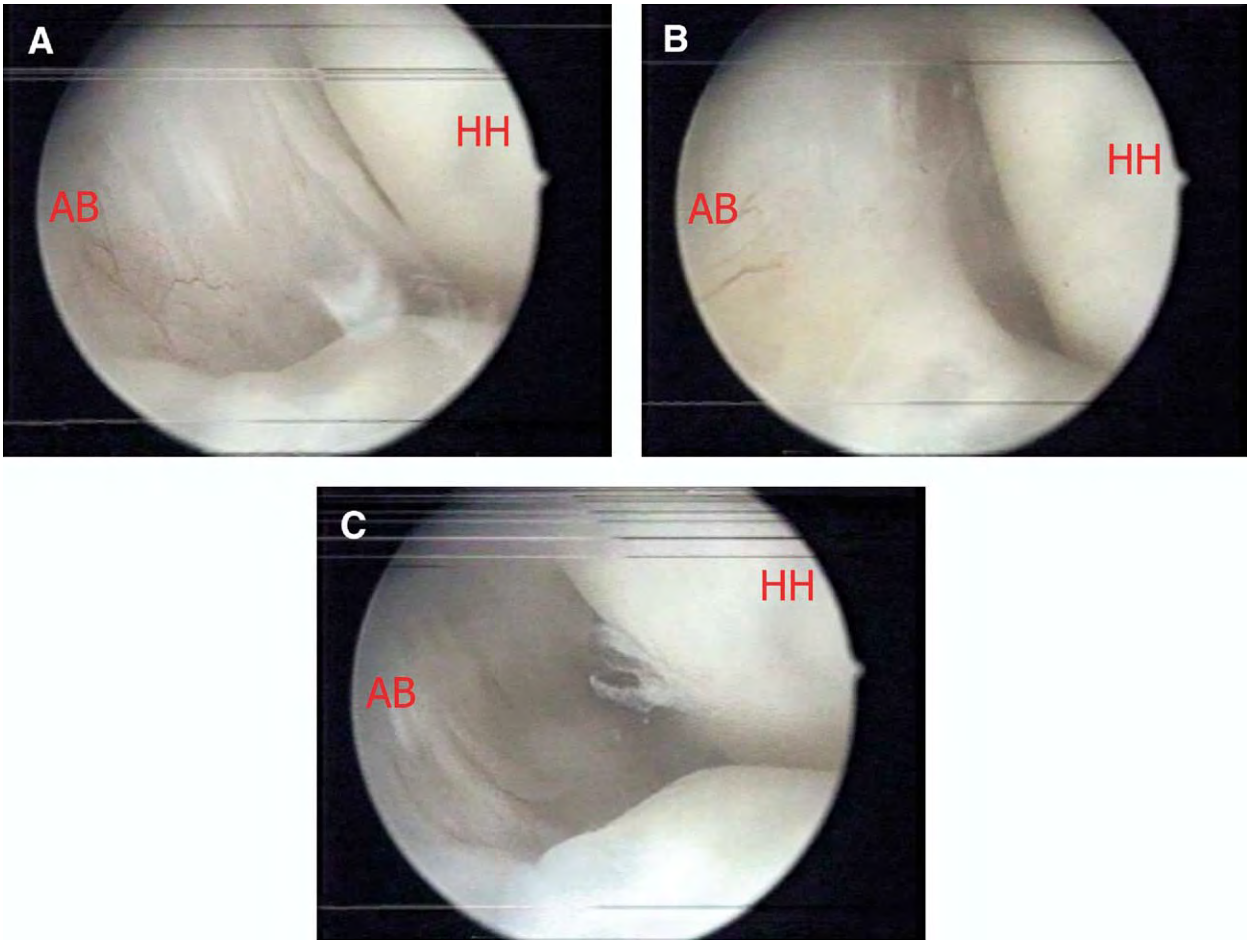


**Figure 2** Disappearing folds: arthroscopic view from internal (**A**) over neutral (**B**) to external (**C**) rotation with the arm in 45° of abduction. *HH*, Humeral head; *AB*, region of anterior band of IGHL.

fore, no positional evaluation was possible. In our glenoid-block specimens, this did allow visualization of folds and bands around the foramina. In their other unembalmed specimens, Turkel et al studied the capsule through a posterior incision. They marked the ligaments and then observed them radiographically while moving the arm. They did not look at the ligaments directly while moving the arm. That the capsule is observed with the arm in a neutral position but not in other positions seems to be a general limitation of experimental studies on dissection. If the arm was moved, this was seldom beyond 90° of total abduction or elevation. In embalmed specimens, this might actually be difficult as a result of stiffness.[11,12] During arthroscopy, the shoulder is usually held in 30° of abduction and is rarely brought over 90° of total abduction. In these lower degrees of abduction or elevation, some folds may remain visible, especially when there is a more pronounced foramen of Rouvière. Therefore, experimental as well as clinical studies using arthroscopy tend to describe the ligaments in detail, as they rarely move the arm through its range of motion. In addition, we believe that the classification of DePalma et al[2] of the anterior capsule should be viewed as what it really is: a description of the possible synovial recesses. It classifies the anatomic variation of the foramina of Weitbrecht and Rouvière. The ligamentous folds possibly are a consequence of these foramina that represent breaches in the underlying fibrous structure of the capsule, and therefore, the folds may represent the edges of the histologically defined capsular ligaments. This corresponds with the description of Ferrari[3] of the anterior capsule from the outside, where he correlated the visibility of the ligaments with the presence of the openings to the superior and inferior subscapularis bursae (which are the foramina of Weitbrecht and Rouvière).

The folds can probably also be linked to the histologic structure of the capsule as described by Gohlke et al.[5] They studied the capsule under polarized light microscopy and revealed a layered structure. In the more superficial layer, the collagen fibers had a more dominant circular orientation, whereas the deeper, articular-sided layer demonstrated a predominantly radial orientation. In the anteroinferior part of the capsule, the fibrous structure was even more complex, with 2 main patterns. The circular fibers corresponded with the fasciculus obliquus described by Delorme.[1] In about half of their specimens, the IGHL and MGHL were separate, and then both had predominantly radially oriented fibers. In the other half, the MGHL and IGHL partially overlapped, and then the fibers of the IGHL had a more diagonal course. The entire capsule formed a cylinder with cross-linked radial and circular fibers. Gohlke et al likened the functional effect of this capsular construction to the mechanism of a Chinese finger trap. When compressed, the mesh wires of this construct are doubled up in the longitudinal direction, whereas in distraction, they are stretched out and close together in the direction of the distraction. Transferring this model to the anterior shoulder capsule allows us to comprehend the mechanism underlying the appearance and disappearance of the capsular folds during motion. The capsular folds represent the tension bands of the capsular ligaments, not the entire ligament. They are the mesh wires of the Chinese finger trap.

In an ongoing study of living subjects with shoulder instability, we use the same functional method of observation of the capsule as in the present study. Preliminary results show that the anterior and posterior folds do not seem to appear during rotation of the humerus. In unstable shoulders, one expects the radially oriented fibers of the IGHL and possibly the MGHL to be disrupted or at least stretched. Therefore, the capsular cylinder no longer works as a functional unit. The Chinese finger trap becomes bigger and requires more compression or distraction for the same effect. For the shoulder, this implies more abduction and especially more rotation, but the surrounding soft tissues restrict this increased motion.

Likewise, in the examination of patients with frozen shoulders, no folds whatsoever could be observed. In this specific situation, the Chinese finger trap is probably too small and in a permanently packed state.

On the basis of these considerations, we believe that visualization of the folds has 2 possible clinical implications, as the failure of folds to appear with humeral rotation signifies capsular redundancy. In the absence of capsulolabral tears, this phenomenon implies midsubstance stretching of the capsular ligaments (or humeral avulsion of the glenohumeral ligament) for patients with traumatic instability, whereas for patients with atraumatic instability, it denotes capsular laxity. In the latter group, arthroscopic capsular shrinkage is becoming a popular procedure. One of the difficulties of this type of surgery is knowing when the capsule has been sufficiently tightened. For patients with traumatic instability, obvious capsulolabral tears are repaired to the glenoid rim. In recent years, more and more emphasis has been placed on reattaching the capsular ligaments to the correct height on the rim, thereby restoring adequate capsular length. For both situations, be it mechanical repairs or shrinkage, the reappearance of folds on (gentle) humeral rotation might be an indication that adequate capsular tension has been reached. To test this hypothesis, we have initiated a further study of the glenohumeral folds in patients with instability.

We conclude that the concept of distinctly visible ligaments during arthroscopy of the glenohumeral joint should be abandoned. The folds that are generally described are no more than just that. They systematically appear at the site of the histologic reinforcements of the capsule, and these are more or less prominent visually. Their observability depends on more or less pronounced foramina to the inferior and superior subscapularis bursa and on the position of the humerus. These folds are, however, not the capsular ligaments themselves. In arthroscopic surgery, they might serve as a landmark for the underlying ligaments and might act as a guideline for adequate capsular tension. On the other hand, in open surgery, they can seldom be discerned.

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# Reconciling Arthroscopic and Anatomic Morphology of the Humeral Insertion of the Inferior Glenohumeral Ligament

Nicole Pouliart, M.D., and Olivier Gagey, Ph.D.

**Purpose:** To clarify the morphology of the humeral insertion of the inferior glenohumeral ligament (IGHL). **Type of Study:** Cadaveric and arthroscopic anatomic analysis. **Methods:** The morphology of the humeral insertion was studied in 200 nonembalmed cadavers through open dissection (100 specimens), by arthroscopy (50 specimens), or both (50 specimens). In addition, the morphology was studied in 100 living subjects with stable shoulders undergoing shoulder arthroscopy. **Results:** On the humeral side, the insertion of the inferior capsular fibers is usually in the form of a V, the point of which is covered by the tendon of the latissimus dorsi. When viewed intra-articularly, the inferior insertion usually gives a collar-like impression because the capsular recess is filled with frenula capsulae. **Conclusions:** Our description corresponds with that found in the classic literature. Our results are, however, in contrast with those of others who have observed about 50% of V-shaped insertions. This difference may be explained by the method of observation and by the small numbers of specimens studied. The form of the humeral insertion of the IGHL is linked to the formation of a supporting hammock that can accommodate the humeral head during movement as described by several authors. **Clinical Relevance:** We believe that the difference between arthroscopic and anatomic observation of the humeral insertion may have 2 major clinical implications. An observed tear of the frenula capsulae may not necessarily represent a humeral avulsion of the glenohumeral ligaments (HAGL). In the case of a HAGL, the capsule may have to be reattached in its V-form to adequately retension the inferior capsule. **Key Words:** Inferior glenohumeral ligament—Anatomy—Cadaver—HAGL.

Many studies have been published on the biomechanical properties and function of the inferior glenohumeral ligament (IGHL). Those by Bigliani et al.,[1] Ticker et al.,[2] and Turkel et al.[3] may be the best known. There are fewer articles on the anatomy of the glenohumeral ligaments. Most researchers have studied the glenoid labrum[4-10] or the morphology of the glenohumeral ligaments.[11-15] Only in the articles by O'Brien et al.,[16] Ticker et al.,[2] and Turkel et al.,[3] and in some textbooks,[17-21] have we found a description or drawing of the humeral insertion of the IGHL. Duparc et al.[22] studied the blood supply of the glenohumeral capsule but not its anatomy.

On the glenoid side, the IGHL is said to invariably attach on the inferior third of the labrum. However, for the humeral insertion of the IGHL, 2 variations are described (Fig 1): a collar-like attachment close to the articular cartilage (in half of the specimens studied by O'Brien et al.[16] as well as by Ticker et al.[2]) and a V-shaped attachment that has its base close to the cartilage rim and its point more inferiorly on the humeral metaphysis (in the other specimens of O'Brien et al.[16] and Ticker et al.,[2] and in all other referenced studies).[3,17-21]

Our dissection of glenohumeral capsules showed a consistent V-shaped form, whereas arthroscopic observation almost uniformly showed a collar-like shape. To resolve this apparent paradox, a study that combined observation by dissection and by arthros-

*From the Departments of Orthopaedics and Traumatology, and Human Anatomy, Academic Hospital Vrije Universiteit Brussel (N.P.), Brussels, Belgium; and Service d'orthopédie, Hôpital Bicêtre, Université Paris-Sud, and the Institut d'Anatomie (O.G.), Paris, France.*

*Address correspondence and reprint requests to Nicole Pouliart, M.D., Academic Hospital Vrije Universiteit Brussel, Laarbeeklaan 101, 1090 Brussels, Belgium. E-mail: izvptn@az.vub.ac.be*



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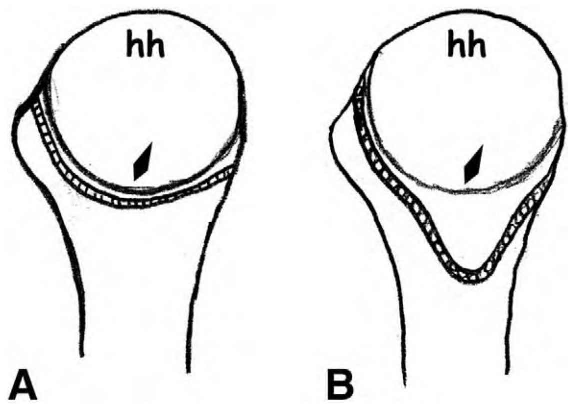


FIGURE 1. Line drawing of humeral insertion: (A) Collar-like and (B) V-shaped type. (hh, humeral head; arrow indicates margin of articular cartilage.)

copy in cadavers, as well as arthroscopy in living subjects, was undertaken. Our hypothesis was that the humeral insertion of the capsular fibers of the IGHL is V-shaped, but that the trough that is formed by the fibrous structure is usually obscured by the synovial lining of the capsule, thereby giving the intra-articular appearance of a collar-like attachment.

## METHODS

Five groups were studied. Group A consisted of 200 nonembalmed cadaver shoulders (aged 49 to 103 years). Specimens with signs of shoulder surgery, limited range of motion, or sequelae of traumatic shoulder lesions were excluded from the study.

Fifty shoulders (group A.1) were dissected through a superior approach. First, the surrounding muscular structures were carefully separated from the underlying capsule, taking great care to leave the capsule intact. After a first inspection of the IGHL through a posterior and superior horizontal incision in the capsule, the capsule was detached from the glenoid. Then the humerus, with the attached capsule, was fully dislocated by cutting the restricting soft tissues. The humeral attachment of the capsule was then progressively detached from the bone from superior downward under low magnification. This allowed determination of the exact form of insertion of the IGHL from the inside. In 20 specimens, the humeral head was resected at the anatomic neck before detaching the capsule to allow better visualization of the entire IGHL through the posterosuperior incision.

Fifty shoulders (group A.2) were dissected through an inferior approach. The humeral insertion of the inferior capsule was exposed by detaching and reflecting the tendons of the latissimus dorsi and the teres major. Then, the insertion of the IGHL was progressively peeled off the humerus to determine its exact form from the outside.

In another 50 shoulders (group A.3) the IGHL was only examined arthroscopically through the standard posterior portal with the arthroscope passing under the humeral head.

Finally, another 50 specimens (group A.4) were first studied arthroscopically and then dissected. We used the superior approach (as for group A.1.) in 25 of these specimens and the inferior approach (as for group A.2.) in the other half of this group.

Group B consisted of 100 patients who had undergone shoulder arthroscopy for reasons other than instability. They were examined in the same way as group A.3.

## RESULTS

When dissected from the outside, through the axillary approach, the insertion of the IGHL on the humerus appeared in the form of a V in all cases. The point of the V was at a distance of 1 to 2 cm from the inferior pole of the humeral head, and the branches progressively approached the border of the articular cartilage. The most superior part of the IGHL anteriorly joined the inferior fibers of the inferior tendon of the subscapularis muscle. The superior fibers of the tendon of the latissimus dorsi muscle always covered the point of the V. The extent of this coverage varied with the exact position of the tendinous insertion. Usually, there were loose connective fibers between capsule and latissimus dorsi tendon. The most distal point of the capsule can descend as far as the uppermost fibers of the teres major tendon, but never reached as far distally as to be covered by this tendon. By dissection we found the V-form of the capsule in all cases. The distance from the upper border of the latissimus dorsi tendon to the cartilage rim along the crista tuberculi majoris varied between 1 and 3.5 cm, whereas that from the upper border of the tendon of the teres major to the cartilage rim varied between 3 and 4.5 cm. When progressively peeling off the capsule from the humerus from inferior to superior, we noticed that the area between the fibrous branches of the V was filled with easily removed loose connective tissue and a few denser fibrous bands reaching up to the cartilage rim in most cases.

When viewed from the inside, through the superior approach, the insertion of the IGHL on the humerus

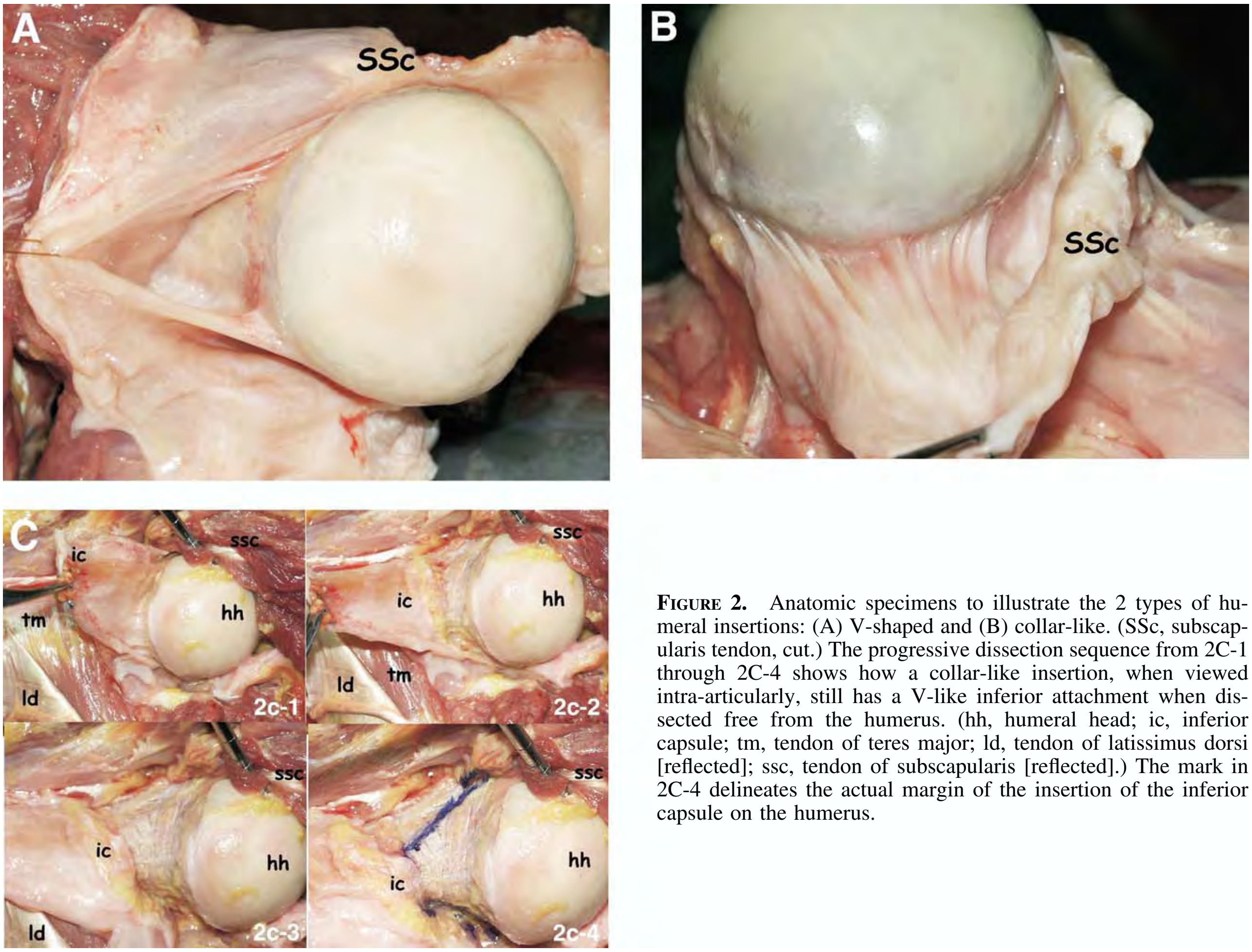


**FIGURE 2.** Anatomic specimens to illustrate the 2 types of humeral insertions: (A) V-shaped and (B) collar-like. (SSc, subscapularis tendon, cut.) The progressive dissection sequence from 2C-1 through 2C-4 shows how a collar-like insertion, when viewed intra-articularly, still has a V-like inferior attachment when dissected free from the humerus. (hh, humeral head; ic, inferior capsule; tm, tendon of teres major; ld, tendon of latissimus dorsi [reflected]; ssc, tendon of subscapularis [reflected].) The mark in 2C-4 delineates the actual margin of the insertion of the inferior capsule on the humerus.

appeared rounded off (Fig 2A) in all but 12 cases. In these 12 specimens, we observed a pronounced V-shaped indentation at the inferior pole of the humeral insertion (Fig 2B). By fine dissection of this inferior capsular area, we could easily remove the synovial lining of the capsule from the underlying fibrous structure. Thereby, we retrieved the V-shaped form of the capsular fibers of the IGHL. The area in between the branches of the V was filled mainly with loose easily resectable tissue and a few fibrous bands.

Arthroscopically (cadavers and living subjects), the inferior part of the IGHL appeared rounded off in the majority of cases with capsular folds that varied in appearance with rotation of the humerus (Fig 3A). In all cases, these “frenula capsulae”[19,20] reached up to the cartilage rim and gave the impression of a collar-like insertion. In about 30%, these folds seemed to fill the examined zone completely. When the filling was less complete, rotation of the humerus often led to the appearance of more prominent folds and edges in certain positions. Without examining the humeral insertion in varying degrees of rotation the indentation behind these edges could be mistaken for a V-shaped inferior recess. A clearly definable indentation, creating a true inferior recess that did not disappear with rotation (Fig 3B), was only observed in 5 specimens (2 of which were dissected as well) and in 3 patients. These deep and permanent indentations seen during arthroscopy corresponded with the V-shaped form when dissecting.

## DISCUSSION

In their study of the IGHL in 11 cadaver shoulders, O’Brien et al.[16] observed 2 types of humeral insertion through an anterosuperior incision in the capsule. Six of their specimens had a collar-like insertion immediately inferior to the cartilaginous margin of the hu-

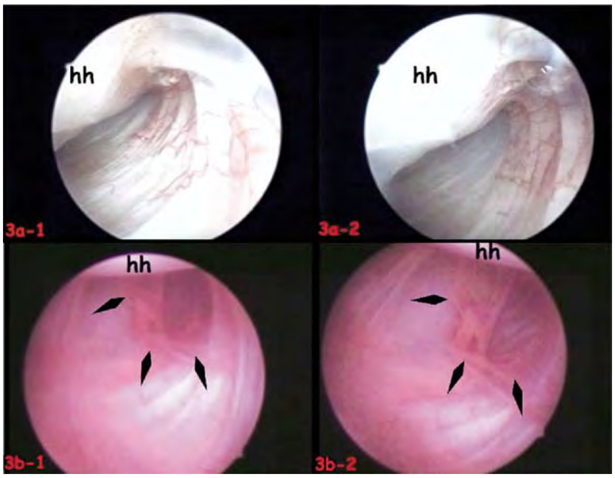


**Figure 3.** Arthroscopic view of humeral insertion: Collar-like type (A-1 and A-2) and V-shaped type (B-1 and B-2). The arrows in 3A indicate the margin of the inferior recess, which is partially filled (on the left side) with a synovial fringe. hh; humeral head.

meral head. The other 5 cases had a V-shaped insertion. Ticker et al.[2] found an even distribution of both insertion types in their 8 cadavers through a superior incision with removal of the humeral head.

The humeral insertion of the glenohumeral capsule is extensively described in 3 classic anatomic textbooks.[19-21] According to these books, the fibrous capsule is attached to the anatomic neck of the humerus, close to the articular margin, except in its inferior part. There it progressively moves away from the articular margin, so that the insertion is about 1 cm away from the cartilage rim in relation to the inferior pole of the humeral head. In addition, the synovial lining follows the capsule onto the bone and then covers the anatomic neck up to the cartilage rim. Paturet[19] and Rouvière and Delmas[20] explicitly describe some recurrent fibers of the inferior capsule that attach to the cartilage rim of the humeral head. These bulging fibrous bands lift up the synovial membrane and together they are called the "frenula capsulae." The arterial circle of the blood supply to the humeral head described by Duparc et al.[22] passes along these frenula capsulae. Blachut and Day[23] describe a synovial-lined inferior pouch, but do not specify its form of attachment onto the humerus.

The V-shaped description is illustrated by the typical drawing of the humeral insertion of the glenohumeral capsule (Fig 1B) that is most frequently found in orthopaedic text books.[3,17,18]

Our work shows that there is always a humeral insertion in the form of a V when examining the capsule from the outside, but that this V looks more or less rounded off from the inside because of connecting synovial bands. In only 8% of all shoulders that were examined from the inside, be it arthroscopically or by open dissection, did we observe a V-shape intra-articularly. The V-form of the capsule together with the connecting frenula capsulae confirms the classic description.[3,17-21] That O'Brien et al.[16] and Ticker et al.[2] report a collar-like intra-articular aspect in about half of their shoulders can probably be explained by considering that these authors only inspected the humeral insertion but did not dissect it. Therefore, they described the attachment of the synovial lining rather than that of the fibrous capsule. It is much more difficult to explain why they observed 45% to 50% of V-shaped insertions. We observed that rotation of the humerus can create the impression of a shallow V when inspecting the humeral insertion arthroscopically. This same impression may be created when looking at the inferior capsule through an anterosuperior incision. This positional bias may be an explanation for the high incidence of V-shaped humeral insertions in O'Brien's series. Although the article by Ticker et al.[2] offers little information on the method used to examine the humeral insertion—these authors only state that they used the same dissection method as Bigliani et al.[1]—the same explanation may be valid. On the other hand, the high percentage may also be attributable to the small number of studied specimens (19 for both studies combined *v* 300 in the present study). It is only by combination of axillary dissection of the inferior capsule and its intra-articular inspection that we can resolve the apparent paradox between both approaches.

A collar-like type of insertion of the capsular fibers in combination with a dependent capsule and the external V-shape would imply that the direction of the inserting capsular fibers is from inferior upward to the cartilage rim. With the arm in adduction this seems plausible. With the arm in abduction, however, the capsule would be doubled up on itself and would therefore be stretched at 180° to its humeral insertion fibers (Figs 4 and 5A).

This still does not explain why the inferior fibrous insertion should be V-shaped. We believe that a V-shaped inferior capsule is a part of the hammock-like function of the IGHL as described by O'Brien et al.[16] and Turkel et al.[3] The humeral part of the axillary pouch of the IGHL corresponds with the V-shaped extension of the inferior capsule along the humeral neck. The vertical humeral part along the tuberculum minus corresponds with the anterior band of the

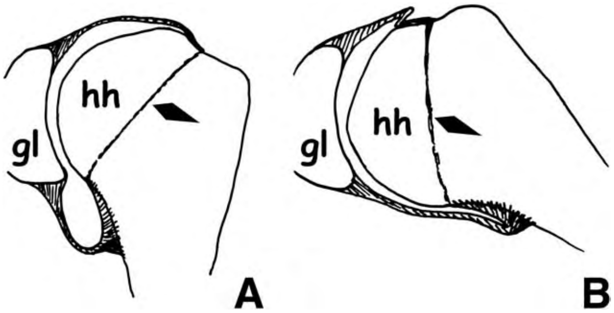


**FIGURE 4.** Line drawing illustrating a coronal section through the middle of the glenohumeral joint with the humerus in (A) adduction and (B) abduction. The stretching of the inferior capsule when going from A to B shows that the capsular fibers would be doubled up on themselves if they were to attach from the point of the capsular V back upward to the cartilage rim. (hh, humeral head; gl, glenoid.) The arrows indicate the margin of the articular cartilage.

IGHL. In adduction (Fig 5A), the axillary part of the IGHL has a diagonal course from the glenoid downward to the humerus, whereas the anterior band of the IGHL has a horizontal course from the glenoid to the humerus. In abduction (Fig 5B), the axillary part has a more horizontal course, whereas the anterior band runs diagonally upward. This allows reciprocal tightening of both structures with different positions of the glenohumeral joint. In that regard, these macroscopic observations may link the microscopic observations made by O'Brien et al.[16] and those by Gohlke et al.[11] O'Brien's axillary pouch then would correspond with Gohlke's IGHL, whereas O'Brien's anterior band would correspond with the anteroinferior part of Gohlke's fasciculus obliquus. We have planned further anatomic and histologic work to study this hypothesis.

Knowledge of the dual aspect of the humeral insertion may be important to clinicians who treat shoulder instability. Tears of the frenula capsulae seen during arthroscopy may not necessarily signify a humeral avulsion of the (inferior) glenohumeral ligaments (HAGL). When treating shoulder instability, theoretically it would seem best to restore adequate length and tension to the capsular structures and, more specifically, the various components of the IGHL. Therefore, we believe that the capsule would best be reattached in its original V-form on the humerus when surgically repairing HAGL lesions.

## CONCLUSIONS

The humeral insertion of the capsule usually has a dual aspect: V-shaped on the outside, and collar-like on the inside due to filling by synovial tissue and fibrous bands collectively described as "frenula capsulae." This dual aspect may have implications for diagnosis and treatment of lesions that involve the inferior humeral insertion. On the one hand, an observed tear of the frenula capsulae may not necessarily

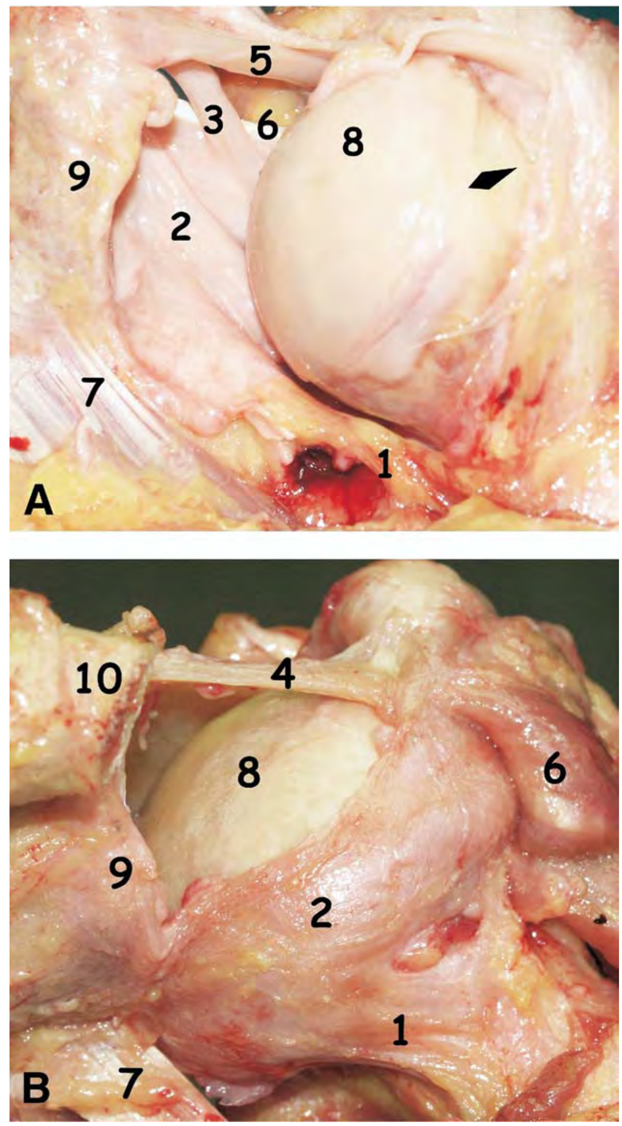


**FIGURE 5.** (A) Posterior view of the IGHL with the posterior capsule removed and with the humerus in adduction and neutral rotation, showing the stretched axillary part in this position and anatomically illustrating the drawing of Fig 4B. (B) Anterior view of the IGHL with the humerus in abduction and neutral rotation. (1, axillary part of IGHL; 2, anterior band of glenohumeral ligament; 3, middle glenohumeral ligament; 4, superior glenohumeral ligament; 5, long tendon of biceps; 6, subscapularis; 7, long tendon of triceps; 8, humeral head; 9, glenoid; 10, coracoid process [cut].) The arrow indicates the margin of the articular cartilage.

represent a HAGL. On the other hand, in the case of a true HAGL, the capsule may have to be reattached in its V-form to adequately retension the inferior capsule.

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ORIGINAL COMMUNICATION

# Significance of the Latissimus Dorsi for Shoulder Instability. I. Variations in Its Anatomy Around the Humerus and Scapula

N. POULIART[1,2,3]* AND O. GAGEY[3,4]

[1]*Department of Orthopaedics and Traumatology, Academic Hospital Vrije Universiteit Brussel, Brussels, Belgium*
[2]*Department of Human Anatomy, Vrije Universiteit Brussel, Brussels, Belgium*
[3]*Institut d'Anatomie, Université Réné Descartes Paris V, Paris, France*
[4]*Service d'Orthopédie, Hôpital Bicêtre, Université Paris-Sud, Kremlin-Bicêtre, France*

In a cadaveric instability model that leaves all muscles intact initially, we studied anteroinferior glenohumeral dislocation behavior after section of the ligaments on the humeral side of the joint. In this study, the latissimus dorsi seemed to play a role when complete section did not result in a locked anteroinferior dislocation. We therefore initiated a study to test the hypothesis that the latissimus dorsi may, in certain circumstances, depending on variations in its anatomy, influence dislocation behavior. Here, in Part I, we present the results of the anatomic study of latissimus dorsi and its tendons. The anatomy of the latissimus dorsi pertaining to the scapula and humerus was studied in 100 cadaver specimens. The distance between the uppermost part of the tendon of both the latissimus dorsi and the teres major and the edge of the articular cartilage of the humeral head (tendon-cartilage distance, TCD) as well as the width and length of the tendons were measured. Furthermore, the relationship between latissimus dorsi and the inferior angle of the scapula was studied. The tendon of the latissimus dorsi inserted at a variable distance from the cartilage of the humeral head: the TCD ranged from 12.6 to 31.6 mm (mean 21.06 mm ± 5.11 mm). The latissimus dorsi can have muscular fibers arising from the inferior angle of the scapula (type 1 scapular connection, 43%). Alternatively, there may be only a few fibrous strands between the muscle and the scapula or there may be an intervening bursa (type 2 scapular connection, 57%). This variability in the morphology of the latissimus dorsi may be a factor explaining the differences observed in a study of humerus-based sequential cutting of the glenohumeral capsule. This possibility is explored in Part II of the study. The latissimus dorsi may also complete the tendinous protection of the humeral side of the capsule generally provided by the rotator cuff. Clin. Anat. 18:493–499, 2005. 

**Key words: anatomy; latissimus dorsi; teres major; anatomical variants; shoulder; joint stability; dislocation**

## INTRODUCTION

Several muscles around the shoulder joint are known to be important for maintaining shoulder stability (Glousman et al., 1988; Brostrom et al., 1989; Blasier et al., 1992; Pagnani and Warren, 1994; Kronberg and Brostrom, 1995; Bigliani et al., 1996; Itoi et al., 1996; McMahon et al., 1996; Soslowsky et al., 1997; Wuelker et al., 1998). The latissimus dorsi, however, has only been attributed a role in providing a stable scapular platform against which the humerus moves. The present study investigates the anatomy of the latissimus dorsi in relation to the glenohumeral joint, a topic on which relatively little has been published (Testut, 1884; Debierre, 1890;

*Correspondence to: Dr. Nicole Pouliart, Department of Orthopaedics and Traumatology, Academic Hospital Vrije Universiteit Brussel, Laarbeeklaan 101, 1090 Brussels, Belgium.
E-mail: nicole.pouliart@az.vub.ac.be

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Testut and Latarjet, 1948; Beck and Hoffer, 1989; Williams et al., 1999).

To study the consequences of ligament section on glenohumeral instability, we developed a cadaver model of the shoulder in which all the surrounding muscles were initially maintained intact. In this model, we studied anteroinferior dislocation behavior of the humeral head after section of the glenohumeral capsuloligamentous structures on the humeral side of the joint. In the first 30 specimens of this study, a load-and-shift test aimed at dislocating the shoulder did not result in a locked dislocation in 31% of specimens, even after section of all glenohumeral ligaments. We had the impression that the tendon of the latissimus dorsi played a role in this altered dislocation behavior. To verify this impression and to find an explanation for the difference in dislocation behavior, the present study was carried out. This first part of the study deals with the anatomic study of the latissimus dorsi and its tendon. Our hypothesis was that the latissimus dorsi might play a role for two reasons. First, the tendon may be inserted so close to the humeral head that it buttresses the head like a hammock. Second, the course of the tendon and the tension generated in it in certain positions of the glenohumeral joint may be influenced by a possible attachment of the muscle to the inferior angle of the scapula as described in *Gray's Anatomy* (Williams and Warwick, 1980), several other anatomy textbooks (Henle, 1855; Beaunis and Bouchard, 1868; Testut, 1884; Debierre, 1890; Testut and Latarjet, 1948; Bergman et al., 1988), and in Rockwood and Matsen's classic shoulder orthopedic textbook (Jobe, 1998). The initial dynamic study of capsuloligamentous lesions on the humeral side was expanded. We included capsuloligamentous sections on the glenoid side for comparison with additional specimens with humeral-sided cuts. After the capsule was completely cut, the role of latissimus dorsi and subscapularis was studied by cutting either tendon and repeating the testing procedure. The results of this part of the study are detailed in Part II.

## MATERIALS AND METHODS

We used 80 fresh shoulder specimens from cadavers donated to the Institut d'Anatomie of the Université Réné Descartes and 20 embalmed specimens from the Department of Anatomy of the Vrije Universiteit Brussel. The length and width of the tendon of the latissimus dorsi and the tendon of the teres major were measured, as detailed in Figure 1.

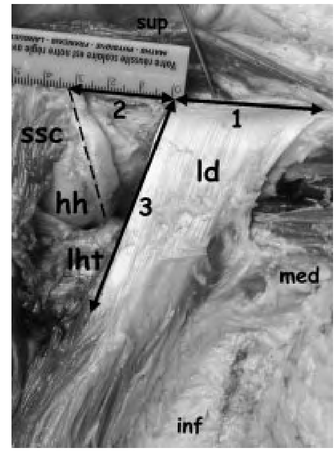


**Fig. 1.** Measurements of the tendon of the latissimus dorsi. 1, width of tendon; 2, tendon-cartilage distance (TCD); 3, length of tendon at upper border; dashed line, margin of humeral articular cartilage; hh, humeral head; ssc, subscapularis; ld, latissimus dorsi; lht, long tendon of triceps brachii. Orientation: left shoulder axillary view with humerus in abduction and external rotation; med, medial; sup, superior; inf, inferior.

We also determined the distance between the proximal border of each tendon and the cartilage rim of the humeral head along the proximal projection of the crest of the lesser tubercle on to the anterior rim of the greater tubercle (tendon-cartilage distance, TCD). All measurements were repeated for each specimen (five times for the latissimus dorsi and three times for the teres major), and then averaged per specimen. These averaged values were used for statistical analysis (SPSS for Windows, version 11.5, ®SPSS Belux, 1020 Brussels, Belgium). Parametric correlations were examined on the basis of Pearson's correlation coefficient and nonparametric correlations were examined on the basis of Spearman's rho coefficient.

We also studied the posterior part of the latissimus dorsi. After removing the skin and subcutaneous fat from the back, we observed the course and any possible attachment of the latissimus dorsi around the scapula. We did not study the origin of the latissimus dorsi from the back (spinous processes of thoracic vertebrae 7 to 12, thoracolumbar fascia, iliac crest, lower ribs) (Williams and Warwick, 1980), as we felt that this was not relevant to the present question. After fully detaching the glenohumeral capsule, the latissimus dorsi was studied in more detail. The spatial relationship of the latissimus dorsi to the humeral head and the inferior angle of the scapula as well as the orientation of the muscle and its tendon in different positions of abduction and rotation of the humerus were noted.

## RESULTS

### Morphology and Measurements of the Tendons

The tendon of the latissimus dorsi was attached anteriorly on the humerus to the lateral border of the crest of the lesser tubercle. The tendon had either a wing-like or a quadrilateral shape (Fig. 1). Most of the deep surface of the tendon of the latissimus dorsi was separated from the underlying tendon of the teres major by a bursa. Close to their insertions these tendons became adherent but could be separated easily by dissection. The teres major tendon was inserted more medially on the crest of the lesser tubercle. The point of insertion of the proximal border of the teres major tendon was slightly more distal than that of the tendon of the latissimus dorsi. The tendon of the teres major was generally narrower than the tendon of the latissimus dorsi and, in most cases, thus completely obscured from view by the latissimus dorsi in an anterior dissection of the shoulder. The teres major originated from the inferolateral part of the dorsal surface of the scapula. As the latissimus dorsi ran from its origin on the back to its humeral insertion, it wound upon itself and around the teres major. This description conforms with that found in most anatomy textbooks (Henle, 1855; Beaunis and Bouchard, 1868; Hyrtl, 1871; Krause, 1879; Hartmann, 1881; Testut, 1884; Debierre, 1890; Testut and Latarjet, 1948; Williams and Warwick, 1980)

### Relationship of Latissimus Dorsi to Scapula

The latissimus dorsi crossed the inferior angle of the scapula. We observed three different forms in the relationship of the latissimus dorsi to the inferior angle of the scapula. For ease of reference in the following presentation of Results and Discussion, we will designate them type 1, type 2a, and type 2b scapular connections. In 43 out of 100 specimens, a substantial amount of muscular fibers of the latissimus dorsi arose from the inferior angle (type 1; Fig. 2a). In 57 out of 100 specimens, there were only a few or no muscular fibers from the scapula to the latissimus dorsi. In these specimens, there was either a soft fibrous link between the bulk of the latissimus dorsi and the inferior angle of the scapula in 36 specimens (type 2a; Fig. 2b), or a bursa and no connecting tissue between the two structures in the remaining 21 shoulders (type 2b; Fig. 2c).

### Relationship of Latissimus Dorsi to Humeral Head

The tendon of the latissimus dorsi always covered the most inferior part of the humeral insertion

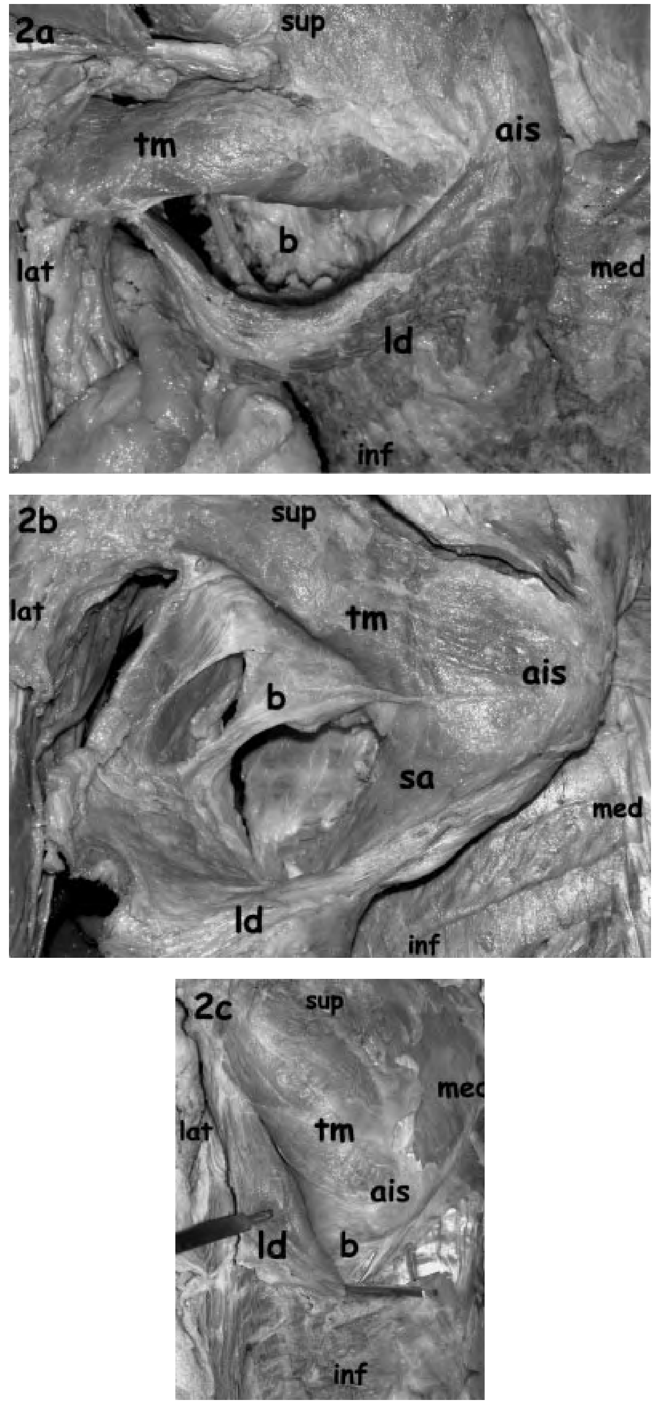


**Fig. 2.** The different types of scapular connection of latissimus dorsi in relation to the inferior angle of the scapula. **A:** substantial amount of muscular fibres from the latissimus dorsi arising from the inferior angle (type 1); **B:** soft fibrous link between the bulk of the latissimus dorsi and the inferior angle but few or no connecting muscular fibres (type 2a); **C:** bursa and no connecting tissue between the bulk of the latissimus dorsi and the inferior angle (type 2b). ld, latissimus dorsi; tm, teres major; b, bursa; ais, inferior angle of the scapula; sa, serratus anterior; ssc, subscapularis; hh, humeral head; lht, long head triceps. Orientation: left shoulders dorsal view with humerus in neutral position; med, medial, lat, lateral, sup, superior, inf, inferior.

of the glenohumeral capsule. The bottom of the V-like insertion of the capsule on the humerus descended 2–12 mm under the proximal border of the tendon of the latissimus dorsi. This tendon has to be carefully elevated from the underlying capsule to be able to detach the capsule from the humerus in a humerus-based cutting sequence, as carried out in Part II. The distance between the proximal border of the tendon of the latissimus dorsi and the cartilaginous rim closest to its line of insertion (TCD) was 12.6–31.6 mm (mean 21.1 mm ± 5.11 mm). The insertion of the tendon itself was 41.4–62.8 mm wide (mean 50.4 mm ± 5.54 mm) and the upper border of the tendon was 50.4–98.4 mm long (mean 66.6 mm ± 9.83 mm). The TCD for the teres major tendon was 31.3–45.0 mm (mean 37.4 mm ± 3.68 mm) whereas the distance between the proximal borders of the two tendons was 10.0–19.0 mm (mean 16.2 mm ± 2.27 mm). Distally, the tendons of latissimus dorsi and teres major ended at the same level, except in 12 specimens in which the distal border of the tendon of the teres major was inserted up to 20 mm more distally than that of the latissimus dorsi.

When moving the humerus into abduction and external rotation, the following observations were made:

1. When the TCD for the latissimus dorsi was small and when there was a type 1 scapular connection, abduction and external rotation tensed the latissimus dorsi by winding it around the rotating humerus, giving the tendon a more vertical course. When the latissimus dorsi was tightly connected to the inferior angle of the scapula, these fibers also retained the muscle and its tendon more medially. The medial border of the tendon of the latissimus dorsi then ran parallel with or even crossed over the lateral border of the subscapularis in the apprehension position. In this situation, the proximal border of the tendon of the latissimus dorsi formed an anteroinferior hammock for the humeral head (Fig. 3a).
2. When the TCD for the latissimus dorsi was small, combined with a type 2 scapular connection, the latissimus dorsi was not retained medially in abduction and external rotation. In 90° of external rotation and 90° of abduction (apprehension position), the subscapularis and latissimus dorsi diverged and a gap occurred between the two muscles at the level of the anteroinferior part of the humeral head. Nevertheless, the small TCD still caused the latissimus dorsi to have an inferior hammock-like effect, even in absence of a muscular connection to the scapula (Fig. 3b).
3. With a large TCD and irrespective of the scapular connection, the tendon of the latissimus dorsi did not cross over or just under the humeral head in abduction and external rotation. The medial border of the latissimus dorsi tendon and lateral border of the subscapularis diverged from one another even in adduction, again leaving a gap between the two tendons in the apprehension position (Figs. 3c and 3d).

## Statistical Analysis

There was very little overlap of TCD measurements between types of scapular connection. A type 2 scapular connection was usually associated with a wider TCD.

- For the value of TCD, the association with type of scapular connection was highly significant (Pearson's chi-square, $P < 0.001$).
- We used the mean for the TCD as a cut-off value to divide the TCD into two subgroups: close-by ("1" for statistical analysis), when the TCD was less than 20 mm, and far-off ("2" for statistical analysis), when the TCD was more than 20 mm.
- There were 50 tendons with a close-by insertion that had a type 1 scapular connection and 104 that had a type 2 scapular connection. In contrast, there were 125 tendons with a far-off insertion that had a type 1 scapular connection and 21 that had a type 2 scapular connection. This association was also highly significant (Pearson's chi-square, $P < 0.001$).

In addition, there were significant correlations between:

- type of TCD (close-by vs. far-off) and length (Spearman's rho = 0.310, $P = 0.02$) and width (Spearman's rho = 0.292, $P = 0.02$) of the tendon of the latissimus dorsi,
- value of TCD and length (Pearson's r = 0.339, $P = 0.008$) and width (Pearson's r = 0.301, $P = 0.02$),
- length and width of the tendon (Pearson's r = 0.555, $P < 0.001$).

A tendon of the latissimus dorsi that was close-by was, on average, shorter (63.6 mm vs. 69.9 mm, $P =$

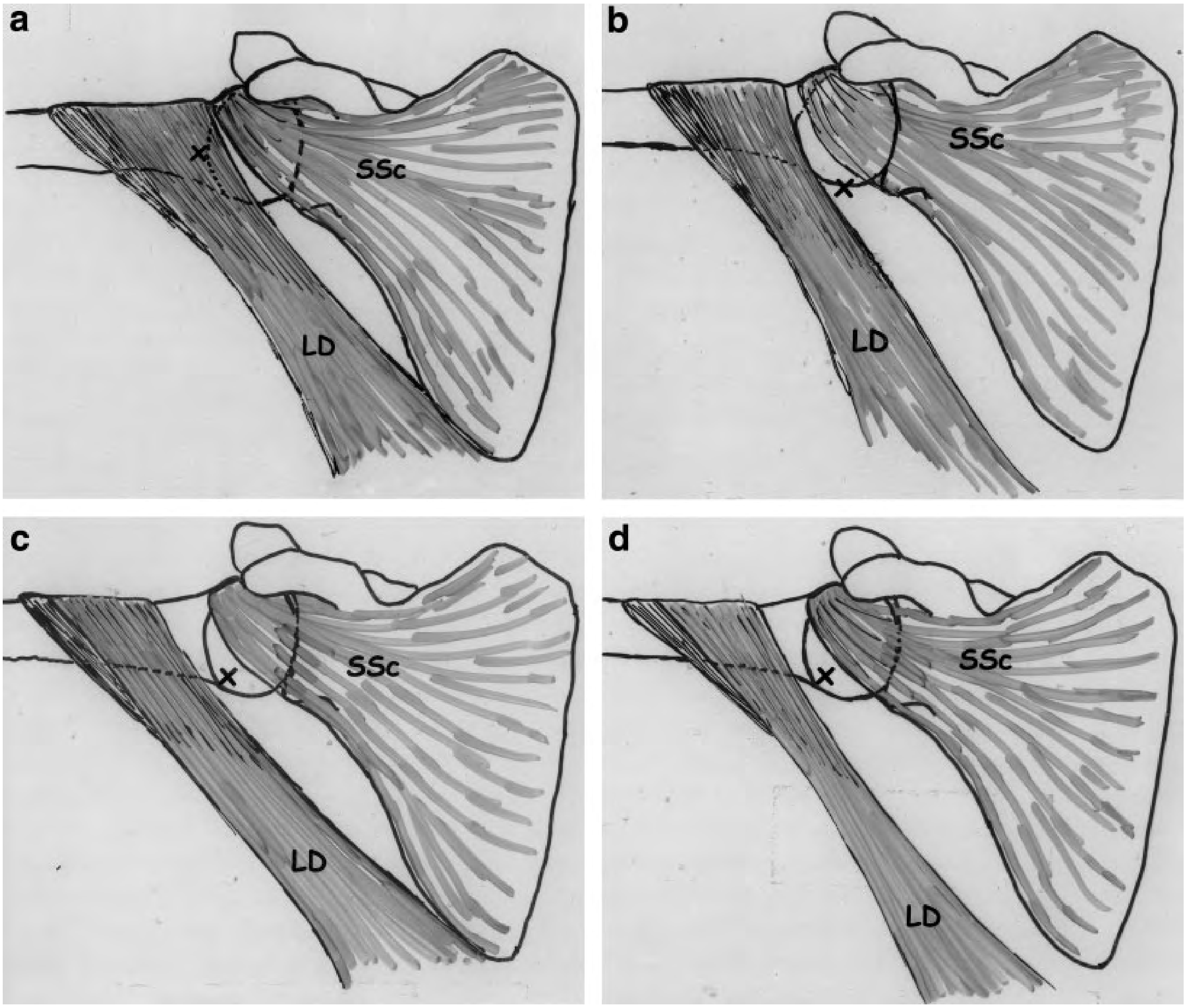


**Fig. 3.** Schematic representation of the relationship between the tendon of the latissimus dorsi and the humeral head cartilage with overlying subscapularis and with the inferior angle of the scapula (frontal view). The right humerus is positioned in 90° of glenohumeral abduction and 90° of external rotation. **A:** Small TCD with type 1 scapular connection; **B:** small TCD with type 2 scapular connection; **C:** wide TCD with type 1 scapular connection; **D:** wide TCD with type 2 scapular connection. LD, latissimus dorsi; SSc, subscapularis; x, humeral head with cartilaginous rim.

0.011) and less wide (48.7 mm vs. 52.3 mm, $P$ = 0.010) than a tendon that was far-off.

## DISCUSSION

The morphology of the latissimus dorsi has mainly been studied for its relevance to reconstructive surgery. A PubMed search revealed more than 300 papers that describe the use of this muscle as a pedicled or as a free vascularized transfer flap in the treatment of severe soft tissue defects after tumor surgery or trauma and for cardiomyoplasty. It also is often transferred locally for compensating functional defects as for example caused by brachial plexus palsy or massive rotator cuff defects. Unfortunately, the anatomic studies of the latissimus dorsi seem to be limited to the form of its muscle belly and its neurovascular supply (Bostwick et al., 1980; Bartlett et al., 1981; Tobin et al., 1981; Stevenson et al., 1984; Angrigiani et al., 1995; Cleeman et al., 2003). Some anatomy textbooks give detailed descriptions of the latissimus dorsi, but do not give dimensions of the tendon. Most authors state that the latissimus dorsi covers the inferior part of the scapula and that there is usually an intervening bursa, although an accessory muscle bundle can arise from the inferior angle of the scapula (Henle, 1855; Beaunis and Bouchard, 1868; Hyrtl, 1871; Krause, 1879; Testut, 1884; Debierre, 1890; Testut and Latarjet, 1948; Bergman et al., 1988). Other studies mainly addressed anatomic variations such as Langer's arcus axillaris (Sachatello, 1977; Clarijs et al., 1996; Clarys et al., 1996; Yuksel et al., 1996; Petrasek et al., 1997; Kalaycioglu et al., 1998; Miguel et al., 2001; Bonastre et al., 2002). Two notable exceptions are the case study of the conjoint tendon of teres major and latissimus dorsi by Beck and Hoffer (1989) and the article on the scapulothoracic articulation by Williams et al. (1999). Thus, despite an extensive literature search, we found no study referring to the effect of the latissimus dorsi on the behavior of the humeral head when subjected to a dislocating maneuver.

The study by Williams et al. (1999) is, to our knowledge, the only one that describes the relationship of the latissimus dorsi to the inferior angle of the scapula. Although the superior fibers of the latissimus dorsi covered this angle in all of their eight cadaver shoulders, no specimen demonstrated an actual muscular scapular connection (type 1 in our study). Half of their specimens had a well-defined bursa between muscle and scapula (type 2b in our study), whereas in the other half this space was filled with loose areolar tissue (type 2a in our study). In the present study, 43% of shoulders demonstrated a partial muscular origin of the latissimus dorsi from the inferior angle of the scapula. This connection may limit the free excursion of the tendon and muscle when the humerus is moved into abduction and into external rotation. The more vertical course of the latissimus dorsi as compared with a more oblique course depending on the type of scapular connection may be one factor in the role the latissimus dorsi seems to play in altering dislocation behavior. Cleeman et al. (2003) have measured the tendons of latissimus dorsi and teres major, but this was confined to their width and length and their distance to surrounding neurovascular structures for use of the latissimus dorsi in reconstructive surgery. We found no reference to the distance between the tendon and the articular cartilage of the humeral head. In our 100 specimens, we found a broad range in this distance. It is conceivable that the hammock, formed by the tendon of the latissimus dorsi under or even in front of the humeral head when the tendon is closer to the humeral head, may restrain the head when it is subjected to a dislocating force in abduction and external rotation.

The inferior edge of the humeral insertion of the glenohumeral capsule is covered by the tendon of the latissimus dorsi. Although tendon and capsule are not actually interconnected, the double-layered configuration may provide some stronger tendinous protection for the relatively weak inferior capsule. The tendon of the latissimus dorsi would in that respect close the ring of tendinous reinforcement around the humeral side of the capsule, which is formed by the tendons of the rotator cuff.

The second part of this study, tries to correlate TCD and the type of scapular connection with actual dislocation behavior in a sequential cutting protocol.

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Clinical Anatomy 18:500–509 (2005)

ORIGINAL COMMUNICATION

# Significance of the Latissimus Dorsi for Shoulder Instability. II. Its Influence on Dislocation Behavior in a Sequential Cutting Protocol of the Glenohumeral Capsule

N. POULIART[1,2,3]* AND O. GAGEY[3,4]

[1]*Department of Orthopaedics and Traumatology, Academic Hospital Vrije Universiteit Brussel, Brussels, Belgium*
[2]*Department of Human Anatomy, Vrije Universiteit Brussel, Brussels, Belgium*
[3]*Institut d'Anatomie, Université Réné Descartes Paris V, Paris, France*
[4]*Service d'Orthopédie, Hôpital Bicêtre, Université Paris-Sud, Kremlin-Bicêtre, France*

In a cadaveric instability model that leaves all muscles intact initially, the latissimus dorsi seemed to play a role when complete section of the glenohumeral capsuloligamentous structures did not result in a locked anteroinferior dislocation. The present study was carried out to determine whether the latissimus dorsi does truly affect dislocation in a modified cutting protocol, and to find an anatomic explanation for this apparent behavior. This article (Part II) details the results of a sequential cutting study and relates these results with the anatomic findings of Part I. In 75 shoulders, the influence of the latissimus dorsi on dislocation behavior in the apprehension position after section of all capsuloligamentous structures was examined. After cutting all capsuloligamentous structures, either on the glenoid or on the humeral side, the tendon of either the latissimus dorsi or the subscapularis was cut. Capsular lesions on the glenoid side (20 shoulders) resulted in a locked dislocation in 16 specimens. In the other four shoulders, there was a metastable dislocation after cutting the entire capsule, which did not change after cutting either tendon. With lesions on the humeral side (55 shoulders), three possibilities arose: metastable (17 shoulders), locked anterior (9 shoulders) or locked anteroinferior (29 shoulders) dislocation. This difference in dislocation behavior was related to the variability of the tendon–cartilage distance (TCD) and the type of scapular connection of the latissimus dorsi. A locked anteroinferior dislocation was always observed when the TCD was more than 20 mm, regardless of the type of scapular connection. With a TCD $<$ 20 mm, a metastable dislocation was the result when there was a type 1 scapular connection and a locked anterior dislocation was seen when there was a type 2 scapular connection. The tendon of the latissimus dorsi can restrain the humeral head from dropping inferiorly or can lead to a spontaneous reduction of a dislocation, depending on its anatomy. This effect can only take place in the infrequent situation of humeral avulsion of the glenohumeral ligaments. This may be an explanation for the relative paucity of these lesions in clinical instability series. Clin. Anat. 18:500–509, 2005. © 2005 Wiley-Liss, Inc.

**Key words: shoulder; dislocation; instability; experimental; latissimus dorsi; muscle; glenohumeral ligaments**

## INTRODUCTION

To study the consequences of ligament section on glenohumeral stability, we developed a cadaver shoulder model in which all the surrounding muscles were initially maintained intact. In this model, we studied the anteroinferior dislocation behavior of the

*Correspondence to: Dr. Nicole Pouliart, Department of Orthopaedics and Traumatology, Academic Hospital Vrije Universiteit Brussel, Laarbeeklaan 101, 1090 Brussels, Belgium.
E-mail: nicole.pouliart@az.vub.ac.be

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humeral head after section of the glenohumeral capsuloligamentous structures on the humeral side. In the first 30 specimens of this study, a load-and-shift test aimed at dislocating the shoulder did not result in a locked dislocation in 31% of specimens, even after section of all glenohumeral ligaments. We had the impression that the tendon of the latissimus dorsi played a role in this dislocation behavior. To verify this impression and to find an explanation for the difference in dislocation behavior, the initial dynamic study of capsuloligamentous lesions on the humeral side was expanded. We included capsuloligamentous sections on the glenoid side for comparison with additional specimens with humeral-sided cuts and, for both sites, we added section of the posterior capsule and section of either the tendon of the latissimus dorsi or the tendon of the subscapularis to the sequential cutting protocol. The results of this expanded protocol are presented in this Part II. Part I details the results of an anatomic study of the latissimus dorsi.

## MATERIALS AND METHODS

### Dynamic Cutting Study

In total, 75 fresh adult cadaver shoulders from donated cadavers (aged 50–103 years) at the Institut d'Anatomie of Université Réné Descartes were studied. These shoulders were submitted to the testing protocol outlined below after section of the capsuloligamentous structures on the humeral side of the joint (55 shoulders) or after section on the glenoid side (20 shoulders). An axillary approach to the inferior glenohumeral capsule and the tendon of the latissimus dorsi was used. Any surrounding muscles and the capsule were not cut initially. The belly of the biceps brachii, the deltoid, and the pectoralis major muscles were reflected laterally. The neurovascular bundle was removed and the quadrilateral space was cleaned out. The subscapularis, the underlying glenohumeral capsule, and the tendons of latissimus dorsi and teres major were freed from fatty and loose areolar tissue. The subscapularis was carefully dissected from the underlying capsule and reflected superiorly without cutting it.

**Division of the capsule and ligaments.** On the humeral side, in 40 (humerus-based) shoulders, the capsuloligamentous was cut starting at the posterior part of the inferior glenohumeral ligament and proceeding anteriorly to include the axillary part and the anterior part of the same ligament. Next, the middle and then the superior glenohumeral ligament were cut. In the other 15 shoulders in which the humeral side of the capsule was cut, the sequence started in the anterior part of the inferior glenohumeral ligament, before proceeding to the posterior part of the inferior, the middle and then the superior glenohumeral ligament. Finally, in all 55 specimens, the posterior capsule was also detached to be absolutely sure that no capsuloligamentous structures could be responsible for any observed difference in dislocation behavior. On the humeral side, the entire inferior capsule could easily be reached by slightly lifting the tendon of the latissimus dorsi from the inferior pole of the capsular insertion. By carefully separating the tendon of the subscapularis from the underlying capsule and reflecting the muscle belly superiorly, the middle humeral ligament zone could be reached. Once this was cut, the superior capsular zone could also be reached. The middle and superior capsular zones were cut immediately medial to the area where the rotator cuff tendons and the capsule merge. By carefully controlling the depth of the capsular cut, the tendons of the rotator cuff and the long tendon of the biceps brachii could be maintained intact. On the glenoid side, in all 20 specimens in which the capsule was cut at the glenoid side, the cutting sequence was started in either the anterior part of the inferior glenohumeral ligament or in the middle glenohumeral ligament, before proceeding to the other capsuloligamentous zones. On the glenoid side, care was taken not to cut the long tendon of the biceps brachii and the capsule was not detached from the insertion of the long head of the triceps brachii. The middle and anteroinferior capsular zone could be reached by separating the subscapularis belly from the underlying capsule and reflecting the muscle upwards. The superior zone could be reached after cutting the middle zone and the posteroinferior zone could be reached after cutting the anteroinferior zone. The integrity of all tendons was verified at the end of the entire testing procedure by further dissection and inspection of the intraarticular aspect of the capsule and the rotator cuff tendons.

**Load-and-shift tests.** After each capsuloligamentous cut, the specimens were subjected to a load-and-shift test in the apprehension position to determine whether they had dislocated or not. A goniometer was used to position the humerus in 90° of external rotation and 90° of abduction (scapulohumeral elevation in the coronal plane). We opted for the apprehension maneuver to test instability in our dislocation studies, as this closely mimics the clinical situation where anteroinferior instability commonly occurs in abduction with external rotation and is usually examined with this maneuver. The clinical apprehension position usually involves 90° of total humeral abduction and thus more closely corre-

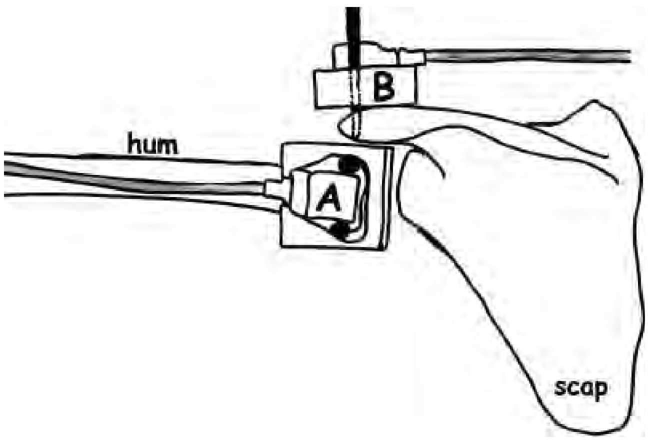



**Fig. 1.** Diagrammatic representation of the alignment of the electromagnetic sensors with the humerus and the scapula in a left shoulder seen from behind. A, master sensor fixed to the superolateral part of the humerus with threaded stainless steel pins through a custom-made polyethylene holder; B, slave sensor fixed to superior part of the acromion; hum, humerus; scap, scapula.

sponds to 60° of glenohumeral abduction. However, according to the original article by Gerber and Ganz (1984), the test can be done at 45°, 90°, and 135° of humeral abduction. In this test, the inferior glenohumeral is stressed at 90° or more of abduction and the middle glenohumeral ligament and subscapularis at 45° of abduction. We opted for 90° of glenohumeral abduction because anteroinferior stability was the main objective for this test and this position was more reproducible in the present manual testing procedure. It was also used by Black et al. (1999) in their biomechanical study of the Bankart repair.

An electromagnetic tracking device (EMTD) ("Flock-of-birds", ®Ascension Technology, Burlington, Vermont 05402, USA) was used to record the position of the humeral head in relation to the glenoid. One sensor was fixed rigidly with threaded pins to the lateral side of the humerus 2 cm distal to the greater tubercle. This allowed the sensor to be positioned in line with the direction of the anterior force in the apprehension position while not interfering with mobility. The second sensor was fixed rigidly on top of the acromion so that it was at right angles with the first sensor in the apprehension position (Fig. 1). The electromagnetic transmitter was placed near the specimens in use and in line with the scapular plane. The entire setup was positioned on a granite table, so as to remove all ferromagnetic materials from the work field. This system provided an objective measure of the resulting instability, as the amount of anterior, medial, and inferior translation of the sensors due to the apprehension maneuver can be recorded before and after application of the applied forces. A handheld dynamometer (MicroFET2, ®Hoggan Health Industries, distributed by Biometrics EuropeBV, 1322 AD Almere, The Netherlands) was used to apply an anteriorly directed translation force of 50 N (maximal) combined with a compression force of 50 N (maximal) in line with the axis of the humerus in the apprehension position. The relative positions of both sensors and thus the obtained translation were recorded. Then, the humerus was brought back into its resting position of 0° of abduction and neutral rotation without a reduction maneuver. The relative positions of humeral head and glenoid were again recorded to verify whether or not reduction had occurred. Anterior or anteroinferior dislocation is a combination of anterior translation with medial (and inferior) translation. Dislocation does not occur without medial translation, which is therefore the crucial determinant for dislocation. We defined a locked dislocation as a dislocation where the humerus does not return to its normal glenohumeral relationship upon being brought into the resting position without a reduction maneuver. A metastable dislocation is defined as one where the humerus is relocated onto the glenoid when bringing the humerus back to the resting position without a reduction maneuver (Fig. 2). For this study, the important endpoint for the apprehension maneuver was dislocation and possible relocation after returning the arm to the resting position.

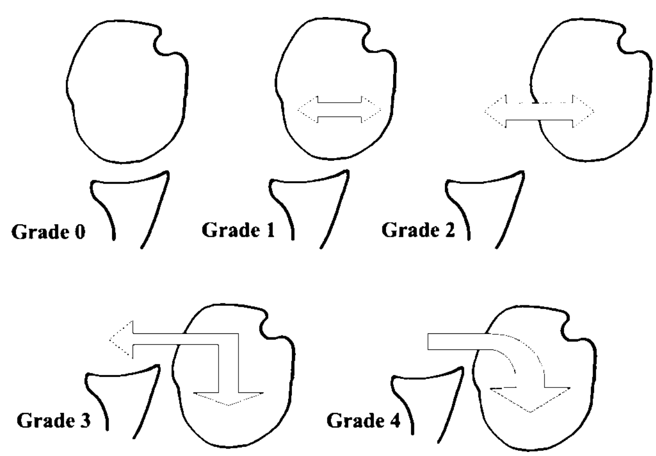



**Fig. 2.** Scale used for grading of dislocation behavior under influence of a load-and-shift test (expansion of ASES scale of instability). Grade 0: no translation of the humeral head in relation to the glenoid. Grade 1: anterior translation of the humeral head of less than 10 mm; no medial translation; the head does not pass the glenoid rim (drawer). Grade 2: anterior translation of the humeral head of more than 10 mm, so that it passes the glenoid rim; still no medial translation; the head does not pass over plane of glenoid (subluxation). Grade 3: anterior translation of the humeral head of more than 10 mm, so that it passes the glenoid rim, in association with medial translation; the head reduces spontaneously when the arm is brought back into neutral position (metastable dislocation). Grade 4: displacement of the humeral head as in grade 3, but the head does not reduce without a specific reduction maneuver (locked dislocation).

**TABLE 1. Overview of Experimental Results With Anatomic Correlation**

| Selective cutting side (number of specimens) | Dislocation after all capsuloligamentous cuts | Tendinous cut if not blocked | Dislocation after additional (tendinous) cut | Number of specimens with TSC type 1 | TCD for LD (mm) |
|---|---|---|---|---|---|
| *Glenoid-based (GB) (n = 20)* | | | | 8 | |
| Locked | 16 (80)[a] | | | 5 | NA |
| Metastable | 4 (20) | | | 3 | NA |
| LD | | 2 | 2[b] | 2 | NA |
| SSc | | 2 | 2[b] | 1 | NA |
| *Humerus-based (HB) (n = 55)* | | | | 23 | |
| Locked | 38 (69) | | | 6 | |
| Anteroinferior | 29 (53) | | | 6 | >20 |
| Anterior | 9 (16) | | | | |
| LD | | | 0[c] | 0 | ≤20 |
| Metastable | 17 (31) | | | 17 | |
| LD | | 9 | | | |
| Locked | | | 9[c] | 9 | ≤20 |
| SSc | | 8 | | | |
| Locked | | | 8[c] | 8 | ≤20 |

LD, latissimus dorsi; SSc, subscapularis; TCD, tendon-cartilage distance; TSC, type of scapular connection; NA, not available.
[a]Values in parentheses indicate percentages.
[b]Metastable dislocation after cutting SSc or LD.
[c]Locked anteroinferior after cutting LD in all 9.

In those shoulders where a locked dislocation did not result from complete capsuloligamentous section, the load-and-shift test was repeated after cutting either the latissimus dorsi tendon or the subscapularis tendon. In one half of specimens, the proximal 2 cm of the latissimus dorsi insertion was detached from the humerus. In the other half, the subscapularis tendon was completely detached from the humerus while leaving the latissimus dorsi intact. This was done to verify that the subscapularis tendon was not the major factor explaining the restraint seemingly provided by the latissimus dorsi.

### Anatomo-Dynamic Correlation

Finally, the dislocation behavior after complete section of the capsuloligamentous structures and either tendon in the dynamic part of the study was linked with the observations from the morphological study of the relevant specimens (Part I). For group comparisons, the parametric one-way ANOVA test, with post hoc Bonferoni analysis where appropriate, and the nonparametric Kruskal–Wallis test were used. Parametric correlations were examined on the basis of Pearson's correlation coefficient and nonparametric correlations on the basis of Spearman's rho coefficient.

## RESULTS

### Dynamic Cutting Study (Table 1)

1. In the glenoid-based group (20 specimens), after sectioning all the capsuloligamentous structures on the glenoid side, the load-and-shift test produced a locked anteroinferior dislocation in 16 specimens (80%). In the other four specimens, applying the load-and-shift test after complete sectioning of the capsuloligamentous complex resulted in a metastable dislocation (20%). After cutting the latissimus dorsi tendon (two specimens) or detaching the subscapularis (two specimens), the result of the load-and-shift test remained a metastable dislocation.
2. In the humerus-based group (55 specimens), after sectioning the capsuloligamentous structures on the humeral side, there were three possible results.
   a. The apprehension maneuver resulted in a locked dislocation in 38 specimens (69%).
      i. In 29 of these 38 shoulders, the locked dislocation was anteroinferior.
      ii. In the other 9 of these 38 shoulders, the apprehension maneuver resulted in a locked anterior dislocation with the humeral head translated anteriorly and medially, but not inferiorly. Cutting the latissimus dorsi tendon in these nine specimens resulted in an inferior drop of the humeral head.
   b. In the remaining 17 of 55 shoulders (31%), the apprehension maneuver resulted only in a metastable dislocation. After detaching the subscapularis tendon (eight shoulders) or incising the latissimus dorsi tendon (nine shoulders), the apprehension maneuver resulted in a locked anteroinferior dislocation in all these specimens.

As the relevant endpoint of this study was the grade of dislocation resulting from cutting the entire

TABLE 2. Comparison of Metric Parameters of Latissimus Dorsi for Type of Scapular Connection

| | TSC | N | Mean | SD | Std. error | 95% Confidence interval for mean Lower Bound | 95% Confidence interval for mean Upper Bound | Minimum | Maximum | P values* |
|---|---|---|---|---|---|---|---|---|---|---|
| TCD | Type 1 | 27 | 18.43 | 4.38 | 0.84 | 16.70 | 20.16 | 12.6 | 30.8 | |
| | Type 2 | 33 | 23.21 | 4.69 | 0.82 | 21.55 | 24.87 | 14 | 31.6 | |
| | *Total* | *60* | *21.06* | *5.11* | *0.66* | *19.74* | *22.38* | *12.6* | *31.6* | 0.001 |
| LD-width | Type 1 | 27 | 50.45 | 4.18 | 0.80 | 48.80 | 52.11 | 42.2 | 61.6 | |
| | Type 2 | 33 | 50.40 | 6.52 | 1.13 | 48.09 | 52.71 | 41.4 | 62.8 | |
| | *Total* | *60* | *50.42* | *5.54* | *0.72* | *48.99* | *51.86* | *41.4* | *62.8* | 0.972 |
| LD-length | Type 1 | 27 | 67.80 | 7.72 | 1.48 | 64.75 | 70.85 | 54.2 | 84.6 | |
| | Type 2 | 33 | 65.68 | 11.31 | 1.97 | 61.68 | 69.69 | 50.4 | 98.4 | |
| | *Total* | *60* | *66.64* | *9.83* | *1.27* | *64.10* | *69.18* | *50.4* | *98.4* | 0.419 |

TSC, type of scapular connection; TCD, tendon-cartilage distance; SD, standard deviation; LD, latissimus dorsi.
*One-way ANOVA, type 1 vs. type 2.

capsule, we decided to detail only these results and not those of the individual capsuloligamentous cutting steps. The original study protocol of capsuloligamentous lesions on the humeral side was adapted by cutting the tendon of the latissimus dorsi before the ligamentous cutting sequence was carried out. In this way, the protocol was adjusted for the possible effect that the latissimus dorsi could have.

### Anatomo-Dynamic Correlation

**Relation of scapular connection type, tendon size, and tendon–cartilage distance (Table 2).** For the 75 specimens studied dynamically, the distribution of both types of scapular connection (see Part I for details of this categorization) was: type 1 in 31 specimens (41.3%), type 2a in 29 specimens (38.7%), and type 2b in 15 shoulders (20.0%).

A tendon with type 1 scapular connection was closer to the cartilage rim than its counterpart with type 2 (a or b) scapular connection ($P < 0.001$), but showed no statistically significant difference for its length ($P = 0.972$) or width ($P = 0.412$).

**Relation of dislocation behavior, type of scapular connection, and tendon–cartilage distance of the latissimus dorsi (Tables 1 and 3).** When looking at the latissimus dorsi in relation to the dislocation behavior of the humerus during positioning of the humerus in the apprehension position and during the load-and-shift test, the following observations could be made (Table 1):

1. **glenoid-based capsuloligamentous section (20 specimens)** In these 20 specimens, the tendon–cartilage distance (TCD) could not be measured, as the glenohumeral capsule covered the area proximal to the tendon of the latissimus dorsi and thus obscured the articular cartilage of the humeral head. A type 1 scapular connection was found in 8 specimens, of which 5 demonstrated a locked dislocation and 3 a metastable dislocation after complete capsuloligamentous section. A type 2 scapular connection was seen in the other 12 specimens, of which 11 demonstrated a locked dislocation and 1 a metastable dislocation.
2. **humerus-based capsuloligamentous section (55 specimens)**
   - *(a) metastable dislocation (17 specimens):* All 17 specimens presented with a type 1 scapular connection of the latissimus dorsi. All these specimens had a TCD for the latissimus dorsi of less than 20 mm. In this situation, the humeral head abutted against the proximal border of the tendon of the latissimus dorsi (Fig. 2).
   - *(b) locked dislocation (38 specimens):* i. In nine specimens, inferior displacement did not occur at locked dislocation. These specimens did not have a type 1 scapular connection, but the latissimus dorsi tendon always had a TCD of less than 20 mm. Without the presence of a scapular origin, the subscapularis and latissimus dorsi diverged in the apprehension position, leaving a gap that seemed to allow locked dislocation. Inferior displacement seemed to be prevented by the tendon of the latissimus dorsi that ran very close under the humeral head because of a small TCD.

     ii. In six specimens with a locked anteroinferior dislocation after humeral ligamentous section, there was a type 1 scapular connection,

**TABLE 3. Comparison of Metric Parameters of Latissimus Dorsi for Grade of Dislocation**

| | Dislocation grade | N | Mean | SD | Std. error | 95% Confidence interval for mean Lower Bound | 95% Confidence interval for mean Upper Bound | Minimum | Maximum | P values* |
|---|---|---|---|---|---|---|---|---|---|---|
| TCD | Grade 3 | 17 | 16.40 | 1.92 | 0.46 | 15.42 | 17.38 | 12.6 | 18.6 | |
| | Grade 4 ant | 9 | 17.22 | 1.70 | 0.57 | 15.92 | 18.53 | 14 | 19.6 | |
| | Grade 4 antinf | 29 | 25.68 | 2.94 | 0.55 | 24.56 | 26.79 | 21.2 | 31.6 | |
| | *Total* | *55* | *21.43* | *5.16* | *0.70* | *20.03* | *22.82* | *12.6* | *31.6* | < 0.001 |
| LD-width | Grade 3 | 17 | 51.06 | 4.31 | 1.04 | 48.85 | 53.27 | 42.2 | 61.6 | |
| | Grade 4 ant | 9 | 44.13 | 1.84 | 0.61 | 42.72 | 45.54 | 41.4 | 46.8 | |
| | Grade 4 antinf | 29 | 52.30 | 5.77 | 1.07 | 50.10 | 54.49 | 42.6 | 62.8 | |
| | *Total* | *55* | *50.58* | *5.64* | *0.76* | *49.05* | *52.10* | *41.4* | *62.8* | < 0.001 |
| LD-length | Grade 3 | 17 | 67.75 | 6.87 | 1.67 | 64.22 | 71.28 | 57.8 | 79.6 | |
| | Grade 4 ant | 9 | 55.00 | 4.17 | 1.39 | 51.80 | 58.20 | 50.4 | 64.2 | |
| | grade 4 antinf | 29 | 69.92 | 10.09 | 1.87 | 66.08 | 73.75 | 53.2 | 98.4 | |
| | *Total* | *55* | *66.81* | *9.90* | *1.33* | *64.13* | *69.48* | *50.4* | *98.4* | < 0.001 |

TCD, tendon–cartilage distance; SD, standard deviation; LD, latissimus dorsi.
*One-way ANOVA, all groups.

but the latissimus dorsi did not cross over the humeral head in the apprehension position because of a TCD of more than 20 mm. The medial border of the latissimus dorsi tendon and lateral border of the subscapularis diverged from one another even in adduction, again leaving a gap between both tendons in the apprehension position.

iii. In the other 23 specimens with lesions on the humeral side, the locked dislocation was anteroinferior. In these shoulders, the latissimus dorsi had a type 2a or 2b scapular connection in combination with a TCD of more than 20 mm (Fig. 2).

Since the tendon of the teres major was further away from the humeral head and since it had a more horizontal course, it did not appear to be in a position to impede the displacement of the humeral head in any of the specimens.

### Statistical Analysis

Table 3 summarizes the statistical data for these observations. It shows that the differences in TCD as well as the length and width of the latissimus dorsi were significant for the type of dislocation. Post hoc analysis attributed these differences to a significant difference (Bonferoni, $P < 0.001$) when comparing TCD for locked anteroinferior dislocation with either metastable dislocation or locked anterior dislocation. For tendon width and length, significant differences were seen (Bonferoni, $P < 0.001$) when comparing these parameters for locked anterior dislocation with either metastable or locked anteroinferior dislocation. Type 1 scapular connection was seen in both metastable and locked anteroinferior dislocation, whereas type 2 (a or b) scapular connection was not observed in metastable dislocations. With type 1 scapular connection, the difference in TCD for grade of dislocation (metastable or locked anteroinferior) was significant (one-way ANOVA, $P < 0.001$). With type 2 scapular connection, the differences in TCD ($P < 0.001$) and tendon width ($P < 0.001$) and length ($P < 0.001$) for the type of dislocation (locked anterior or anteroinferior) were significant (one-way ANOVA). Descriptive statistics and box-plot analysis grouped per grade of dislocation showed that there was no overlap whatsoever for TCD measurements between locked anteroinferior dislocation versus metastable or locked anterior dislocation, nor for type of scapular connection between metastable dislocation and locked (anterior or anteroinferior) dislocations. In our series, metastable and locked anterior dislocations were always associated with a TCD of less than 20 mm, whereas locked anteroinferior dislocation was always associated with a TCD of more than 20 mm. The Kruskal–Wallis chi-square test for comparison of type of dislocation and type of scapular connection was highly significant ($P < 0.001$). Correlation analysis also illustrated this intimate dependency with a high correlation coefficient between type of dislocation and value of TCD (Spearman's rho = 0.846, $P < 0.001$) as well as type of TCD (Spearman's rho = 0.955, $P < 0.001$).

## DISCUSSION

Several studies have examined the role of the so-called dynamic stabilizers of the glenohumeral joint (Cain et al., 1987; Blasier et al., 1992; Itoi et al., 1993, 1994a,b; Malicky et al., 1996; Pagnani et al., 1996; Soslowsky et al., 1997; Wuelker et al., 1998; Weiser et al., 1999; Lee et al., 2000; Halder et al., 2001; Kim et al., 2001; Lee and An, 2002). From these studies, we know that most of the muscles acting at the shoulder can stabilize the glenohumeral joint even in the face of severe capsuloligamentous lesions. The long and short heads of the biceps brachii, all the elements of the rotator cuff, the deltoid, and the coracobrachialis have thus been implicated in stabilizing the shoulder against anterior and inferior dislocating forces. The latissimus dorsi is mentioned as part of the scapular rotator muscles that may play a secondary role in shoulder stability by providing a stable scapular platform against which glenohumeral motion can then take place (Glousman et al., 1988; Brostrom et al., 1989; Blasier et al., 1992; Pagnani and Warren, 1994; Kronberg and Brostrom, 1995; Bigliani et al., 1996; Itoi et al., 1996; McMahon et al., 1996; Soslowsky et al., 1997; Wuelker et al., 1998). The latissimus dorsi has not, however, been considered to contribute directly to shoulder stability. This may, in part, be explained by the fact that most cadaver studies addressing static or dynamic stabilizers use an experimental setup in which the surrounding muscles are removed partially or even completely. Often, only the rotator cuff muscles and/or the long head of the biceps tendon are left in place (Ovesen and Sojbjerg, 1986; Wuelker et al., 1994; Apreleva et al., 1998; Lee et al., 2000).

In the present "intact muscle shoulder model" we demonstrated that the latissimus dorsi tendon can play a static role in impeding locked dislocation when the capsuloligamentous lesions are situated on the humeral side. This is due to its anatomic position in relation to the humeral head and its possible partial origin on the scapula. The latissimus dorsi plays no passive role when the lesions are on the glenoid side. Although our model leaves all muscles surrounding the glenohumeral joint intact, it is still a static model. In some cadaver studies, muscle forces and action have been simulated, but this was not part of our experimental protocol. Therefore, no inference from our results towards dynamic consequences should be made.

When relating dislocation type to scapular origin or TCD, three different situations for humerus-based lesions were observed.

1. The first determinator of dislocation behaviour under influence of the latissimus dorsi seemed to be the TCD. A locked anteroinferior dislocation occurred when the TCD was more than 20 mm (from here on designated as far-off), regardless of the type of scapular connection. When the TCD was less than 20 mm (close-by), the type of scapular connection came into play as a second determinator.
2. With a close-by tendon, locked anterior dislocation occurred when the latissimus dorsi did not have a muscular connection to the scapula (type 2 a or b).
3. Metastable dislocation occurred when the latissimus dorsi was close-by and had a type 1 scapular connection.

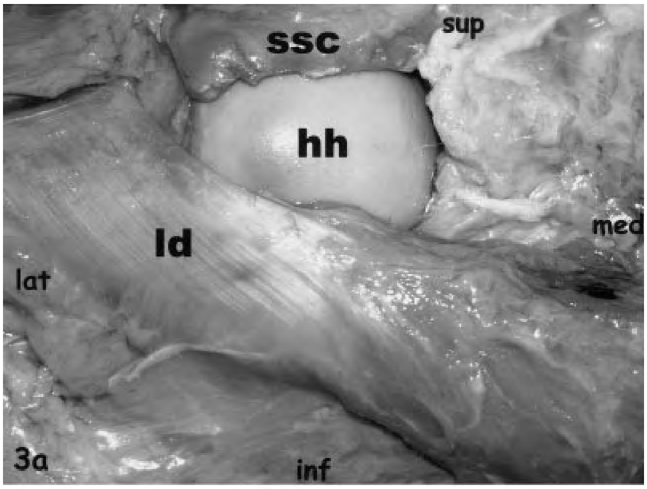
For glenoid-based lesions, the latissimus dorsi did not seem to be able to play a restraining role against dislocation.

These four possible situations have to be considered when trying to explain the mechanism behind the static role of the latissimus dorsi.

*Situation 1: Metastable dislocation in case of humerus-based lesions with the latissimus dorsi forming a hammock for the humeral head because of an insertion close to the cartilaginous rim and type 1 scapular connection* (Fig. 3a).

The abducted and externally rotated humeral head is crossed by or catches on the superior border of a taut latissimus dorsi tendon. Although the humeral head can be dislocated anteriorly, its inferior displacement is prevented by the hammock formed by the latissimus dorsi. When the subscapularis is intact in this situation and the capsule remains attached to it, these structures form a medial buttress that guides the humeral head back over the hammock into its reduced position. The dislocation can be considered metastable as it is reduced spontaneously by bringing the arm back into the neutral position. On the other hand, if the subscapularis is avulsed—simulated by detachment in this study—from its humeral insertion and subsequently retracted, the attached capsule can also recede medially. The buttressing effect of both structures is lost. The humeral head can then move more medially, away from the tendon to the soft muscular part of the latissimus dorsi, and the subscapularis cannot guide the humeral head back over the hammock into its reduced position. In this situation, a locked dislocation is the result, despite a catching latissimus dorsi tendon.

*Situation 2: Locked anterior dislocation in case of humerus-based lesions with the latissimus dorsi partially blocking the humeral head because of an insertion close to the cartilaginous rim but with absence of type 1 scapular connection.*



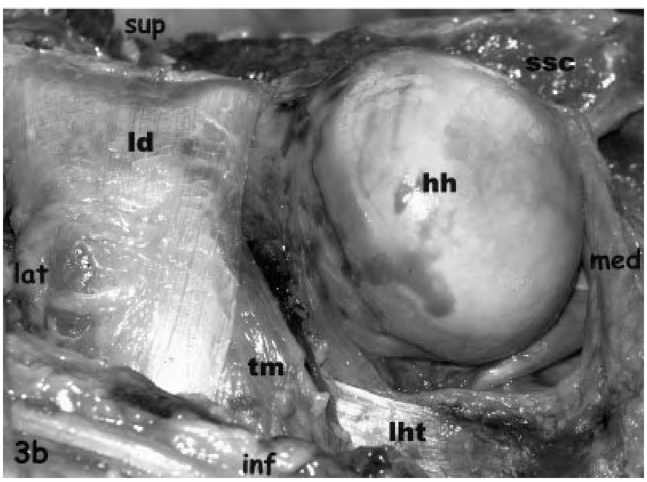




**Fig. 3.** Photographs illustrating the anatomo-clinical correlation and the relationship between latissimus dorsi and humeral head in the apprehension position. **a:** Humerus-based lesions with the latissimus dorsi forming a hammock for the humeral head because of type 1 scapular connection and an insertion close to the cartilaginous rim (as in Situation 1); **b:** Humerus-based lesions with the latissimus dorsi not blocking the humeral head due to an insertion far from the cartilaginous rim without type 1 scapular connection (as in Situation 3). ld, latissimus dorsi; tm, teres major; lht, long head triceps; hh, humeral head; ssc, subscapularis. Orientation: right shoulder, axillary view, humerus in 90° of external rotation and 90° of abduction; sup, superior; inf, inferior; med, medial; lat, lateral.

In this situation, there is a gap between the subscapularis and the latissimus dorsi in the apprehension position, because a scapular tether does not pull the tendon of the latissimus dorsi medially. The humeral head dislocates through this gap, which results in a purely anterior locked dislocation as the head cannot drop inferiorly because of the hammock formed by the latissimus dorsi tendon.

*Situation [vn8] 3: Locked anteroinferior dislocation in case of humerus-based lesions with the latissimus dorsi not blocking the humeral head because of an insertion far from the cartilaginous rim, regardless of scapular origin* (Fig. 3b).

Because the tendon of the latissimus dorsi is further away from the cartilage, it runs more laterally. In this situation, the humeral head can move more inferiorly during the apprehension manoeuvre, since it is not blocked by the latissimus dorsi. The head ends up distal to the subscapularis, thereby avoiding the medial capsular buttress formed by the subscapularis and the capsule. An anteroinferior locked dislocation ensues. The type of scapular connection does not play a role when the tendon is far away from the articular cartilage.

*Situation 4: Locked anteroinferior dislocation in case of glenoid-based lesions where the latissimus dorsi is never in a position to block the humeral head.*

The humeral head dislocates through a capsular gap that is more medial than when humerus-based lesions are present. It thus passes through the quadrilateral space and in front of the long head of the triceps brachii. Here, the latissimus dorsi is too far away from the humeral head to block it, and in consequence, a locked anteroinferior dislocation is possible.

The ability of the subscapularis to relocate the humeral head, even in a static manner, corresponds with the findings of Turkel et al. (1981), Ovesen and Nielsen (1985), and Glousman et al. (1988) that the subscapularis is an important dynamic stabilizer of the glenohumeral joint in the lower ranges of abduction.

The experimental observations made in the present study may have three clinical implications.

The passive function of the latissimus dorsi may be one of the factors explaining the occurrence of secondary instability. A first trauma results in a subluxation or in a spontaneously reduced (metastable) dislocation, and may therefore not be recognized as a dislocation. The dislocation may remain undocumented because it is reduced before radiographs can be taken. Locked dislocation may, in these cases, be prevented by the latissimus dorsi. If humeral lesions of the glenohumeral capsuloligamentous structures are present, these may therefore equally remain undiagnosed, especially when no chronic instability ensues. However, the capsuloligamentous structures have been damaged and will insufficiently contribute to stability if they do not heal properly. Thereafter, a second (“atraumatic”) event or progressive stretching of the secondary stabilizers (including the latissimus dorsi) may eventually result in a locked dislocation and subsequent chronic instability. More attention to the history and more detailed investigation for all possible lesions may identify this subset of patients with atraumatic recurrent dislocation. With respect to the important difference between a locked and a metastable dislocation, we would like to recommend a modification of the scale of instability adopted by the American Society of Elbow and Shoulder Surgeons (Richards et al., 1994). In the original grading scale, all types of dislocation are grouped together into grade 3. We would like to differentiate metastable from locked dislocations and therefore propose to expand the scale to four grades that retain grades 0 through 2, but differentiate grade 3 instability (metastable dislocation) and grade 4 instability (classic locked dislocation) (Fig. 1).

The possible clinical relevance of the latissimus dorsi has to be put into proper perspective, though. In the majority of clinical dislocations, lesions occur on the glenoid side of the capsuloligamentous structures. Therefore, the possible static role of the latissimus dorsi can only be relevant for a limited number of patients. On the other hand, for the reasons outlined above, the occurrence of humeral avulsion of the glenohumeral ligaments (HAGL) may be underestimated. In addition, the tendon of the latissimus dorsi may actually protect the humeral insertion of the inferior glenohumeral ligament, as the tendon usually covers the latter. This may complement the protection of the capsule on the humeral side by the tendons of the rotator cuff, of the middle glenohumeral ligament by subscapularis, of the superior glenohumeral ligament by supraspinatus, and of the posterior capsule by infraspinatus and teres minor. This circular protective tendinous structure may be a reason why HAGL occurs less often in clinical cases of shoulder instability which contrasts with the frequency of failure on the humeral side of the ligaments in experimental loading studies (Bigliani et al., 1992; Gagey et al., 1993).

Another thought to consider is that the interval between subscapularis and latissimus dorsi (inferior interval) may have importance for anteroinferior stability similar to that of the rotator cuff interval (RCI) between subscapularis and supraspinatus. The RCI coincides with the superior glenohumeral and coracohumeral ligaments. The inferior interval may coincide with the anterior part of the inferior glenohumeral ligament and the fasciculus obliquus. The fasciculus obliquus is that neglected part of the anteroinferior capsuloligamentous structure that passes obliquely from the inferior part of the glenoid labrum and the long tendon of the triceps to the middle glenohumeral ligament and the subscapularis superolaterally. It was originally described by Delorme (1910) and recently has been rediscovered by Gohlke et al. (1994) and Kolts et al. (2001). A detailed anatomic study of this area with reference to the inferior muscular interval would be interesting.

We conclude that the latissimus dorsi may influence dislocation behaviour by blocking the humeral head in the limited group of patients with shoulder instability that have capsuloligamentous lesions on the humeral side. In this study, this was the case in one-third of the specimens even with a humerus-based circular section of all capsuloligamentous structures. This passive action of the latissimus dorsi may be a factor in occult (undiagnosed) dislocations and therefore in secondary chronic instability. In addition, the results from the present work help to stress the importance of musculotendinous structures around the shoulder in maintaining stability.

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Nicole Pouliart
Olivier Gagey

# The effect of isolated labrum resection on shoulder stability

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N. Pouliart (✉)
Department of Orthopaedics and Traumatology, Academic Hospital Vrije Universiteit Brussel, Laarbeeklaan 101, 1090 Brussels, Belgium
E-mail: izvptn@az.vub.ac.be
Tel.: +32-2-4776547

O. Gagey
Service d'Orthopédie, Hôpital Bicêtre, Université Paris-Sud, 78 Avenue du Général Leclerc, 94270 Le Kremlin-Bicêtre, France

O. Gagey
Institut d'Anatomie, 45 rue des Saint Pères, 75006 Paris, France

**Abstract** The present study was initiated to determine whether glenohumeral instability and dislocation can result from isolated lesions of the glenoid labrum in an arthroscopic cadaver model. Adjacent combinations of four zones of the labrum (superior, anterosuperior, anteroinferior and inferior) were sequentially removed with a motorised shaver, taking great care to leave the capsule intact in 24 cadaver shoulders. Stability was tested before and after inserting the scope and after each resection step. Inferior stability was examined by performing an inferior drawer test. Anterior stability was evaluated with an anteroposterior drawer test in 0° of abduction and with a load-and-shift test in external rotation and 90° abduction. Labral resection of all four zones maximally resulted in a grade 1 inferior instability (< 10 mm inferior translation). When two adjacent labral zones were resected, a grade 2 anterior drawer (> 10 mm anterior but no medial translation) was seen in 17% of the specimens. This was seen in one more specimen after the addition of a third zone. There were no differences in the stability of the load-and-shift test after any amount of labral resection. Total labral debridement increased inferior and anterior translation, but did not allow the humeral head to dislocate. The degree of stability in the cocked-arm position, which is the most prone to dislocation, is not altered. In patients, isolated labral tears, that is, without evidence of capsuloligamentous damage, can probably be safely debrided without risking glenohumeral instability to the point of dislocation. Nevertheless, anterior translation may significantly increase when two or more zones are resected.

**Keywords** Labral injury · Glenohumeral instability · Cadaver · Shoulder surgery · Arthroscopy

## Introduction

The glenoid labrum serves as an aid in anchoring the capsuloligamentous structures to the glenoid rim. Furthermore, the labrum increases glenohumeral stability by playing an important role in concavity-compression, generating negative intraarticular pressure and by a direct wedge-like blocking effect [11, 14, 16, 17, 19, 23, 24, 31, 47].

During arthroscopy, one often encounters fraying and even isolated tearing of the glenoid labrum. In recent years numerous clinical reports on these isolated labral lesions have been published. Most authors agree that labral lesions in stable shoulders occur predominantly in the superior half and that they can safely be debrided without risking instability [1–3, 9, 12, 22, 27, 28, 31, 35, 44, 47]. Lesions of the anteroinferior labrum, however, seem to be found more often in association with instability, but then they are usually accompanied by capsuloligamentous lesions [1, 12, 22, 31].

In the present study, (extensive) labral debridement was simulated by removing the glenoid labrum arthroscopically while leaving the capsuloligamentous structures attached to the glenoid neck. This simulation was undertaken to test our hypothesis that the debridement of isolated labral lesions, and the extrapolation of isolated labral damage, that is, without Bankart or other capsuloligamentous lesion, is not sufficient to allow the humeral head to dislocate.

## Methods

Sequential arthroscopic resection of the labrum was performed and stability testing was done in 24 fresh cadaver shoulders, aged 73–97 at the time of death. These were all shoulders that showed no signs of previous shoulder surgery, that had at least 90° of glenohumeral exorotation in 90° of abduction, and that showed neither glenohumeral intraarticular pathology nor rotator cuff lesions on arthroscopic examination. No soft tissues were removed around the shoulder. The standard posterior and anterior portals were used, avoiding any additional damage to the surrounding structures. The labrum was divided into five zones (Fig. 1): zone SG, superior (11.00–1.00 p.m.); zone MG, anterosuperior (1.00–3.00 p.m.); zone AB, anteroinferior (3.00–5.00 p.m.); zone PB, inferior (5.00–8.00 p.m.) and posterior (8.00–11.00 p.m.). Four of these zones (SG–MG–AB–PB) were sequentially removed with a motorised shaver, taking great care to leave the capsule intact. Because we were interested in testing the influence of labral debridement on anteroinferior stability, we did not resect the posterior zone. Various combinations of adjacent zones were resected to simulate more or less extensive debridement (Table 1).

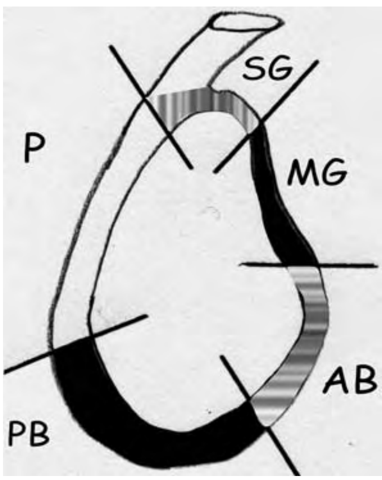


**Fig. 1** Schematic diagram showing the resected zones. *PB* zone including posterior band of inferior glenohumeral ligament, *AB* zone including anterior band of inferior glenohumeral ligament, *MG* zone including middle glenohumeral ligament, *SG* zone including superior glenohumeral ligament, *post* zone of posterior capsule

Glenohumeral stability was tested and graded before and after inserting the scope and after resecting any zone. Inferior stability was evaluated by an inferior drawer test (sulcus sign). Anterior stability was evaluated by an anteroposterior drawer test in 0° of abduction and neutral rotation (drawer sign), as well as by a load-and-shift fulcrum test in the apprehension position of 90° of external rotation and 90° of abduction (LAS). First, the humeral head was centred on the glenoid by applying a 50 N axial compression force and then a 50 N anteriorly (or inferiorly for the sulcus test) directed translation force was applied with a handheld dynamometer (MicroFET2, Hoggan HealthIndustries, distributed by Biometrics Europe BV, 1322 AD Almere, The Netherlands). The stability was graded on the basis of measurements obtained with an electromagnetic tracking device (EMTD, “Flock of Birds”, Ascencion Technology, Burlington, VT 05402, USA). One sensor was rigidly fixed on the acromion with threaded steel pins and the second sensor was rigidly fixed to the humeral head (Fig. 2). The values of medial, anterior and inferior translation recorded with the EMTD were then translated into a grading system where Grade 0 represents no translation, Grade 1 represents less than 10 mm of translation (inferior or anterior), Grade 2 (subluxation) encompasses more than 10 mm of inferior or anterior translation and Grade 3 corresponds with dislocation and has more than 10 mm of translation (inferior or anterior) in combination with medial translation [38]. In addition, maximal glenohumeral abduction (hyperabduction test or HAT) and maximal external rotation in 90° of glenohumeral abduction were measured (ABERmax, hyperrotation test). The HAT is a validated measure of inferior instability [7]. External rotation in abduction is known to increase in the dominant arm of overhead athletes [25, 40, 41], but has not been systematically evaluated in experimental instability studies.

On completion of the testing procedure, all shoulders were dissected to verify that the capsuloligamentous structures were effectively still intact and attached to the glenoid.

### Discussion of methodology

We used 50 N for the applied loads as this was found to be the maximum force tolerated for reproducible

**Table 1** Overview of the sequence and combinations of the resected zones

| One zone resected | Number of specimens | Two zones resected | Number of specimens | Three zones resected | Number of specimens | Four zones resected | Number of specimens |
|---|---|---|---|---|---|---|---|
| PB | 4 | PB–AB | 4 | PB–AB–MG | 4 | PB–AB–MG–SG | 4 |
| AB | 8 | AB–PB | 4 | AB–PB–MG | 4 | AB–PB–MG–SG | 4 |
| | | AB–MG | 4 | AB–MG–PB | 4 | AB–MG–PB–SG | 4 |
| MG | 8 | MG–AB | 4 | MG–AB–SG | 4 | MG–AB–SG–PB | 4 |
| | | MG–SG | 4 | MG–SG–AB | 4 | MG–SG–AB–PB | 4 |
| SG | 4 | SG–MG | 4 | SG–MG–AB | 4 | SG–MG–AB–PB | 4 |
| Total | 24 | | 24 | | 24 | | 24 |

*PB* inferior zone, *AB* anteroinferior zone, *MG* anterosuperior zone, *SG* superior zone

stability testing in cadavers of similar age [43]. We applied the translatory forces manually, which makes them less standardised. Although it reproduces the clinical testing method, this is another limitation of our experimental setup in comparison with forces applied by a servohydraulic actuator. The use of the "Flock of Birds" electromagnetic tacking device for instability testing was described by Tibone et al. [45] in a clinical setting and on cadavers by Reis et al. [37]. In the clinical situation translation values are difficult to obtain accurately and shoulders are usually tested for instability by drawer sign, apprehension and load-and-shift-type tests ([10, 20]: Chap. 2). In addition, Hawkins et al. [18] and Harryman et al. [15] already warned that increased translation in itself does not equal instability. A wide range of values for anterior, posterior and inferior translation can be found in normal, stable shoulders and several studies have demonstrated a large overlap of this range with that recorded in unstable shoulders [18, 26, 40, 41]. To correspond more closely with the clinical setting, we opted to use the same type of tests in our study and consequently also apply the grading of instability that was adopted by the American Shoulder and Elbow Society [38]. Several authors have used this grading system to evaluate normal and unstable shoulders [5, 25, 32, 46]. For anterior translation, Tillander and Norlin [46] have shown that the reproducibility of this grading and its correlation with instrumented measurement of anterior translation is very high. The drawback of this approach is that the grading is only semi-quantitative and makes statistical analysis less straightforward. As the grading is an ordinal categorical variable, non-parametric tests should be used, although Oliashirazi et al. [32] and Faber et al. [5] analysed their results as if grading was a normally distributed scale variable.

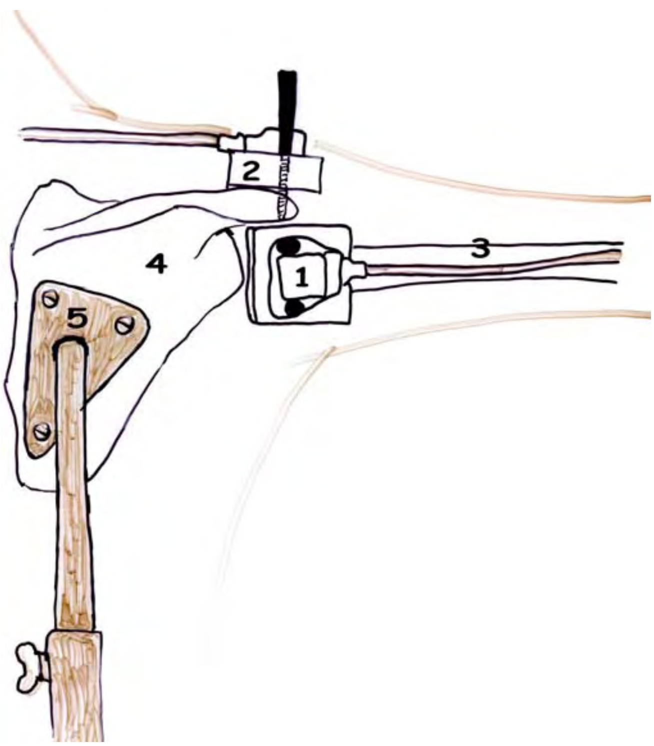


**Fig. 2** Schematic drawing of the experimental set up for a right shoulder seen from the back. *1* Master sensor of electromagnetic tracking device fixed to superolateral aspect of humerus with the aid of a custom- made block and threaded stainless steel pins, *2* slave sensor fixed to the top of the acromion, *3* humerus, *4* scapula, *5* custom-made stainless steel device for rigidly fixing the scapula

## Statistical analysis

The sample size needed for a power of 90% with $\alpha = 0.05$, supposing that an additional zone resulted in a mean difference of 1 grade with a standard deviation of 1 grade, was estimated at 23. This estimate is only valid for differences between the number of zones resected. Therefore, comparisons when grouping per sequence will not reach sufficient power as there are four or maximally eight specimens per sequence. Although it may seem interesting to correct for the influence of a specific order in the sequence of ligamentous zones that were cut, the limited number of specimens per ordered sequence actually precluded a valid statistical analysis.

Non-parametric tests (SPSS for Windows, version 11.5, SPSS Belux, 1020 Brussels, Belgium) were used for statistical analysis because grading results in an ordered categorical variable and because HAT and ABERmax are not distributed normally and do not have homogeneity of variance. Spearman's rho was used to test the correlation between HAT, ABERmax, degree of sulcus sign, degree of drawer sign, LAS and amount of resected zones. Pearson's chi-square test (for sulcus sign, degree of drawer sign and LAS) and the Jonckheere–Terpstra test (for HAT and ABERmax) were used to test the amount of independence in the function of the resected zones. The Jonckheere–Terpstra test is a variant of the Kruskal–Wallis $H$ test, which is more powerful when there is a natural priori ordering of the $k$ populations (ascending or descending) from which the samples are drawn.

## Results

### Resection

In seven specimens, the labral resection was very easily accomplished on the anterior part of the glenoid, from 11.00 to 5.00 p.m. for a right shoulder, because the capsule was inserted directly onto the glenoid neck immediately medial to the labrum itself. In all the other specimens, the resection needed to be done carefully in the more medial layers of the labrum so as not to damage the attached capsule. However, even in these specimens, the capsule seemed to continue onto the glenoid neck on the medial aspect of the labrum. In the inferior third, the capsule was continuous with the labrum and this made it difficult to resect the labrum. Nevertheless, it was possible to remove the majority of the fibrous structure of the labrum leaving a capsular layer. The open dissection of all specimens confirmed that the capsuloligamentous structure was not inadvertently breached or detached from the glenoid at any location. Figure 3 illustrates an arthroscopic labral resection.

### *Inferior instability: sulcus sign*

Resection of all the zones of the labrum resulted in no more than grade 1 sulcus sign. One specimen demonstrated grade 1 sulcus sign after introducing the scope, which has a venting effect. After the full resection of the labrum, the degree of sulcus sign in this specimen remained at 1. In two shoulders, grade 1 sulcus sign was seen after resecting the anteroinferior labrum. In one shoulder, grade 1 sulcus sign was seen after resecting the anterosuperior zone. Another specimen demonstrated grade 1 sulcus sign after resection of three zones (inferior to anterosuperior).

The differences in the degree of sulcus sign in function of the amount of resected zones were not significant (Pearson's chi-square, $P > 0.05$).

The correlation (Spearman's rho) of the degree of sulcus sign was:

- high for HAT (0.770, $P < 0.0005$) and for degree of drawer sign (0.641, $P < 0.0005$);
- intermediate for LAS (0.481, $P < 0.0005$); and
- low for ABERmax (0.210, $P = 0.021$).

### *Anterior instability: drawer test*

Two shoulders demonstrated grade 1 drawer sign after venting. This progressed to grade 2 drawer sign after

**Fig. 3** Arthroscopic images illustrating the progression of labral resection with a motorised shaver

resecting both the anterior zones in one specimen and after resecting three zones (inferior to anterosuperior) in the other specimen. In total, eight specimens had grade 1 drawer sign after resecting either anterior zone. Therefore, resection of the inferior or superior zone only did not result in a drawer sign. In eight shoulders, both the inferior zones were resected. This resulted in grade 1 drawer sign in one of these eight shoulders and in grade 2 drawer sign in two of them. In the eight specimens with both the anterior zones resected, three demonstrated grade 1 drawer sign and one had grade 2. The same distribution was seen for the eight specimens where both superior zones were resected. One specimen moved from grade 1 to grade 2 drawer sign when the anterosuperior labrum was resected in addition to both the inferior zones. No changes in the degree of drawer sign were seen after the addition of a fourth zone.

The differences in the degree of sulcus sign for any increase in the amount of resected zones were not significant (Pearson's chi-square, $P > 0.05$).

The correlation (Spearman's rho) of the degree of drawer sign was:

- high for HAT (0.646, $P < 0.0005$) and for degree of sulcus sign (0.691, $P < 0.0005$);
- intermediate for ABERmax (0.451, $P < 0.0005$); and
- low for LAS (0.277, $P = 0.002$) and amount of resected zones (0.270, $P = 0.003$).

### *Anterior instability: load-and-shift test*

One specimen had a grade 1 instability with the LAS-test even before venting and this did not increase with the subsequent labral resection. In all the other shoulders, no amount of labral resection had an influence on the degree of instability.

The differences in the degree of instability for any increase in the amount of resected zones were not significant (Pearson's chi-square, $P > 0.05$).

Spearman's rho showed that the degree of instability had a low correlation with the other tested parameters that only reached significance for the degree of sulcus sign (0.481, $P < 0.0005$) and the degree of drawer sign (0.277, $P = 0.002$).

### Hyperabduction and hyperrotation test

The HAT increased to a maximum of 100° in four shoulders. All the other specimens remained at 90°. The differences in HAT in function of the amount of resected zones were not significant (Pearson's chi-square, $P > 0.05$).

With the labrum still intact, 22 of the 24 specimens had an ABERmax of 90°. After resecting one zone, only five shoulders retained an ABERmax of 90°. The majority had an ABERmax of 100°, with only one specimen at 110°. After resecting two zones, the majority was still at 100°, with four shoulders at 90° and three at 110°. Resecting three zones led to an ABERmax at 90° in two specimens, at 100° in 15 and at 110° in seven. Resecting all four zones showed an ABERmax at 100° in 13 shoulders and at 110° in the other 11. The differences in hyperrotation for an increase in the amount of resected zones were significant (Pearson's chi-square, $P < 0.0005$). In addition, the correlation (Spearman's rho) between ABERmax and the amount of resected zones was high (0.660, $P < 0.0005$).

## Discussion

The labrum can be considered from two viewpoints. However, a discussion of the anatomy and biomechanics of the labrum is beyond the scope of this paper. We will focus on the published biomechanical studies of labral lesions and on the published clinical studies on the treatment of labral lesions.

Labral injuries have been implied to contribute to shoulder instability since Bankart's first description of the anteroinferior capsulolabral detachment as the quintessential lesion in 1923 [4].

Due to the type of surgical procedures available and also due to the limited array of imaging procedures, it used to be difficult if not impossible to distinguish purely labral lesions from capsulolabral lesions. Only since the advent of arthroscopy, surgeons have been able to differentiate and catalogue these lesions more accurately. When reviewing the recent literature on (the arthroscopic treatment of) labral lesions [1, 3, 12, 22, 24, 27, 28, 31, 33–35, 42, 44, 47], the following conclusions can be drawn. Most authors agree that many labral lesions, especially fissuring, partial detachment and ossification, occur frequently, even in asymptomatic patients, and can often be considered as degenerative changes. In the cadaver part of his work, Kohn [22] discovered that labral lesions existed as early as the second or third decade. Snyder et al. [42] grouped and classified lesions to the superior labrum under the heading of SLAP (superior labrum anterior to posterior). Anterosuperior or superior lesions were rarely accompanied by capsuloligamentous lesions [1–3, 9, 12, 22, 27, 28, 31, 35, 44, 47]. As anterosuperior or superior labral lesions were usually found in patients that had stable shoulders, many authors believe that superior labral lesions contribute little to clinical instability. Most patients with anterosuperior or superior lesions complained of painful shoulders, regularly but not always associated with clicking or locking. These patients rarely felt subluxation or dislocation of their shoulder. Their shoulders were usually stable, even when examined under anaesthesia. Pappas et al. [34] called the phenomenon of clicking, catching or locking of an anatomically stable glenohumeral joint functional instability. When these isolated labral lesions were treated by debridement, results generally varied from good to excellent. Labral debridement in itself did not result in increases in instability. In the rarer instances when superior and anterosuperior lesions did occur in association with instability and capsuloligamentous lesions, Kohn [22] only addressed the latter lesions and left the labral lesions untreated with a good outcome.

Anteroinferior lesions, on the other hand, were frequently found in association with capsuloligamentous lesions and overt instability [1, 12, 22, 31]. In this situation, isolated labral debridement usually gave fair to poor results. When the labral tears were treated together with the capsuloligamentous lesion, the results were better. Apparently, isolated anteroinferior labral injury was found by Rowe and Zarins in patients with the "dead arm syndrome" [39]. Protzman [36], Garth and Allman [8] and McGlynn and Caspari [30] have also observed these lesions in patients with occult or transient subluxation. In these patients, there seems to be a subtle form of instability.

Some experimental studies on labral lesions have been published. Pagnani et al. [33] concluded from their cadaver study in nine specimens that creating an isolated superior labral avulsion increased anterior translation by about 6 mm and inferior translation by about 2 mm, whereas an isolated anterosuperior labral avulsion did not affect translation. These values are relatively low and may remain within the normal variation of inferior and anterior translation observed in asymptomatic shoulders [18, 40, 41]. In addition, this study tells us nothing about the dislocating potential of anterosuperior and complete superior lesions. Lazarus et al. [24] removed the anteroinferior portion of the labrum and the adjacent part of the glenoid cartilage. The anteroinferior stability ratio was decreased by 65% and all five shoulders demonstrated dislocation on maximum translation force after minimal anterior translation of 1.4 mm (SD 1.2 mm) and anteroinferior translation of 3.2 mm (SD 3.3 mm). Klein et al. [21] studied the influence of Bankart lesions on glenohumeral stability in six shoulders. Strain in the inferior glenohumeral ligament and torsional resistance decreased with the increasing depth of the created lesion. Speer et al. [43] also evaluated a simulated Bankart lesion in nine cadaver shoulders and observed very small increases (maximally 3.4 mm) in anterior, posterior and inferior translation. Fehringer et al. [6] noted a shift of the humeral head upon a centring compressive force in the direction of the subperiosteal labral dissection created in five shoulders. Removal of the anteroinferior labrum resulted in an increased glenohumeral contact area and pressure in eight specimens studied by Greis et al. [13]. These studies, however, did not investigate labrum-only lesions as the capsule was detached as well or some cartilage was also removed. Therefore, to the best of our knowledge, there seems to be no experimental study on isolated labral lesions, that is without associated capsuloligamentous lesions. This apparent lack of cadaver studies on isolated labral lesions led us to conduct the present study. Although isolated inferior and anteroinferior labral tears are observed in relatively few patients, we included simulation of debridement of these lesions in our protocol to obtain a more complete picture. Almost all experimental cadaver studies have the inherent limitation that the age group studied does not closely correspond with the typical age distribution of patients with instability, be it overt or functional. However, degenerative lesions of the labrum that may require debridement are more frequent with increasing age. Another limitation of the cadaver experiments is that these only allow studying the passive stabilisers and cannot account for the influence of dynamic stabilisers, more specifically muscular contraction.

Excising the labrum has been shown to reduce the effectiveness of concavity-compression in resisting a translatory force by 20–65% [24, 29]. In other studies, negative intraarticular pressure was seen to diminish when capsulolabral or rotator cuff lesions are present [11, 17, 23]. In this study, venting only (introduction of the arthroscope) had an effect on the inferior grade of instability (sulcus sign) in only one specimen and on the grade of drawer sign in only two shoulders. In all other shoulders, no difference in grading solely due to venting was seen. Therefore, the elimination of negative intraarticular pressure may increase translation as a result of applied forces and diminish the force required to obtain a specific displacement, but by itself does not lead to an appreciable degree of instability on clinical testing manoeuvres.

Capsuloligamentous and labral lesions are predominantly found in the anteroinferior zone in the clinical series of instability, which correlates with the blocking wedge theory proposed by Howell and Galinat [19]. After resecting one labral zone (anteroinferior or inferior for sulcus sign, anteroinferior or anterosuperior for drawer sign), testing in the direction of the resected zone resulted in a decrease in the inferior stability in 2 shoulders (out of 7) and in a decrease in anterior stability in 7 shoulders (out of 16). The same proportion of shoulders had increases in drawer sign after resection of either the anteroinferior or the anterosuperior labral zone. Combination of one of these zones with the other zones increased the degree of sulcus sign in 1 shoulder and the degree of drawer sign in 7. These results confirm that the labrum can have a stabilising affect and that labral loss results in increased anterior and inferior translation in neutral rotation and 0° of abduction. Minimal or no differences were observed in 80% of the shoulders for inferior translation and in 55% of the shoulders for anterior translation. Hyperabduction was not observed, regardless of the extent of labral resection (maximal increase to 100°). This result is another confirmation of the retained inferior stability. Therefore, labral resection with intact capsuloligamentous structures does not seem to have important consequences as far as clinically detectable instability is concerned. In addition, no changes in degree of instability in the cocked-arm position were observed. Therefore, the chock-block effect of the labrum and its relevance for concavity-compression do not seem to be major contributing factors for stability in 90° of external rotation with 90° of abduction. As our study aimed to preserve capsule and ligament, we would like to suggest that the retained stability was assured by these structures. This corresponds with the hypothesis that capsuloligamentous tension is the more important static stabiliser in this position.

The present work leads us to conclude that even complete resection of the inferior or the superior labrum influences shoulder stability only to a minor extent. Anterior labral resection leads to an increase of anterior translation in about half of the specimens, whereas inferior labral resection leads to an increase of inferior translation in about a fifth of specimens. Neither subluxation nor dislocation in the cocked-arm position was observed. This confirms our hypothesis about the consequences of isolated labral lesions for shoulder stability.

In patients with symptoms due to labral tears, including those patients suffering from functional instability (but not subluxation or dislocation), these tears can probably be debrided without consequences for glenohumeral stability. This study left the capsuloligamentous structures intact. Therefore, no conclusions should be drawn from this work concerning labral tears in association with capsuloligamentous lesions. We would like to stress that a diligent search for clinical signs of subtle instability, including examination under anaesthesia, as well as a thorough inspection for associated glenohumeral ligament laxity and lesions should be performed in all cases before proceeding to an isolated labral debridement. Tests such as the apprehension test with relocation test (anterior) and the HAT (inferior) can help in discerning cases with subtle instability.

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# Simulated Capsulolabral Lesion in Cadavers: Dislocation Does Not Result From a Bankart Lesion Only

Nicole Pouliart, M.D., Ph.D., Simon Marmor, M.D., and Olivier Gagey, M.D., Ph.D.

**Purpose:** Although an anteroinferior capsulolabral detachment (typical Bankart lesion) has been evaluated in other experimental studies, it has not yet been tested with an apprehension test in an intact shoulder model. **Methods:** Adjacent combinations of 4 zones of the capsuloligamentous complex were sequentially detached from the glenoid neck in 50 cadaveric shoulders. Stability was tested before and after each resection step: inferior stability with a sulcus test and anterior stability with an anterior drawer test and with a load-and-shift test in the apprehension position. **Results:** A metastable anteroinferior dislocation occurred in 18 specimens after section of 3 zones and in 14 only after section of 4 zones. A locked dislocation occurred after section of all 4 zones in 33 specimens and in the other 17 shoulders only after the posterior capsule was also cut. **Conclusions:** The humeral head cannot dislocate anteroinferiorly when there only is a Bankart lesion. In our study superior and posterior extension was necessary before the tensioning mechanism in external rotation and abduction failed enough for dislocation to occur. **Clinical Relevance:** Because the Bankart lesion is most likely not the only lesion present in patients with recurrent dislocation, a careful search for other lesions needs to be done when one is attempting surgical treatment. These lesions would need to be treated as well if one wants to avoid the risk of residual instability. **Key Words:** Bankart lesion—Capsulolabral lesion—Glenohumeral instability—Shoulder—Cadaver—Arthroscopy.

Since the description of an anteroinferior glenohumeral capsulolabral detachment in recurrent shoulder dislocation by Bankart,[1,2] this has been regarded as the quintessential lesion in instability. Although the Bankart lesion is indeed frequently observed in chronic instability, as well as in primary dislocations, some patients do not have this type of lesion. Whether the Bankart lesion in itself is responsible for recurrences remains debatable. Several experimental studies have evaluated the consequences of this type of lesion for shoulder stability.[3-10] Most, however, use a cadaveric model in which only the joint and its capsule are retained. The removal of the muscular envelope may influence the obtained results because the totality of passive restraints is altered. In addition, in almost all of these studies, instability is evaluated on the basis of increased translation or increased mobility, which is closely linked to but does not equal the ability to dislocate.

The consequences of a Bankart lesion have not yet been tested with an apprehension maneuver in an intact shoulder model. We used this type of model to test our hypothesis that more extensive capsulolabral lesions than an isolated Bankart lesion are necessary to allow the humeral head to dislocate.

## METHODS

Sequential arthroscopic capsulolabral detachment was performed and stability testing was done in 50 fresh nonembalmed cadaveric shoulders, aged 49 to 103 years (mean, 75.5 ± 12.8 years) at the time of

*From the Department of Orthopaedics and Traumatology, Academic Hospital Vrije Universiteit Brussel (N.P.), Brussels, Belgium; the Department of Human Anatomy, Vrije Universiteit Brussel (N.P.), Brussels, Belgium; the Service d'Orthopédie, Hôpital Bicêtre, Université Paris-Sud (S.M., O.G.), le Kremlin-Bicêtre, France; and the Institut d'Anatomie (O.G.), Paris, France.*

*Address correspondence and reprint requests to Nicole Pouliart, M.D., Ph.D., Department of Orthopaedics and Traumatology, Academic Hospital Vrije Universiteit Brussel, Laarbeeklaan 101, 1090 Brussels, Belgium. E-mail: izvptn@az.vub.ac.be*



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death. Shoulders that showed signs of previous shoulder surgery or of glenohumeral intra-articular disease on arthroscopic examination were discarded. In addition, only specimens that reached at least 130° of elevation and 90° of glenohumeral abduction with 90° of glenohumeral exorotation in 90° of glenohumeral abduction were included. The specimens consisted of intact and complete upper limbs amputated through the scapulothoracic and sternoclavicular joints. No soft tissues were removed around the shoulder. The scapula of each specimen was rigidly fixed on a custom-made device (Fig 1). Standard posterior and anterior arthroscopic portals were used, avoiding any additional damage to the surrounding structures. The capsulolabral complex was divided into 5 zones corresponding to the classic ligamentous areas of the glenohumeral capsule (Fig 2). In all shoulders 4 of these zones were cut with an arthroscopic knife so that the labrum with the capsuloligamentous structures of the corresponding zone was fully detached along the circumference of the glenoid rim and neck (Fig 3). Various combinations of adjacent zones were resected to enable the influence of the Bankart type of lesion and more extensive lesions to be determined. Power calculation indicated a required sample size of 46 for comparing differences in the number of zones cut and of 14 for comparing differences between various com-

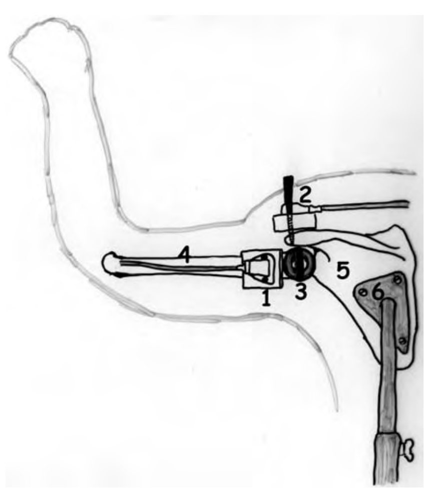


**Figure 1.** Experimental setup for a left shoulder seen from the back in 90° of external rotation and 90° of glenohumeral abduction. (1, master sensor of electromagnetic tracking device fixed to superolateral aspect of humerus with the aid of a custom-made block and threaded stainless steel pins; 2, slave sensor fixed to top of acromion; 3, handheld dynamometer applied to posterior aspect of humeral head during anterior stability testing; 4, humerus; 5, scapula with medial border vertical when testing; 6, custom-made stainless steel device for rigidly fixing the scapula.)

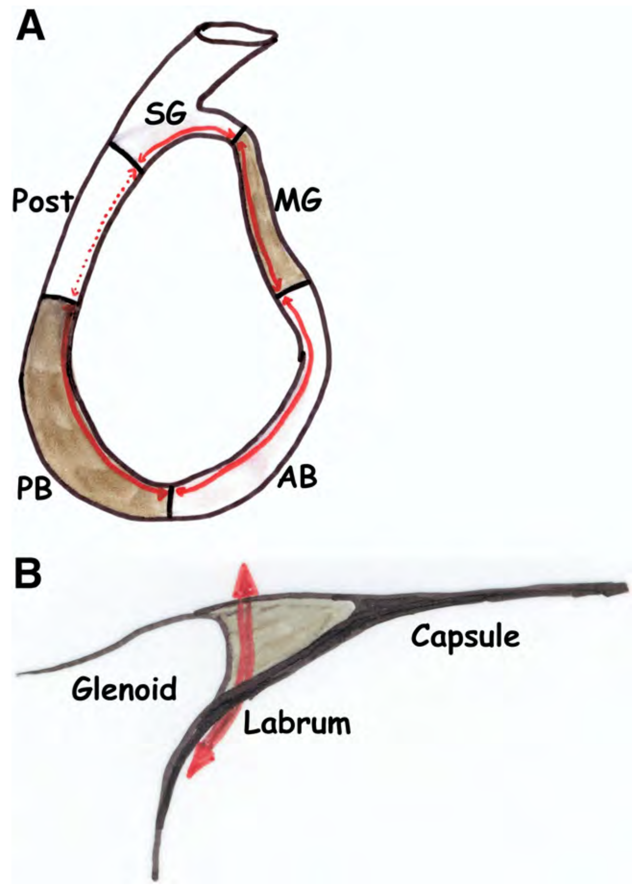


**Figure 2.** (A) Division of the labrum into 5 zones for a right shoulder and the number of specimens allocated to each cutting sequence. The arrows indicate the sagittal location of the capsulolabral cut within each zone. The tangential marks within the labrum indicate where the continuity of the labrum was disrupted between each capsulolabral zone. (PB, posteroinferior zone from 9 to 6 o'clock; AB, anteroinferior zone from 6 to 3 o'clock; MG, anterior zone from 3 to 1 o'clock; SG, superior zone from 1 to 11 o'clock; Post, posterior zone from 11 to 9 o'clock.) (B) Transverse location of the capsulolabral cut close to the glenoid neck.

binations within the same number of zones cut. Therefore the shoulders were allocated to a specific sequence of cut zones so that there were at least 15 specimens in each combination from 2 zones cut onward (Table 1). Itoi et al.[9] found that a simulated Bankart lesion only resulted in an unstable shoulder when the continuity of the labrum was disrupted as well. Therefore we decided to break the continuity of the labral ring by cutting the labrum, but not the capsule, tangentially between each zone in addition to the circular labral detachment (Fig 3).

The testing method for instability has been described in detail elsewhere.[11,12] In summary, medial, anterior, and inferior translation resulting from an

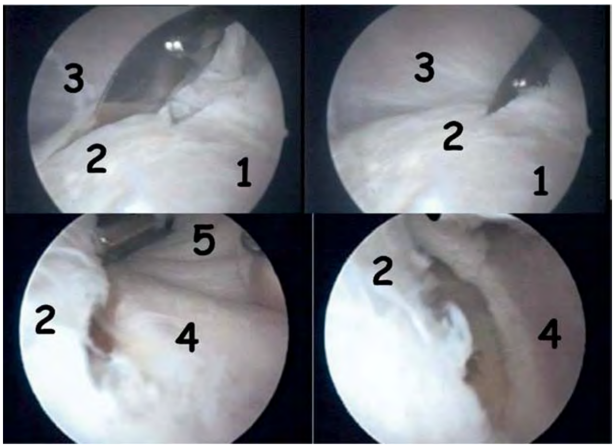


**FIGURE 3.** Arthroscopic images illustrating creation of posteroinferior (top) and anteroinferior (bottom) capsulolabral cut with curved knife. (1, glenoid fossa; 2, labrum; 3, posterior band of IGHL [posteroinferior zone]; 4, anterior band of IGHL [anteroinferior zone]; 5, middle glenohumeral ligament [anterior zone].)

inferior sulcus test, an anterior drawer test in 0° of abduction and neutral rotation, and a fulcrum test in 90° of external rotation and 90° of abduction (load-and-shift [LAS] test) was recorded with an electromagnetic tracking device (Flock of Birds; Ascension Technology, Burlington, VT). These measurements were used to grade the resulting instability on a modified American Shoulder and Elbow Surgeons scale.[13] This modification subdivides dislocation into metastable (grade 3) and locked (grade 4) dislocation[11,12,14] (Table 2). If grade 4 instability did not result from cutting the 4 ligamentous zones, the posterior labrum was also detached. On completion of the testing procedure, all shoulders were dissected to verify that the capsuloligamentous structures were completely detached from the glenoid rim and neck.

Because grading results in a categoric variable, nonparametric tests were used for statistical analysis (except for power estimation). Pearson $\chi^2$ was used for group comparisons and Spearman $\rho$ for correlation analysis (SPSS for Windows, version 11.5; SPSS Belux, Brussels, Belgium).

## RESULTS

Insertion of the arthroscope, which has a venting effect, did not change anterior stability during the LAS test to an appreciable degree. Venting resulted in increases in anterior and inferior translation, with 18 shoulders having a grade 1 drawer sign and 19 specimens having a grade 1 sulcus sign. Two shoulders even had a grade 2 drawer sign and one had a grade 2 sulcus sign after venting. For each additional zone cut, there was a progressive increase in the number of specimens with a specific degree of instability in the apprehension position (Fig 4). Dislocation, metastable (grade 3) or locked (grade 4), during the LAS test was only seen after detachment of at least 3 capsulolabral zones. Of the specimens, 34% even required cutting of the posterior capsule before locked dislocation would occur. In the drawer test (Fig 5) and sulcus test (Fig 6), we also observed a progressive increase in anterior and inferior translation, respectively. However, no specimens dislocated inferiorly after any number of zones were cut, and only 3 specimens reached dislocation, either metastable (2) or locked (1), during the anterior drawer test.

The differences in the degree of instability between each step were significant (Pearson $\chi^2$, $P < .001$). The differences between the various combinations of zones cut within the same step were not significant ($P > .05$) except within 3 zones cut. Here the degree of instability from the combination of the superior to anteroinferior zone was less statistically significant

**TABLE 1.** *Overview of Capsulolabral Zone Combinations Planned and Actually Cut*

| One Zone Cut | No. of Specimens | Two Zones Cut | No. of Specimens | Three Zones Cut | No. of Specimens | Four Zones Cut | No. of Specimens | Posterior Zone Cut (No. of Specimens) |
|---|---|---|---|---|---|---|---|---|
| PB | 10 | PB-AB | 10 | PB-AB-MG | 10 | PB-AB-MG-SG | 10 | 5 |
| AB | 15 | AB-PB | 5 | AB-PB-MG | 5 | AB-PB-MG-SG | 5 | 2 |
| | | AB-MG | 10 | AB-MG-SG | 5 | AB-MG-SG-PB | 5 | 1 |
| | | | | AB-MG-PB | 5 | AB-MG-PB-SG | 5 | 2 |
| MG | 15 | MG-AB | 10 | MG-AB-SG | 5 | MG-AB-SG-PB | 5 | 1 |
| | | | | MG-AB-PB | 5 | MG-AB-PB-SG | 5 | 0 |
| | | MG-SG | 5 | MG-SG-AB | 5 | MG-SG-AB-PB | 5 | 2 |
| SG | 10 | SG-MG | 10 | SG-MG-AB | 10 | SG-MG-AB-PB | 10 | 4 |
| Total | 50 | Total | 50 | Total | 50 | Total | 50 | 17 |

NOTE. PB, inferior zone; AB, anteroinferior zone; MG, anterosuperior zone; SG, superior zone.

**TABLE 2.** *Scale for Grading Anterior Instability*

| Grade | Translation | | | Reduction Upon Release of Anterior Force |
|---|---|---|---|---|
| | Anterior | Inferior | Medial | |
| 0: Stable | None | No | No | Yes |
| 1: Increased translation | <10 mm | No | No | Yes |
| 2: Subluxation | >10 mm | Yes but <10 mm | No | Yes |
| 3: Metastable dislocation | >10 mm | Yes | Yes | Yes |
| 4: Locked dislocation | >10 mm | Yes | Yes | No |

NOTE. For inferior instability, the values for anterior and inferior translation are switched.

than the degree of instability resulting from the combination of the anterior to posteroinferior zone ($P = .019$). The order in which the various zones were cut did not seem to have a statistically significant effect, although these groups were too small to be able to draw definitive statistical conclusions. The degree of instability had a very high correlation with the number of zones cut (Spearman $\rho = 0.916$, $P < .001$) and a relatively high correlation with the degree of the sulcus sign (Spearman $\rho = 0.682$, $P < .001$) and degree of the drawer sign (Spearman $\rho = 0.750$, $P < .001$).

The differences in the degree of the drawer sign were significant (Pearson $\chi^2$, $P < .001$) between each step but were not significant (Pearson $\chi^2$, $P > .05$) between the various zone combinations within the same step. The degree of the drawer sign also had a relatively high correlation with the degree of the sulcus sign (Spearman $\rho = 0.704$, $P < .001$) and with the number of zones cut (Spearman $\rho = 0.706$, $P < .001$).

The differences in the degree of the sulcus sign were significant between each step (Pearson $\chi^2$, $P < .001$) but were not significant (Pearson $\chi^2$, $P > .05$) for the various zone combinations within the same step. The degree of the sulcus sign also had a relatively high correlation with the number of zones cut (Spearman $\rho = 0.726$, $P < .001$).

## DISCUSSION

We were able to confirm our hypothesis that a Bankart lesion, which was represented in this study by cutting of the anteroinferior zone only, is not sufficient to allow the humeral head to dislocate in the apprehension position.

Broca and Hartmann[15,16] were the first to associate an acute anteroinferior shoulder dislocation with a

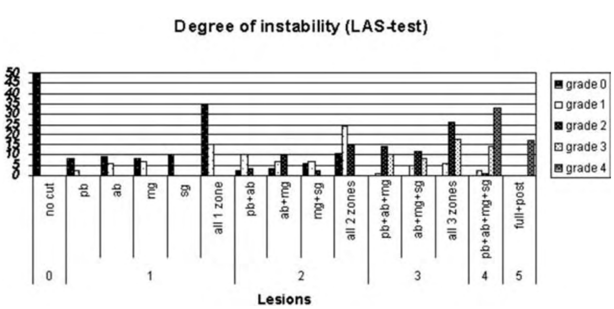


**FIGURE 4.** Degree of instability during the LAS test for each cutting sequence and for each step. Bars represent the number of specimens with a specific degree of instability for a specific combination of zones cut or for the total number of specimens with a specific degree of instability for each step, regardless of the specific combination of zones. With 1 zone cut, most specimens were still stable (grade 0). With 2 zones cut, most specimens had increased translation (grade 1). With 3 zones cut, most specimens had subluxation (grade 2) or a metastable dislocation (grade 3). After 4 zones were cut, most specimens displayed a dislocation, either metastable (grade 3) or locked (grade 4), although 17 specimens required the posterior zone to be cut as well. (pb, posteroinferior zone; ab, anteroinferior zone; mg, anterior zone; sg, superior zone; full+post, all 4 ligamentous zones and posterior zone.)

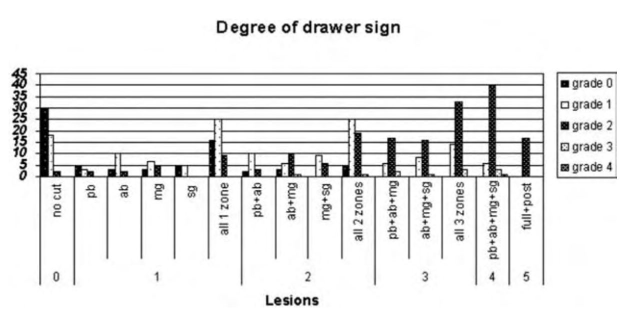


**FIGURE 5.** Degree of drawer sign for each cutting sequence and for each step. Bars represent the number of specimens with a specific degree of instability for a specific combination of zones cut or for the total number of specimens with a specific degree of instability for each step, regardless of the specific combination of zones. With each additional zone cut, there was an increase in the number of specimens with subluxation (grade 2), although few specimens had a dislocation, either metastable (grade 3) or locked (grade 4), even after all 4 zones were cut. (pb, posteroinferior zone; ab, anteroinferior zone; mg, anterior zone; sg, superior zone; full+post, all 4 ligamentous zones and posterior zone.)

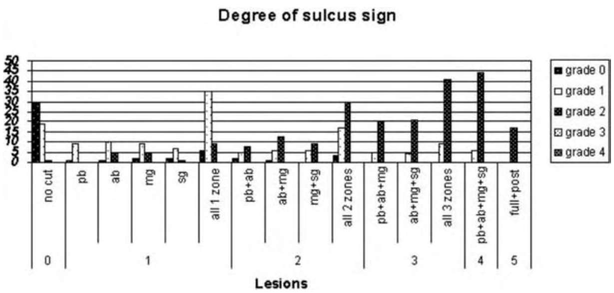


**FIGURE 6.** Degree of sulcus sign for each cutting sequence and for each step. Bars represent the number of specimens with a specific degree of instability for a specific combination of zones cut or for the total number of specimens with a specific degree of instability for each step, regardless of the specific combination of zones. Increased translation (grade 1) was seen in most specimens after 1 zone was cut. From 2 zones cut onward, most specimens had subluxation (grade 2), although no specimens had an inferior dislocation, either metastable (grade 3) or locked (grade 4). (pb, posteroinferior zone; ab, anteroinferior zone; mg, anterior zone; sg, superior zone; full+post, all 4 ligamentous zones and posterior zone.)

direct trauma mechanism resulting in a complete avulsion of the anterior capsule, including partial labral detachment and periosteal avulsion. To these authors, the humeral head could not dislocate without capsular lesions, seen with indirect trauma mechanisms, when there was no development of an anterior pouch as a result of periosteal detachment. A chronic or recurrent dislocation required even more lesions. Ever since Bankart[1,2] insisted that an anteroinferior capsulolabral detachment was the essential lesion for anterior shoulder instability, this type of lesion has carried his name. Since then, several clinical instability series have shown that 8% to 46% of shoulders did not have a Bankart lesion. Variants of the typical Bankart lesion include posterior or superior extension, glenoid avulsion fracture, and midsubstance ligament rupture.

Several experimental studies have shown that the inferior glenohumeral ligament (IGHL) is solicited most in a position of 90° of external rotation and 90° of abduction.[10,17-23] However, most of the Bankart lesion studies do not use this position for testing.[3,4,7-10] We concur with McMahon et al.[24-26] that anteroinferior instability should be tested in a simulated apprehension position. However, only these authors[25,26] and Stefko et al.[27] studied the IGHL in the apprehension position. In the clinical situation the arm is usually positioned in 90° of external rotation and 90° of abduction, because this is easily reproducible, and this corresponds to about 60° of glenohumeral abduction.[6] Because Gerber and Ganz[28] stated that the IGHL should be tested between 60° and 120° of total arm abduction, we used 90° of glenohumeral abduction in our manual testing protocol. According to the same authors, the middle glenohumeral ligament should be tested in 45° of total arm abduction. As yet, there is no validated test that unequivocally isolates the superior glenohumeral ligament; therefore we resorted to using the sulcus test. The manual testing method allows almost unconstrained movement of the humerus while dislocating and when the arm is returned to the neutral position of 0° of adduction and neutral rotation. In this setting it is possible to evaluate spontaneous reduction, and therefore we discerned 2 grades of full dislocation: metastable and locked[11,12,14] (Table 2). In specimens mounted in robotic actuators this freedom of movement does not seem possible.

In most of the simulated Bankart studies, specimens were stripped of all soft tissues, often including the rotator cuff muscles.[3-10] Removing the surrounding muscles, especially the rotator cuff, even with their forces simulated by cables and weights or machines, may alter the integrity of the passive restraints. At present, not enough is known about the interaction between passive and active stabilizers of the glenohumeral joint and about the passive (bulk) effect of these dynamic stabilizers to be able to rule out elimination of these muscles as a confounding factor in passive restraint studies.[29,30] Except in the article by Stefko et al.,[27] the IGHL has been studied as an isolated bone-ligament-bone complex. Our experimental setup does not allow appreciation of biomechanical properties and is not capable of evaluating dynamic effects, but it has an important advantage in that it uses otherwise intact shoulders, which does allow inclusion of the passive effect of the musculotendinous units surrounding the shoulder.

Specimens in this study only underwent dislocation, either metastable or locked, after at least 3 zones were cut. This seems to indicate that a solitary Bankart lesion—that is, only the anteroinferior and anterior zone—is not enough for an acute anterior dislocation. This corresponds with the results of Apreleva et al.[5] As already stated, our study does not evaluate capsular stretching as a possible cofactor, but a dislocation in this study was only possible when the capsulolabral detachment was more extensive. Posterior extension is one possibility, and this confers with the work of Harryman et al.,[31] who concluded that anteroinferior dislocation only occurred when the posteroinferior sling was also cut. A third of specimens in this arthroscopic model only had a locked dislocation when the posterior capsule was cut as well. In these latter cases we cannot exclude that they remained more or less stable because the posterior part of the IGHL complex was insufficiently cut. We tried to avoid this problem by cutting all the way up to the equator, but we cannot rule out the possibility that this posterior part inserts even higher up in some shoulders. Another scenario is that the circle concept, as described by several authors,[10,17,18,32,33] played a role in retaining the humeral head. This concept implies that an anterior dislocation entails not only an anterior capsular lesion but also a posterior lesion. A third option is that the intact muscular envelope and, more specifically, the tendons of the rotator cuff maintained enough passive tension to retain the humeral head.

Black et al.[6] noted that creating a Bankart-like lesion from the IGHL up to and including the superior glenohumeral ligament only resulted in a 3-mm increase in anterior translation in the apprehension position when compared with venting alone (mean, 19.6 mm *v* 16.3 mm). The amount of total translation in their study would be classified as grade 2 instability in our study. According to Gagey et al.,[34] a dislocation maneuver in cadaveric shoulders entailed a tear of the deep layer of the rotator cuff in 28 of 32 specimens. We believe that this deep layer actually corresponds to the superior capsular structures including the superior glenohumeral ligament. Gohlke et al.[35] observed that a pure simulated Bankart lesion resulted in a minimal change in anteroposterior translation, whereas there was significantly more translation when a horizontal extension into the capsule was made. These authors suggest that stability in external and internal rotation is induced by twisting of radially oriented fibers in the capsule. These fibers are also found in the inferior and middle glenohumeral ligaments but mainly appear in the fasciculus obliquus. In an isolated capsulolabral detachment of the Bankart type, as done in most studies including ours, these radially oriented fibers are not damaged. It would therefore seem possible that some degree of stability is retained as long as (part of) the fasciculus obliquus remains anchored to the glenoid rim. In addition, the fasciculus obliquus also inserts onto the tendon of the long head of the triceps. Given that this anchorage is not damaged in a Bankart type of lesion, this may be an additional reason why the resulting degree of instability in the apprehension position remains limited.

All of these findings indicate that, although it may be tempting to repair only the Bankart lesion when no other lesions are present at first sight, this approach induces the risk of residual instability. All components playing a role in the development of chronic instability need to be addressed. It is possible that the poor results of arthroscopic Bankart repairs in the pioneering days are at least in part due to the fact that these additional components were not addressed.

## CONCLUSIONS

This study indicates that the humeral head can only dislocate anteroinferiorly in the apprehension position when isolated capsulolabral detachment from the glenoid rim and neck is very extensive. An isolated typical Bankart lesion is not extensive enough. In acute dislocation, other lesions—posterior or superior extension, capsular stretching, midsubstance tears, glenoid rim fractures, or rotator cuff tears—need to be present as well.

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# Simulated humeral avulsion of the glenohumeral ligaments: A new instability model

Nicole Pouliart, MD, PhD,[a] and Olivier Gagey, MD, PhD,[b] *Brussels, Belgium*

*Humeral avulsion of the glenohumeral ligaments (HAGL) is an infrequent cause of shoulder instability. Experimental studies on this lesion are rare. This study was undertaken to determine the extent of humeral-based capsuloligamentous damage required for dislocation to occur. In 65 fresh cadaver shoulders, a humeral-sided ligamentous cutting sequence was done. After each step, degree of sulcus, translation, and instability were evaluated with an electromagnetic tracking device. There was a high degree of correlation between the amount of cut done and the resulting degree of instability. The order of the ligamentous cuts had no significant influence. For a dislocation to occur at least 3 zones had to be cut. Simulated HAGL can be used as a model for shoulder instability, although further experiments are needed to validate this model fully. Extensive capsuloligamentous lesions on the humeral side seem to be required before dislocation can occur. This may be a factor explaining the relative paucity of HAGL in clinical series. (J Shoulder Elbow Surg 2006;15:728-735.)*

Study of the humeral attachment of the glenohumeral capsuloligamentous complex is underrepresented in the abundant literature on shoulder dislocation, whereas lesions on the glenoid side have been extensively described, simulated, and repaired.

According to several authors, Nicola was the first to describe, and then experimentally reproduce, a humeral avulsion of the glenohumeral ligaments (HAGL).[1,6,23] Some case reports and 2 larger clinical series have been published since 1988.[1,6,13,19,23] In a prospective study, Wolf et al.[24] performed arthroscopy of 64 shoulders with pure anterior instability at examination under anaesthesia. In these shoulders, which had failed conservative therapy, 6 revealed a HAGL (9.3%). Bokor et al.[3] reviewed the records of 514 surgical procedures on shoulders with traumatic anterior instability; 130 shoulders did not have a Bankart lesion, and of these, 35 had a HAGL (7.5%).

Gagey et al.[7] experimentally dislocated cadaver shoulders and noted capsular failure at the humeral side in 63%. In a biomechanical study, Bigliani et al.[2] demonstrated that the isolated inferior glenohumeral ligament complex (IGHL) tore at the humeral insertion in 25% when loaded to mechanical failure. In addition, the relative paucity of capsuloligamentous lesions on the humeral side seems to be in contradiction to the observed lateral thinning of the capsuloligamentous structures from the glenoid to the humerus.[4] Therefore, there seems to be a discrepancy in the reported frequency of HAGL between clinical and experimental studies.[21]

The present work was started to study the contribution of various humeral-sided lesions of the capsuloligamentous complex to anterior shoulder instability in an experimental shoulder model that specifically maintains all surrounding muscular structures. Our purpose was to determine the extent of capsuloligamentous damage necessary to allow the humeral head to dislocate when lesions are on the humeral side.

To the best of our knowledge, no selective cutting study of the humeral side has yet been published.

## MATERIALS AND METHODS

### *Dissection and cutting sequence*

Sixty-five fresh adult cadaver shoulders from 37 donors—27 female and 10 male—aged 54 to 94 years (mean 78.6 $\pm$ 10.8) were studied. The specimens were mounted through the scapula on a custom-made jig (Figure 1).

Through an axillary approach, first the neurovascular bundle was removed. The bellies of the biceps brachii, the deltoid, and the pectoralis major were reflected laterally to allow sufficient exposure. The belly of the subscapularis was carefully separated from the underlying capsule, taking great care not to damage the underlying the capsule, while leaving the tendon and the insertion of the subscapularis intact. In the first 10 specimens, the upper centimeter of the insertion of the tendon of the latissimus dorsi was detached

From the [a]Department of Orthopaedics and Traumatologie, Academic Hospital Vrije Universiteit Brussel and [b]Service d'orthopédie, Hôpital Bicêtre, Université Paris-Sud and Institut d'Anatomie.

Reprint requests to: Nicole Pouliart, Academic Hospital Vrije Universiteit Brussel, Department of Orthopaedics and Traumatologie, Laarbeeklaan 101, Brussels 1090, Belgium (E-mail: *izvptn@az.vub.ac.be*).



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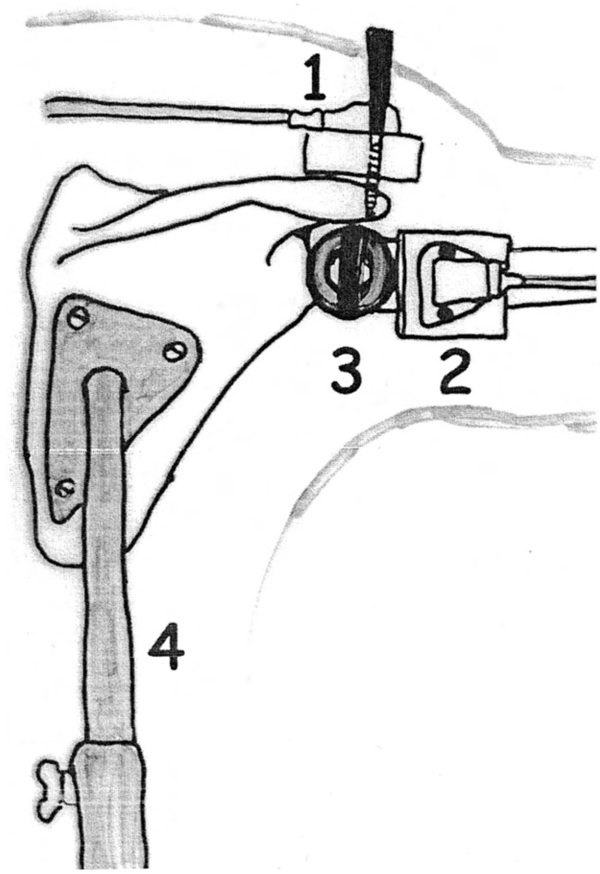


**Figure 1** Diagram of a mounted specimen. Right shoulder seen from the back in 90° of external rotation and 90° of glenohumeral abduction. Legend: 1 = master sensor of electromagnetic tracking device fixed in a custom made block to humeral head with threaded stainless steel pins; 2 = slave sensor fixed to top of acromion; 3 = hand-held dynamometer applied to posterior aspect of humeral head during anterior stability testing; 4 = custom-made stainless steel device for rigidly fixing the scapula.

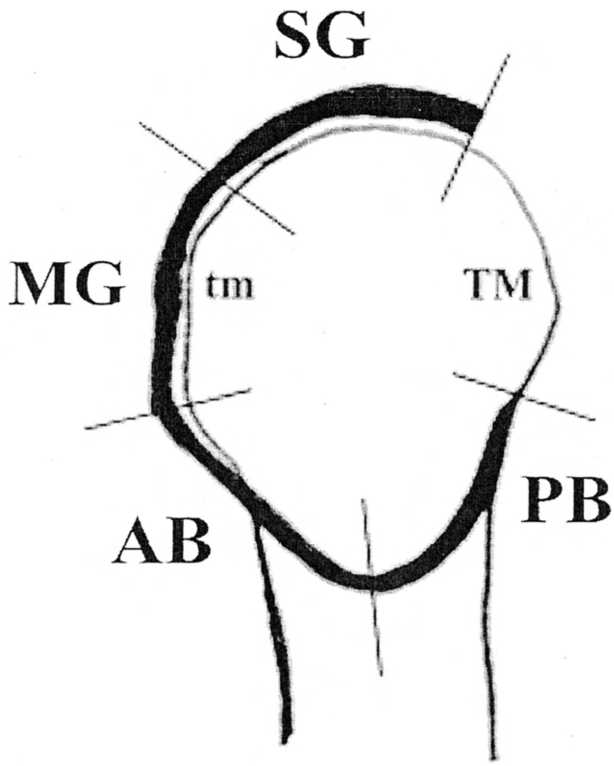


**Figure 2** Localization of the sequential cuts. Thick black line = capsule; straight thin black lines delineate cutting zones. Zone PB, from 9 to 6 o'clock for a right shoulder, was designated to include the posterior band of the inferior glenohumeral ligament (IGHL). Zone AB, from 6 to 3 o'clock, included the anterior band of the IGHL; zone MG, from 3 o'clock to the bicipital groove at approximately 1 o'clock, included the middle glenohumeral ligament (MGHL); zone SG, from the bicipital groove at approximately 1 o'clock to 11 o'clock, included the superior glenohumeral ligament (SGHL); tm = tuberculum minus; TM = tuberculum maius.

from the humerus and from the insertion of the inferior glenohumeral capsule to facilitate the capsular cutting sequence. From these pilot dissections, we learned that it was possible to free and cut the V-shaped inferior part of the capsule by lifting the tendon off the capsule without actually detaching it. Therefore, the tendon of the latissimus dorsi was left intact in all other specimens. After completing the approach to the glenohumeral capsule, a consecutive cutting sequence of the capsuloligamentous structures on the humeral side was done to simulate a HAGL. Four zones to be cut were defined (Figure 2). From an inferior approach, zone MG and SG could only be cut without damage to the rotator cuff muscles when the capsule had been opened through zone AB. Therefore, it was impossible to start the cutting sequence anywhere else than in zone AB or PB, and adjacent zones needed to be cut consecutively. These 2 limitations influenced the variations in cutting sequence that could be done (Table I). The sequence was, nevertheless, altered to determine whether any specific combination of zones influenced the degree of instability. Any of these cuts were carefully controlled in depth in order not to damage the tendons of subscapularis, supra- and infraspinatus, and long head of the biceps. The cutting sequence was terminated once grade 4 instability was reached. At the end of the entire cutting sequence, the intraarticular aspect of the glenohumeral side was inspected to verify that the capsule was indeed cut as desired, and that the tendons were indeed still intact.

### Testing procedure

After each step, each shoulder was subjected to a standard inferior (sulcus) test and to an anterior drawer test (translation) with the arm in 0° of abduction, 0° of elevation, and in neutral rotation (anatomic position), as well as to a load-and-shift test (LAS) in the apprehension position. For the LAS test, a goniometer was used to position the humerus in 90° of external rotation and 90° of abduction in the scapular plane. In the drawer and the LAS test, an anteriorly

**Table I** Overview of planned cutting sequence and actual cuts done

| Lat dors | Zone cut 1 | Planned number | Sequence actually cut | Actual number | Zone cut 2 | Planned number | Sequence actually cut | Actual number | Zone cut 3 | Planned number | Sequence actually cut | Actual number | Zone cut 4 | Planned number | Sequence actually cut | Actual number |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Intact | PB | 20 | PB | 20 | AB | 20 | PB-AB | 20 | MG | 20 | PB-AB-MG | 20 | SG | 20 | PB-AB-MG-SG | 19 |
| Intact | AB | 35 | AB | 35 | PB | 15 | AB-PB | 15 | MG | 15 | AB-PB-MG | 15 | SG | 15 | AB-PB-MG-SG | 13 |
| Intact | | | AB | | MG | 20 | AB-MG | 20 | PB | 10 | AB-MG-PB | 10 | SG | 10 | AB-MG-PB-SG | 9 |
| Intact | | | | | | | | | SG | 10 | AB-MG-SG | 10 | PB | 10 | AB-MG-SG-PB | 9 |
| Incised | AB | 10 | AB | 10 | MG | 10 | AB-MG | 10 | SG | 10 | AB-MG-SG | 10 | PB | 10 | AB-MG-SG-PB | 10 |
| Total | | 65 | | 65 | | 65 | | 65 | | 65 | | 65 | | 65 | | 60 |

Zones as detailed in text and illustrated in Figure 1. Designation for sequences used throughout in other tables.
Lat dors, tendon of latissimus dorsi.

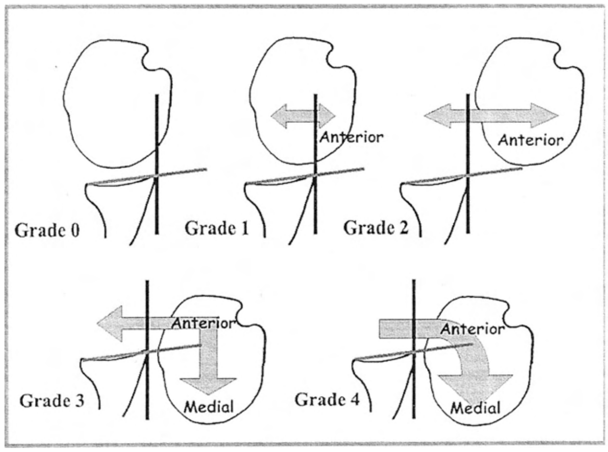


**Figure 3** Diagram illustrating the scale for anterior instability.[12,14,15] Grade 0 = stable: no anterior, medial, or inferior translation; grade 1 = increased translation: anterior translation <10 mm, no medial or inferior translation with return to the original position upon release of translating force; grade 2 = subluxation: anterior translation >10 mm, no medial or inferior translation with return to original position upon release of translating force; grade 3 = metastable dislocation: anterior translation >10 mm, with medial translation; return to the normal glenohumeral relationship when bringing the arm into its anatomic position without a reduction maneuvre (= spontaneous reduction); grade 4 = locked dislocation: anterior translation >10 mm, with medial translation; the humeral head remains displaced in the dislocated position when bringing the arm into its anatomic position without a reduction maneuvre.

directed translation force of 50N combined with an axial compression force of 50N was applied to the humeral head with a handheld dynamometer (MicroFET2®, Hoggan HealthIndustries, distributed by Biometrics Europe BV, 1322 AD Almere, The Netherlands). If dislocation occurred, the humerus was brought back into its anatomic position without a reduction maneuvre. In the sulcus test, an inferiorly directed translation force of 50N was applied to the elbow. An electromagnetic tracking device (EMTD, "Flock of Birds®," Ascencion Technology, Burlington, VT) was used to record translation values and Eulerian angles. The validity of using an EMTD for measuring shoulder laxity was described by Reis et al.[16] The electromagnetic transmitter was placed near the specimen tested and in line with the scapular plane. One sensor (master) was rigidly fixed to the acromion, and the second (slave) was rigidly fixed to the humeral head so that it was at right angles to the master sensor (Figure 1). Translation of each sensor relative to the transmitter was recorded in 3 planes and transformed into a relative translation between both sensors by a custom-made software program. When no angular displacement between humeral head and scapula occurs during testing, the relative translation between both sensors equals the displacement of humeral head relative to the stable scapula. Maximal humeral abduction (hyperabduction test, HAT)[8] and maximal external rotation in 90° of abduction (ABERmax) were recorded with the EMTD and, as a control of its accuracy, also measured with a goniometer.

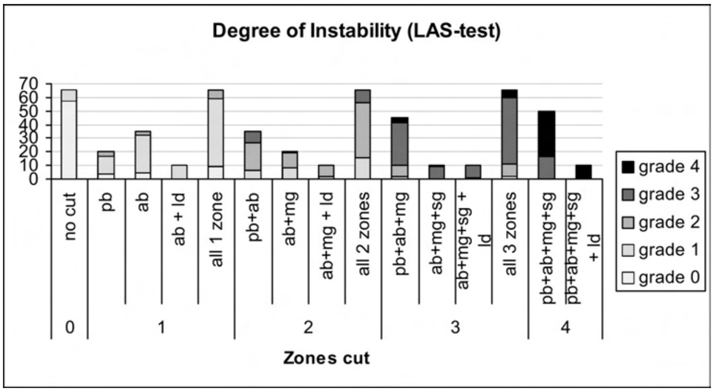


**Figure 4** Graph for the degree of instability during the load-and-shift test (LAS test) for each cutting sequence and for each step. Cumulative bars representing the number of specimens with a specific degree of instability for a specific combination of zones cut and the total for each step. Zones: pb = posteroinferior zone; ab = anteroinferior zone; mg = anterior zone; sg = superior zone; ld = latissimus dorsi.

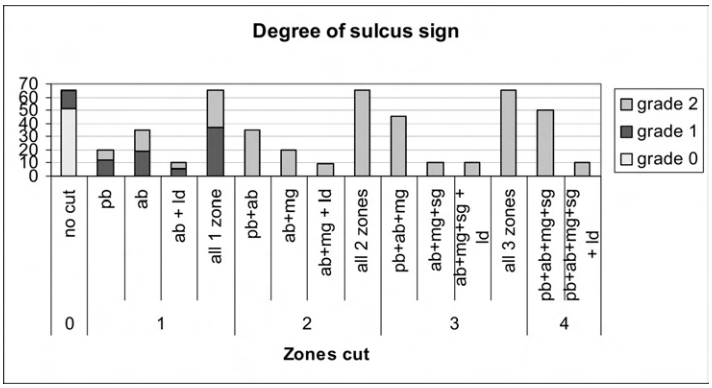


**Figure 5** Graph for the degree of sulcus sign for each cutting sequence and for each step. Cumulative bars representing the number of specimens with a specific degree of sulcus sign for a specific combination of zones cut and the total for each step. Zones: pb = posteroinferior zone; ab = anteroinferior zone; mg = anterior zone; sg = superior zone; ld = latissimus dorsi.

*Classification of instability*

Higher grades of instability (i.e., dislocation) are associated with anterior translation, but also with medial and usually some inferior translation as well. Reduction of a dislocation is accompanied by a return to the predislocation glenohumeral spatial relationship. We incorporated these parameters into 1 grading system to be able to analyze the results statistically. This system is based on the grading of clinical instability adopted by the American Shoulder and Elbow Surgeons.[17] We classified the displacement of the humeral head in relation to the glenoid resulting from the 3 stability tests and from the neutral repositioning to neutral, into 4 grades[12,14,15] (Figure 3).

Statistical analysis was done with SPSS for Windows (version 13.0®, SPSS Belux, 1020 Brussels, Belgium). Nonparametric tests were used, because grading results in ordinal categorical variables: frequency distribution, Pearson's chi-square, spearman's rho correlation coefficient.

## RESULTS

In 5 specimens, the cutting sequence was discontinued because a grade 4 instability was obtained after section of 3 zones. In these 5 specimens, a tear of the subscapularis tendon occurred during the apprehension test after step 3. Table I also details the actual numbers done for each cutting sequence. In 17 of 65 shoulders, grade 4 instability was not achieved after cutting all 4 ligamentous zones. In these specimens, we had the impression that the tendon of the latissimus dorsi seemed to act as a check rein on the

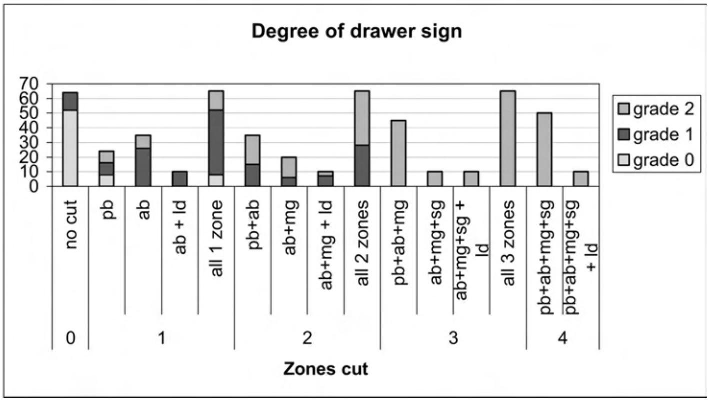


**Figure 6** Graph for the degree of drawer sign for each cutting sequence and for each step. Cumulative bars representing the number of specimens with a specific degree of drawer sign for a specific combination of zones cut and the total for each step. Zones: pb = posteroinferior zone; ab = anteroinferior zone; mg = anterior zone; sg = superior zone; ld = latissimus dorsi.

humeral head, relocating the head when returning the humerus into neutral position. With each step in the cutting sequence, a progressive increase in the degree of instability (Figure 4), degree of sulcus sign (Figure 5), and degree of drawer sign (Figure 6) was observed.

The Pearson chi-square test showed that there was a statistically significant difference for sulcus, translation, and instability between all groups for sequence ($P < .001$) and for number of zones cut ($P < .001$). However, when grouping per sequence within the same number of zones cut, the intergroup difference for sulcus and instability was not statistically significant ($P > .05$). For translation, there was only a statistically significant intergroup difference within 1 zone cut groups ($P < .001$). This difference seemed to be due to the fact that in 40% of specimens, where only zone PB was cut, the degree of translation was 0, whereas cutting zone AB resulted in at least grade 1 translation.

Spearman's rho was calculated to test correlation between all variables. The correlation of the number of zones cut with degree of instability was very high (rho = 0.93, $P < .001$), and a little lower with degree of drawer sign (rho = 0.83, $P < .001$) and with degree of sulcus sign (rho = 0.79, $P < .001$). The correlation of the 3 grading variables with each other was of the same order of magnitude (0.76 < rho < 0.84; $P < .001$).

## DISCUSSION

The present experimental setup certainly has limitations. As no robotic simulator is involved, the standardization of the forces used is not as rigid, and the contribution of static stabilizers only can be studied. To study the dynamic stabilizers as well, one needs at least to simulate muscle forces. Another limitation is that this protocol uses a cutting sequence and, therefore, does not take prefailure stretching of the ligamentous structures into account. A main disadvantage of experimental studies employing a robotic system is that only anterior translation is measured. However, translation is not truly a correct parameter of instability and is difficult to measure accurately in a clinical setting. Several studies have shown that there is a large overlap in anterior translation values between patients with instability and normal subjects.[5,9–11,18,20,22] In clinical situations, instability is not only judged on the basis of anterior translation but also on the basis of a variety of instability tests. Of these, the apprehension and relocation tests, of which the load-and-shift test is a modification, are the most commonly used. The apprehension position is, as far as we know, the only testing position that has a high degree of reliability for evaluating clinical shoulder instability, and more specifically, dislocatability. Moreover, clinically, anterior dislocation most frequently occurs when the humerus is stressed in the apprehension position, which resembles the throwing position (90° or more of external rotation and at least 90° of abduction). We wished to analyze the consequences of our cutting sequence in a way that could more easily be extrapolated to the clinical situation. This led to the development of the current testing protocol that uses the degree of instability, graded on a scale of 5, resulting from a LAS test. The analysis of our results also showed that, in this cutting sequence, a certain number of zones cut corresponded to a very high extent with the resulting degree of instability; 1 zone cut almost always equalled grade 1 instability, 2 zones cut = grade 2, 3 zones cut = grade 3, and 4 zones cut = grade 4. Therefore, the humeral-based capsuloligamentous cutting sequence has the potential of being a valid experimental model for varying degrees of anteroinferior instability and dislocation. As there was some variation in our specimens, this potential model still needs further validation. One requisite is that the subscapularis tendon remains intact, because tearing of this tendon led to grade 4 instability after only 3 zones were cut in 5 specimens. In all other specimens, all 4 zones required cutting before a locked dislocation would occur. In 17 specimens, however, cutting 4 zones still did not result in grade 4 instability. On first impression, the tendons of latissimus dorsi and subscapularis seemed to push the humeral head back into its anatomic position when bringing the humerus back into neutral position after dislocation. This needs further investigation, and if this impression is confirmed, the tendon of the latissimus dorsi should systematically be cut in order for the experimental model to be more valid.

HAGL has rarely been described in clinical series. Over the last 15 years, though, this type of lesion has been reported more frequently.

The incidence of HAGL seems to be limited to about 10% in dedicated series of shoulder instability, such as those by Wolf et al. and Bokor et al.[3,24] In experimental studies provoking dislocation, the capsuloligamentous complex is reported to fail on the humeral side in 25% to 63%.[2,7]

One reason might be that the lesion is more likely in older subjects. Cadaver shoulders are generally obtained from an older population, whereas clinical series of shoulder instability usually concern younger subjects. In the series of Bokor et al.[3], the mean age of the group with HAGL (27.1) was slightly higher than the overall group (23.0) and that of the group without HAGL (22.7).

The clinical incidence of HAGL lesions may be underestimated. In open surgery, when the subscapularis is separated from the capsule, the lesion can easily be attributed to a iatrogenic breach of the capsule. When the subscapularis and the capsule are divided together, the HAGL can also easily be missed. In arthroscopic surgery, it may be missed because it is not specifically looked for or because it is not recognized. On the other hand, lesions on the humeral side may not be appreciated, because they may have healed by the time of surgical exploration. With radiographic imaging, the HAGL is difficult to visualize, either because it has already healed or because adherence of the subscapularis to the capsule prevents contrast from leaking through the lesion.

However, another explanation for the discrepancy between the clinical and experimental incidence might be that the lesion does not immediately lead to recurrent dislocation. On the 1 hand, these lesions might heal relatively well thanks to the adherence of the subscapularis to the capsule or because it concerns a bony avulsion. On the other hand, the lesion itself might not be able to induce instability, as long as it is not extensive enough. In most experimental studies published about shoulder dislocation, the ligaments are isolated and most, if not all, soft tissues are removed. This setup allows focusing on the role of the ligamentous structures but may lead to a misunderstanding of the stabilizing mechanisms and, more specifically, underestimate the role of secondary stabilizers. In this respect, it is important to stress that the present experimental model leaves all surrounding tissues intact initially.

From the present study, we can conclude that the humeral capsuloligamentous complex needs to be cut in at least 3 capsuloligamentous zones in order to obtain a dislocation. For a locked dislocation, a lesion including the superior (SGHL) and posterior band of the inferior glenohumeral ligament is a requisite, unless the subscapularis tendon is avulsed. In the report by Bokor et al.[3], the HAGL was nearly always found in the area of the inferior glenohumeral ligament complex. However, in our study, lesions of the entire inferior complex, that is, zones AB and PB, rarely resulted in a dislocation (grade 3) and never in a locked dislocation (grade 4). Clinical data and experimental work by other authors up to now did not specify that the middle and superior part of the capsuloligamentous complex needs to be involved as well. The role of the middle glenohumeral ligament in anteroposterior instability has already been described in the literature. As far as we know, the superior glenohumeral ligament (SGHL) has, until now, only been described as having a role in anterosuperior instability and rotator cuff interval lesions. The results from the present study imply that the SGHL may have a significant contribution to anteroinferior stability as well. We believe that the intact muscle model plays an important role in this discovery. As already mentioned, in this work, a locked dislocation was also possible when the subscapularis failed before the SGHL was cut. In the experimental models that remove the surrounding shoulder muscles, especially the rotator cuff muscles, dislocation could be encountered before the entire capsuloligamentous complex failed. Again, in a clinical setting, irrecoverable stretching of the capsuloligamentous structures occurs, and this may diminish the extent of frank tears required.

In contrast with glenoid-sided lesions, it may well be that additional soft tissue injuries or very extensive lesions of the capsuloligamentous complex are required for higher grades of instability than when the lesions are on the humeral side of the joint. This may be a factor explaining the paucity of humeral lesions in clinical series of chronic shoulder instability. A glenoid-based cutting sequence should be done using the present experimental model, to verify the experimental part of this hypothesis.

This possible requirement for a combination of lesions may also provide an explanation for 2-stage recurrent dislocation. An initial trauma could result in a capsuloligamentous tear on the humeral side, but still not lead to a (locked) dislocation because the surrounding muscles remain intact or because not all ligaments are torn. The ligaments heal with consistent laxity, and thereafter, the muscles become progressively attenuated or damaged, or the capsular tear becomes larger. Subsequently, the shoulder becomes unstable without a clear new trauma.

The muscular influence might also be an explanation for the apparent paradox between capsuloligamentous thickness and strength on the 1 hand and the preferential site for lesions on the other hand. One would expect the capsuloligamentous structures to rupture more easily and thus more frequently on the humeral side, because they are thinner and weaker at their humeral side than at their glenoid side. However, most experimental and clinical data show a greater tendency for lesions to occur at the glenoid side. On the humeral side, the capsuloligamentous structures seem to be protected by the surrounding muscles. The middle glenohumeral ligament is adherent to the subscapularis muscle and the superior glenohumeral is adherent to the superior part of the rotator cuff. In the majority of specimens the inferior glenohumeral ligament is covered in part by the latissimus dorsi tendon. On the glenoid side, there is no such protection. Dynamic action of the muscles may further increase this humeral static defense. However, as mentioned before, although we use an intact muscle model, we can only appreciate the effect of the surrounding muscles as passive restraints. To determine the amount of additional active stabilization another experimental setup is necessary.

## CONCLUSION

Humeral avulsion of the glenohumeral capsuloligamentous structures is a relatively infrequent finding in clinical series of shoulder instability. In an experimental setting, it results more frequently from dislocation maneuvres. After performing this selective cutting study in an intact muscle shoulder model, we suggest that extensive damage to the humeral side of these structures is necessary to provoke higher grades of instability. Whether associated muscular lesions are an important factor in augmenting instability needs to be determined in another experimental study.

The humeral-based cutting sequence of the capsuloligamentous structures may prove to be a valid model for predictably creating a certain degree of instability, although this should be validated with larger series of experiments.

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# Concomitant Rotator Cuff and Capsuloligamentous Lesions of the Shoulder: A Cadaver Study

Nicole Pouliart, M.D., Ph.D., and Olivier Gagey, M.D., Ph.D.

**Purpose:** Rotator cuff lesions have not yet been evaluated in association with capsuloligamentous lesions in an otherwise intact shoulder model. Our hypothesis was that less extensive capsuloligamentous lesions are necessary to allow dislocation in the presence of rotator cuff lesions. **Methods:** The supraspinatus and infraspinatus, or the subscapularis, or all 3 tendons were carefully detached from their insertion without damage to the underlying capsule. Adjacent combinations of 4 zones of the capsuloligamentous complex were then sequentially detached from the glenoid or from the humerus in 80 cadaver shoulders. Stability was tested before and after each resection step; this included testing of inferior stability with a sulcus test, and of anterior stability with a drawer test and with an apprehension maneuver. Findings from these specimens were statistically compared with those from the same types of tests in specimens with intact rotator cuffs. **Results:** Subluxation during the drawer and sulcus tests is already reached after the tendon lesion has been created, or after only 1 ligamentous zone has been cut. In the presence of a tendon lesion, at least 1 less cutting step is necessary for dislocation to occur in the apprehension position. There is a difference, however, between glenoid- and humerus-based lesions. **Conclusions:** The humeral head dislocates easily with less extensive capsuloligamentous lesions when rotator cuff lesions are present. In the present experimental model, passive stabilization provided by the rotator cuff appears more easily disrupted when associated with ligamentous lesions on the humeral side than with lesions on the glenoid side. This may be so because interdigitation of the cuff tendons with each other and with the capsule through the rotator cable is maintained with glenoid-sided lesions. **Clinical Relevance:** Rotator cuff lesions destabilize the glenohumeral joint. This effect is less pronounced when cuff tendons continue to interact with underlying capsuloligamentous structures. This finding may be relevant for small cuff tears and for partial cuff tears, especially those seen after dislocation. **Key Words:** Shoulder instability—Rotator cuff lesion—Capsuloligamentous lesion—Experimental model—Tendon defect model.

The rotator cuff is an important component of the dynamic stabilizers of the glenohumeral joint.[1,2] It also plays a secondary role in static stabilization because the lateral part of the rotator cuff tendons is interwoven with the glenohumeral capsule.[3,4] Several experimental studies have been undertaken with the goal of measuring the influence of the cuff on stability. The dynamic stabilizing effect has been evaluated by means of experimental models that (1) assessed the increase in muscle force necessary to maintain stability when capsuloligamentous lesions were present, or (2) determined the amount of instability that resulted when a specific tendon was unloaded.[1,5-10] Static effects can be evaluated through a muscle defect model by which a lesion is created in the cuff and stability testing is performed.[1,7,8,11,12] Unfortunately, the underlying superior capsule is usually damaged when the tendon defect is created—a fact that in itself may alter stability.

In previous studies in an intact shoulder model,[13-16] we were able to determine that stability in terms of the potential for dislocation resulted only from extensive

*From the Department of Orthopaedics and Traumatology and Department of Human Anatomy, Academic Hospital Vrije Universiteit Brussel (N.P.), Brussels, Belgium; and Service d'orthopédie, Hôpital Bicêtre, Université Paris-Sud (O.G.), Paris, France.*

*Address correspondence and reprint requests to Nicole Pouliart, M.D., Ph.D., Department of Orthopaedics and Traumatology, Academic Hospital Vrije Universiteit Brussel, Laarbeeklaan 101, 1090 Brussels, Belgium. E-mail: izvptn@az.vub.ac.be*



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capsuloligamentous lesions. In most specimens, at least 3 ligamentous zones had to be cut on the glenoid or the humeral side. In the same model, a rotator cuff lesion was created without damage to the underlying capsule. The present study tests the hypothesis that a rotator cuff lesion may diminish the extent of capsular damage necessary for dislocation to occur.

## METHODS

A total of 80 unembalmed cadaver specimens, aged 53 to 99 years, were studied after exclusion criteria were implemented. Specimens with surgical scars, limited range of motion, or pre-existing rotator cuff tears were excluded. The latter condition was verified through a small deltoid split incision.

We then created 3 groups of rotator cuff tendon lesions: supraspinatus and infraspinatus (group 1, 40 specimens); supraspinatus, infraspinatus, and subscapularis (group 2, 20 specimens); and subscapularis only (group 3, 20 specimens). In groups 1 and 2, the deltoid split incision was used to detach the supraspinatus and infraspinatus tendons from their humeral insertions while tendons from the underlying capsule were separated through sharp dissection (Fig 1). Originally, we intended to separate the supraspinatus and infraspinatus tendons, too. However, because both tendons have a closely interdigitated insertion on the humerus, it proved technically impossible to separate them in an unequivocal manner. The tendons and muscle bellies

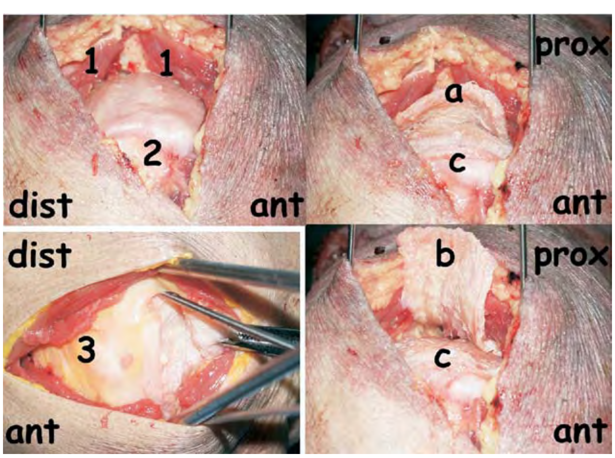


FIGURE 1. The supraspinatus and infraspinatus tendons (a) are detached (b) from the underlying superior capsule (c) through a deltoid split incision. 1, split deltoid muscle; 2, humeral head covered by rotator cuff tendons; 3, subdeltoid bursa. Orientation: Lateral view of right shoulder, except for bottom left picture (here seen from top). ant, anterior; dist, distal; prox, proximal.

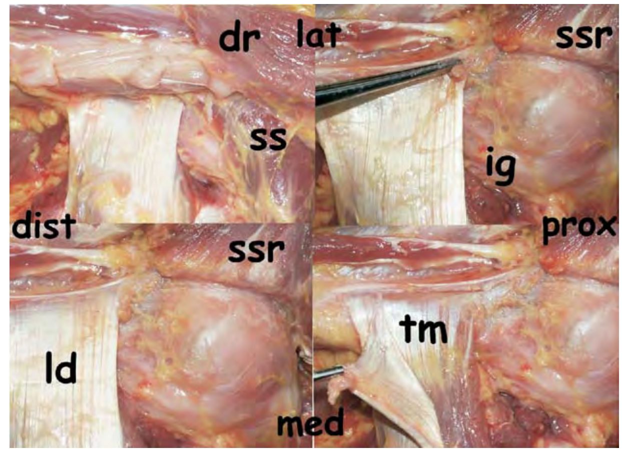


FIGURE 2. The axillary approach to the glenohumeral capsule with the subscapularis (ss) still in place (above right) or detached and reflected (ssr; in the other 3). The tendon of the latissimus dorsi (ld) is partially incised; the underlying tendon of the teres major (tm) is thereby uncovered. The deltoid muscle (dr) is reflected laterally. When the subscapularis is reflected, the glenohumeral capsule along with the inferior glenohumeral ligament (ig) is visible. Orientation: Right shoulder, axillary view with humerus abducted and externally rotated. med, medial; lat, lateral; prox, proximal; dist, distal.

were freed medially until no tether remained. When the intact capsule could not be maintained, the specimen was discarded from the study as well, although a small breach that caused venting of the joint was accepted. A deltoid split incision was also made in the shoulders from group 3, so the cuff could be inspected and the incision itself could not act as a confounding factor. In specimens from groups 2 and 3, the subscapularis was detached from the underlying capsule and was cut as close as possible to its insertion without damage to the capsule through an axillary approach to the glenohumeral joint (Fig 2). In all 80 specimens, the proximal 2 cm of the tendon of the latissimus dorsi was incised because a previous study[14] showed that this tendon influenced dislocation behavior when capsuloligamentous lesions occurred on the humeral side.

After these tendinous lesions had been created, specimens were tested for stability, as was outlined in detail earlier.[13-15] In summary, a maximal translation force of 50 N was applied with a handheld dynamometer, and the amount of anterior, medial, and inferior translation was monitored with an electromagnetic tracking device (Flock of Birds; Ascension Technology, Burlington, VT) during an inferior sulcus test, an anterior drawer test in 90° of abduction and neutral rotation, and a load-and-shift (LAS) test in 90° of abduction and 90° of external rotation. All stability tests were graded on an instability scale that ranged from 1 to 5 (Fig 3).

Subsequently, a sequential cutting protocol was performed through the axillary approach on the humeral side in half of the cases from each tendon group, and on the glenoid side in the other half. An algorithm has been created to detail the number of specimens in each group (Fig 4). On the humeral side, the posterior part of the inferior glenohumeral ligament was always cut first. This sequence was continued to the anterior part of the inferior glenohumeral ligament, then to the zone that includes the middle glenohumeral ligament, and finally to the zone that includes the superior glenohumeral ligament. A locked dislocation was the endpoint, and if this was not reached after all 4 ligamentous zones had been cut, the posterior capsule was cut as well. Because the posterior part of the inferior glenohumeral ligament on the glenoid side cannot be reached through an inferior approach unless the anterior part is cut first, the sequence had to be different from that on the humeral side. Therefore, the sequence on the glenoid side always proceeded from the anterior to the posterior part of the inferior glenohumeral ligament, then to the middle and superior glenohumeral ligament. Here, the part of the inferior

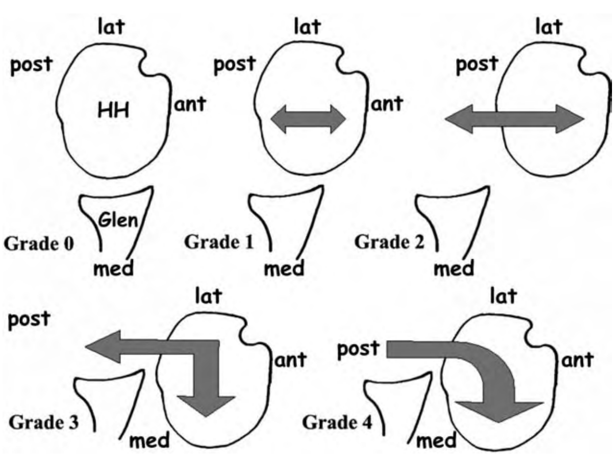


FIGURE 3. Grading method used for the load-and-shift and drawer tests, illustrated in transverse section through the glenohumeral joint. Grade 0, stable (no anterior [ant], medial [med], or inferior translation); grade 1, increased translation (anterior translation <10 mm; no medial or inferior translation); grade 2, subluxation (anterior translation >10 mm; no medial or inferior translation); grade 3, metastable dislocation (anterior translation >10 mm, with medial translation; return to the normal glenohumeral relationship when the arm is brought into its anatomic position without a reduction maneuver); grade 4, locked dislocation (anterior translation >10 mm, with medial translation; the humeral head [HH] remains dislocated when the arm is brought into its anatomic position without a reduction maneuver). glen, glenoid; post, posterior; lat, lateral.

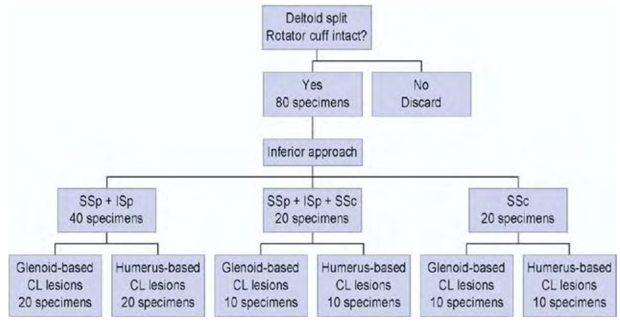


FIGURE 4. The distribution of specimens among subgroups. Cut tendons: SSp, supraspinatus; ISp, infraspinatus; SSc, subscapularis; CL, capsuloligamentous.

capsule that arises from the long tendon of the triceps (fasciculus obliquus) was detached when a grade of 4 could not be attained during the LAS test after all 4 ligamentous zones had been cut. The order of either sequence of ligamentous cuts was not changed from specimen to specimen because previous studies have shown that this would not lead to a statistically significant difference in results attained on instability testing.

Degree of sulcus sign, degree of drawer sign, and grading of the LAS test all constitute ordered categorical variables.[13-15] Therefore, nonparametric tests (Statistical Package for the Social Sciences [SPSS] for Windows, version 13.0; SPSS Belux, Brussels, Belgium) were used for statistical analysis; these consisted of Spearman's rho correlation coefficient and Pearson's $\chi$-square test. Statistical significance was set at $P < .05$.

Results of stability testing for capsuloligamentous lesions in rotator cuff–deficient specimens from this study were compared with results from previous studies that used the same experimental protocol to evaluate the consequences of humerus-sided lesions[13] versus those of glenoid-sided lesions (unpublished data)—both without rotator cuff lesions. Stratification was used to determine whether differences could be noted in the grading of the LAS test, the degree of drawer sign, or the degree of sulcus sign between specimens with and those without rotator cuff lesions, and whether such differences were related to the quantity of cut zones, the type of rotator cuff lesion, the site at which the cutting sequence occurred, or a combination of these subgroups.

## RESULTS

After any type of tendinous lesion was created, 65% of specimens remained fully stable during the LAS test (Fig 5); only 33.75% remained stable during the drawer test (Fig 6), and only 36.25% remained at grade 0 during the sulcus test (Fig 7).

With humerus-based lesions, most specimens became dislocated (metastable [grade 3] or locked [grade 4]) during the LAS test after a maximum of 3 ligamentous zones had been cut, regardless of the type of tendinous lesion that occurred (Fig 5). Locked dislocation occurred only after 4 zones had been cut in 4 specimens, and only after all 4 zones and the posterior capsule had been cut in 3 specimens. For glenoid-based lesions, regardless of the type of tendinous lesion that occurred, only half of specimens were dislocated during the LAS test after a maximum of 3 zones had been cut. In all, 40% of shoulders required that 4 zones should be cut; another 10% required that the triceps anchorage should be severed before dislocation was observed.

The drawer sign was already noted at grade 2 in 24 specimens when the tendinous lesion was created, but the capsuloligamentous structures were still intact (Fig 6). Cutting of a single zone resulted in grade 2 drawer sign in an additional 24 specimens (8 with cuts on the glenoid side, 16 with cuts on the humeral side). With 2 zones cut, a grade 2 drawer sign was not elicited in 12 of 40 specimens with cuts on the humeral side and in only 6 of 40 shoulders with cuts on the glenoid side. Except in 1 specimen, medial translation, which amounts to dislocation, was not observed during the drawer test.

Regardless of the type of tendon lesion and the site of ligamentous cut, 52 specimens reached a grade 2 sulcus sign with no ligamentous cuts; 14 additional specimens showed a grade 2 sulcus sign after 1 zone had been cut (Fig 7). Eight shoulders did not reach a grade 2 sulcus sign after 2 zones had been cut. Inferior dislocation with medial translation was not observed during the sulcus test.

Correlation analysis showed a high degree of correlation (Spearman's rho $> 0.8$, $P < .001$) for grading of the LAS test with the number of zones cut, regardless of stratification, that is, glenoid- versus humerus-based capsuloligamentous cuts, type of tendon lesion, or both (Table 1). Correlation with the other parameters for degree of sulcus sign and degree of drawer

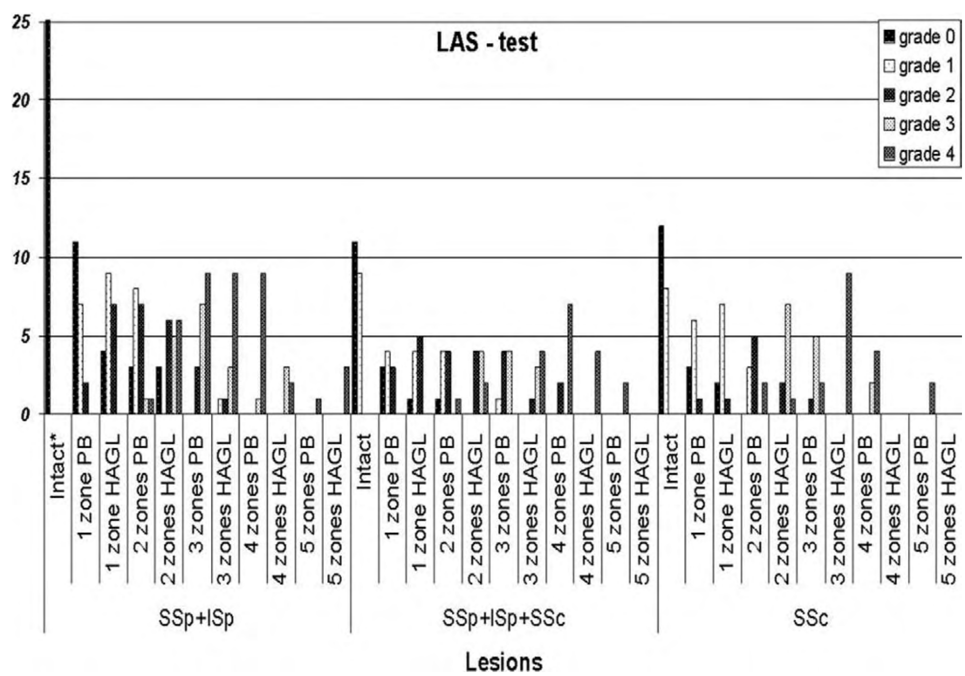


**FIGURE 5.** Grading of the load-and-shift test (LAS test) in 90° of external rotation with 90° of abduction for each step in the cutting sequence, grouped for type of tendinous lesion (group 1: supraspinatus and infraspinatus detached; group 2: supraspinatus, infraspinatus, and subscapularis detached; group 3: subscapularis detached) and for site of capsuloligamentous lesion (PB, glenoid-based; HAGL, humerus-based). Grading: grade 0, stable; grade 1, <10 mm increase in translation; grade 2, subluxation; grade 3, metastable dislocation; grade 4, locked dislocation. Intact*, number of specimens (40) surpassed limit of scale for this graph. Regardless of the type of tendinous lesion, most specimens with humerus-based lesions reach grade 3 with 3 or fewer cut ligamentous zones. This contrasts with specimens with glenoid-based lesions, most of which continue to require a minimum of 4 cut zones before grade 4 is observed. As soon as grade 4 is reached, specimens are not counted in the next step.

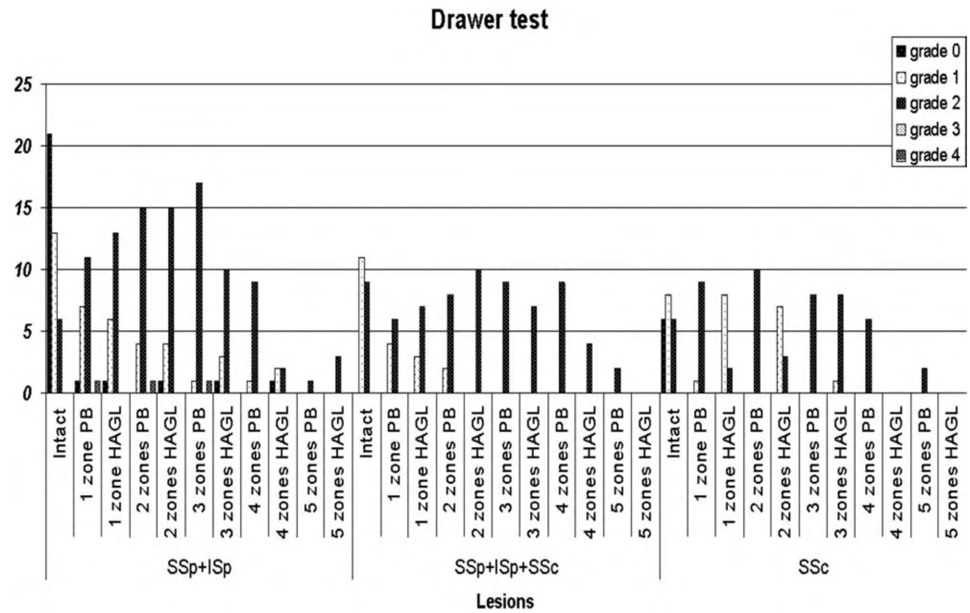


FIGURE 6. Grading of the drawer test for each step in the cutting sequence, grouped for type of tendinous lesion (group1: supraspinatus and infraspinatus detached; group 2: supraspinatus, infraspinatus, and subscapularis detached; group 3: subscapularis detached) and for site of capsuloligamentous lesion (PB, glenoid-based; HAGL, humerus-based). Grading: grade 0, stable; grade 1, <10 mm increase in translation; grade 2, subluxation; grade 3, metastable dislocation; grade 4, locked dislocation. More than half of specimens have already exhibited some degree of drawer sign when only the tendons are cut (especially when this included the subscapularis); 24 were at grade 2. Twenty-four additional specimens were of grade 2 after just 1 zone was cut, and another 14 were of grade 2 after 2 zones were cut. Dislocation occurred only once. Specimens were represented on this graph for drawer sign as long as they did not reach grade 4 in the LAS test.

sign was relatively high (0.8 > Spearman's rho > 0.6, $P < .001$).

Differences in grading of the LAS test, the sulcus test, or the drawer test resulting from different numbers of cut zones were statistically significant (Pearson's $\chi$-square, $P < .001$) for all specimens combined and for all stratifications (site of capsuloligamentous lesion, type of tendon lesion, or both).

When results of specimens with rotator cuff lesions in the present study were compared with those of specimens without rotator cuff lesions in previous studies[13] (unpublished data), higher grades were observed during the LAS, drawer, and sulcus tests for the same numbers of cut capsuloligamentous zones (Table 2). Differences in grading were statistically significant (Pearson's $\chi$-square, $P < .001$) for all specimens combined and for all stratifications outlined previously.

## DISCUSSION

In the present study of specimens with rotator cuff lesions, locked dislocation was attained in most after cutting of at least 1 less capsuloligamentous zone than was required in specimens from previous studies in which the rotator cuff was intact. These results confirm our hypothesis and allow us to conclude that rotator cuff lesions passively destabilize the glenohumeral joint.

A limited number of experimental studies have been dedicated to discerning the effects of rotator cuff insufficiency on glenohumeral stability. These can be subdivided into papers that use muscle loading and muscle unloading models,[1,5,7-9,17] and those that use tendon defect models.[1,7,8,11,12] In muscle unloading models,[1,5,7-9,17] the integrity of the cuff is not altered. Hence, tendons that are loaded can interact on the unloaded tendon because of interdigitation of all cuff tendons near their insertion. Only Blasier et al.[5] studied the effects of capsuloligamentous cuts in a rotator cuff–deficient model. In all studies that used a tendon defect model,[1,7,8,11,12] at least a portion of the underlying capsule and its ligaments were cut, together with the tendon defect. Furthermore, glenohumeral kinematics or anterior translation, but not dislocation, was evaluated. Finally, no rotator cuff insufficiency studies were conducted in specimens with a preserved soft tissue envelope. In all cases, only the rotator cuff, the

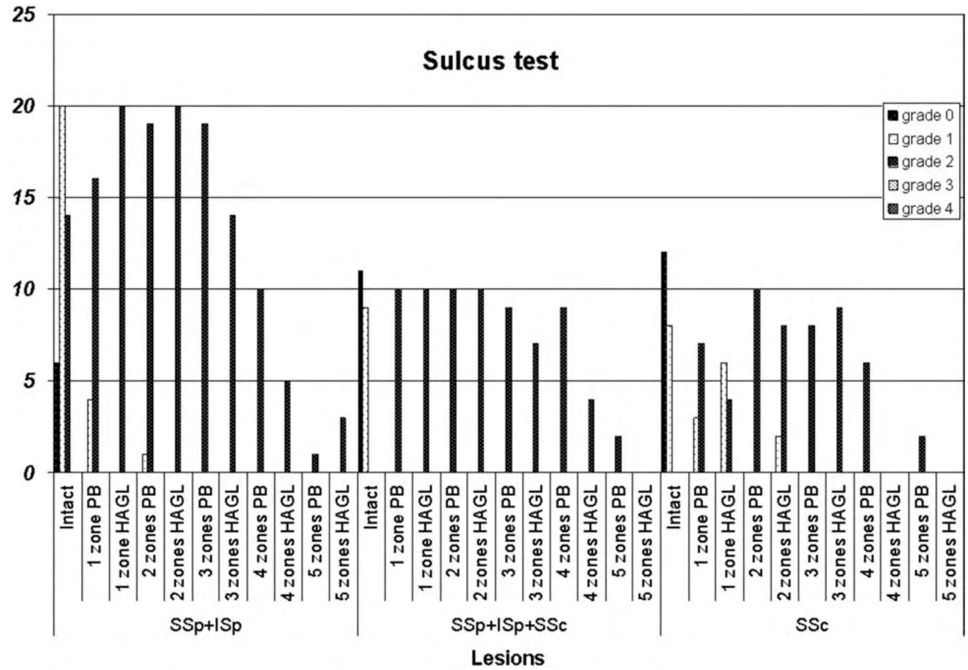


**FIGURE 7.** Grading of the sulcus test for each step in the cutting sequence, grouped for type of tendinous lesion (group 1, supraspinatus and infraspinatus detached; group 2, supraspinatus, infraspinatus, and subscapularis detached; group 3, subscapularis detached) and for site of capsuloligamentous lesion (PB, glenoid-based; HAGL, humerus-based). Grading: grade 0, stable; grade 1, <10 mm increase in translation; grade 2, subluxation; grade 3, metastable dislocation; grade 4, locked dislocation. Fifty-two specimens already were of grade 2 after only the tendons were cut, with 14 additional specimens of grade 2 after just 1 zone was cut, and another 6 after 2 zones were cut. Dislocation was not observed. Specimens were represented on this graph for sulcus sign as long as they did not reach grade 4 in the LAS test.

deltoid, and, in about half, the long tendon of the biceps were maintained. Our model preserved the soft tissue envelope; we created a rotator cuff defect without causing damage to the underlying capsuloligamentous complex, and we used the position at risk (the apprehension position of 90° of external rotation in 90° of abduction) to evaluate the potential for dislocation—not translation in itself.

Translatory forces were applied manually. Although the maximally applied force was standardized, this represents an important limitation of the present study. Another limitation is that we employed a static design in which forces on the retained tendons are not simulated. In addition, any cadaver study involving rotator cuff tendons is limited by the fact that it cannot simulate the active stabilizing role that the tendons play in vivo.

When only rotator cuff tendons were cut, drawer and sulcus signs were already positive in more than 50% of specimens; 75% of specimens remained stable

**TABLE 1.** *Spearman's rho Correlation Coefficients (all at* P < *.0005)*

| Stratification | Subgroup | Zones *v* LAS | Zones *v* Drawer | Zones *v* Sulcus | LAS *v* Drawer | LAS *v* Sulcus | Drawer *v* Sulcus |
|---|---|---|---|---|---|---|---|
| None | all | 0.877 | 0.643 | 0.606 | 0.581 | 0.640 | 0.697 |
| Site | hum | 0.888 | 0.558 | 0.597 | 0.624 | 0.595 | 0.682 |
| | glen | 0.886 | 0.703 | 0.613 | 0.673 | 0.573 | 0.709 |
| Tendon | ssp-isp | 0.876 | 0.620 | 0.617 | 0.572 | 0.642 | 0.691 |
| | ssp-isp-ssc | 0.886 | 0.665 | 0.537 | 0.563 | 0.688 | 0.695 |
| | ssc | 0.891 | 0.686 | 0.719 | 0.699 | 0.602 | 0.727 |
| Tendon and site | hum ssp-isp | 0.867 | 0.494 | 0.587 | 0.554 | 0.614 | 0.615 |
| | hum ssp-isp-ssc | 0.910 | 0.667 | 0.579 | 0.671 | 0.707 | 0.742 |
| | hum ssc | 0.931 | 0.630 | 0.723 | 0.672 | 0.583 | 0.709 |
| | glen ssp-isp | 0.902 | 0.723 | 0.656 | 0.582 | 0.678 | 0.764 |
| | glen ssp-isp-ssc | 0.899 | 0.670 | 0.493 | 0.474 | 0.676 | 0.653 |
| | glen ssc | 0.876 | 0.722 | 0.716 | 0.655 | 0.730 | 0.724 |

NOTE. Zones, number of zones cut in sequence; LAS, grading of the load-and-shift test in apprehension position; drawer, degree of drawer sign; sulcus, degree of sulcus sign; hum, humerus-based lesions; glen, glenoid-based lesions; ssp-isp, supraspinatus and infraspinatus lesions; ssc, subscapularis lesion.

**TABLE 2.** *Comparison of Specimens With and Without Rotator Cuff Lesions: Percentage of Specimens Reaching Highest Grading After Specific Number of Zones Were Cut*

| Grade | No. of Zones Cut | Rotator Cuff Intact | Rotator Cuff Lesion |
|---|---|---|---|
| Grade 2 sulcus sign | 1, all specimens | 15%–40% | 70%–100% |
| Grade 2 drawer sign | 1, all specimens | 20%–40% | 55%–90% |
| Grade 4 in LAS-test | 3, humerus-based | 10% | 77.5% |
| Grade 4 in LAS-test | 3, glenoid-based | 25% | 37.5% |

NOTE. LAS-test is the load-and-shift test in 90° of external rotation with 90° of abduction.

during the LAS test. Cutting of just 1 ligamentous zone destabilized most specimens to the point of inferior (sulcus test) and straight anterior (drawer test) subluxation. The passive stabilizing effects of superior rotator cuff tendons against inferior translation were confirmed by the fact that much less capsular damage was required with lesions to the supraspinatus and infraspinatus for the same degree of sulcus sign to be reached. In the drawer test, these superior tendons and the subscapularis tendon seem to provide passive stabilizing effects against anterior translation. The difference between specimens with or without rotator cuff lesions was less pronounced when lesions occurred on the glenoid side than when they developed on the humeral side. In the apprehension position (LAS test) and when capsuloligamentous lesions were on the glenoid side, rotator cuff tendons seemed to exert no major supplemental passive stabilizing effect against dislocation. This contrasts sharply with cuts on the humeral side, for which a rotator cuff lesion resulted in 1 less zone that needed to be cut to obtain a dislocation (metastable or locked) during the LAS test in at least half of specimens. One possible explanation is the interdigitation of cuff tendons and capsule on the humeral side.[3,4] With capsuloligamentous lesions on the glenoid side, cuff tendons that remain intact continue to help stabilize the joint through their action on the intact humeral insertion of the capsule. Also, diagonal reinforcement of the anteroinferior capsule from the long tendon of the triceps to the superior part of the subscapularis tendon is not damaged by cuts along the glenoid rim.[18-20] On the humeral side, the insertion of this fasciculus obliquus is cut, together with the middle glenohumeral ligament. The stabilizing effect of this part of the capsule has not been studied in detail. In fact, this ligament has received very little attention in the Anglo-American literature, perhaps because of limited access to German texts.

Tears of the rotator cuff are frequently seen in association with shoulder dislocation, especially in patients older than 40 years of age.[21-36] These tears may involve only the superior tendons (usually supraspinatus with or without extension into the infraspinatus), only the subscapularis, or all 3 tendons. Partial- and full-thickness tears have been reported in dislocating shoulders. In dislocations with rotator cuff tears, the middle and inferior glenohumeral ligaments are not necessarily damaged. In contrast, from an anatomic point of view, the superior glenohumeral ligament cannot remain intact when a full-thickness superior cuff lesion is present. Likewise, it is highly unlikely that the middle glenohumeral ligament will remain undamaged when a complete tear of the subscapularis occurs. These anatomic considerations combined with findings of the present study may help to explain why glenohumeral dislocations with associated rotator cuff lesions are prone to recurrence.

## CONCLUSIONS

This study shows that rotator cuff tendons contribute to passive stabilization of the glenohumeral joint. In the presence of cuff lesions, less extensive capsuloligamentous damage may result in dislocation in the apprehension position. With an intact cuff, more damage can be tolerated before destabilization to the point of dislocation occurs. Therefore, we infer that rotator cuff lesions should be looked for whenever a shoulder dislocation occurs; these should then probably be repaired, together with the capsuloligamentous lesions. This cadaver study, however, did not evaluate the active stabilization provided by rotator cuff tendons, nor were any other compensating mechanisms assessed. Nevertheless, investigators stress the importance of using intact shoulder models when the role of the glenohumeral ligaments is studied.

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ANATOMIC BASES OF MEDICAL, RADIOLOGICAL AND SURGICAL TECHNIQUES

V. Molina · N. Pouliart · O. Gagey

# Quantitation of ligament laxity in anterior shoulder instability: an experimental cadaver model

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**Abstract** Three groups of cadaver specimens were studied. In group 1 (20 shoulders) glenohumeral ligaments were detached from the humerus until a permanent dislocation of the humeral head occurred in abduction plus external rotation. On the dislocated joint the ligament was reconstructed using a fascia lata lengthening plasty. After the plasty had been completed, the shoulder was reduced and instability checked in the same position. Then the capsule (including the plasty) was harvested and measured. In group 2 (20 shoulders), after the plasty had been completed in the same conditions as above, the capsule was progressively reduced by 2 mm steps until the instability disappeared. Then the capsule (including the plasty) was harvested and measured. In group 3 (12 shoulders), measurements of the head and of the capsule were done. To dislocate the shoulder the section of the three glenohumeral ligaments was required. Lengthening of the capsule in group 1 was 240—250%. In all cases shortening of the capsule led to the stabilization of the shoulder. After stabilization of the shoulder was reached a residual lengthening of 175—185% was recorded. In 3 out of 4 shoulders the amount of capsule shortening required to return to a stable shoulder was between 16 and 18 mm. This experiment did not reproduce the Bankart lesion; therefore it only concerns atraumatic instability. The main limitation of this model is the low lever force that may be used to dislocate the shoulder; consequently the elasticity of the glenohumeral ligament was not taken in account. The experimental values were likely overestimated. Nevertheless the present results provide useful information for building an experimental model of atraumatic instability of the shoulder.

**Keywords** Atraumatic shoulder instability · Glenohumeral ligaments · Pathophysiology

V. Molina (✉) · N. Pouliart · O. Gagey
Service d'Orthopédie-Traumatologie,
Hôpital Universitaire de Bicêtre,
78, rue du Général Leclerc,
94270 Le Kremlin-Bicêtre, France
E-mail: veromolina@wanadoo.fr
Tel.: +33-1-45213335
Fax: +33-1-45213092

O. Gagey
Institut d'anatomie, 45, rue des Saints-Pères,
75006 Paris, France

## Introduction

Experimental studies dedicated to anterior shoulder instability have mainly focused on the consequences for joint stability of cutting ligaments and surrounding soft tissues [1, 3, 6, 9, 10, 12]. The role of ligament laxity, either traumatic or constitutional, is still well recognized in anterior chronic shoulder instability [4], mainly as regards the inferior and middle glenohumeral ligaments [5]. A few studies concern quantitation of the ligament laxity. A recent paper [7] has focused on the bony factors contributing to shoulder instability. Only one paper has been published regarding quantitation of the laxity of the glenohumeral ligaments performed on arthro-MRI [13]. Quantitation of the laxity of the glenohumeral ligaments is of importance since there are at present no data allowing correct adjustment of the length of the ligaments when performing a Bankart procedure. After the Bankart procedure, ligaments that are too short may leave a stiff shoulder, while those that are too long may led to persistent instability.

This work aimed to build and validate an anatomic model of chronic shoulder instability to provide a first quantitative evaluation of the laxity of the glenohumeral ligaments in atraumatic shoulder instability.

## Material and methods

### Material

Fifty-two (28 left, 24 right) fresh cadaver shoulders were studied in three groups. There were 10 men and 16 women, mean age 81 years (range 67–91 years). Shoulders were selected according to the following criteria: absence of deformity or scars, normal range of motion (ROM). At the beginning, discovery of a cuff tear led to rejection of the specimen. ROM was systematically recorded. Samples of fascia lata were harvested to be used for the ligamentous plasty.

### Prerequisites for the model

The shoulder should be (1) unstable, (2) with a continuous capsule and (3) the initial damage should involve only ligaments, leaving muscles intact. Such a model, in the absence of Bankart lesion 2, reproduces atraumatic shoulder instability associated with constitutional hyperlaxity.

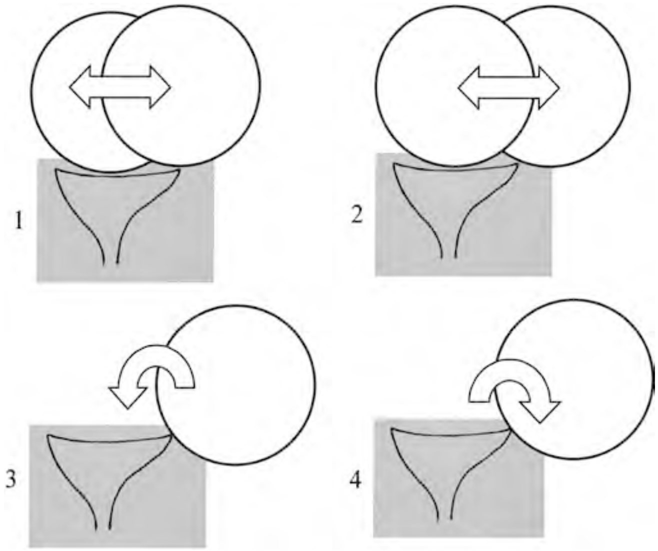


**Fig. 1** The stages of the classification: *0*, stable shoulder; *1*, increased sulcus and horizontal drawer; *2*, anterior translation of the humeral head, the top of the sphere does not pass over the glenoid rim; *3*, dislocation of the head in the apprehension position spontaneously reducing when the arm is left free; *4*, permanent or locked dislocation

### Methods

In group 1 (20 shoulders) dissection was carried out through an axillary approach; the axillary neurovascular bundle was cut and removed from the front of the scapulohumeral joint allowing exposure of the inferior aspect of the capsule anterior to the distal insertion of the latissimus dorsi and posterior to the insertion of the pectoralis major. The capsule was then carefully detached from the posterior aspect of the subscapularis allowing exposure of the capsule from 8 to 2 o'clock (for a right shoulder), which corresponds to the inferior and middle glenohumeral ligaments.

Cutting the capsule between 7 and 2 o'clock provided in all cases an anterior dislocation of the humeral head when the arm was placed in 90° abduction plus full external rotation. This dislocation reduced spontaneously when the arm was left free. In all cases but two a permanent dislocation occurred after the section of the superior glenohumeral ligament. The previous section of the capsule allowed the superior glenohumeral ligament to be cut easily. This result is consistent with those obtained in a study currently in press. This study also proposes a 5-stage classification of the experimental instability (Fig. 1):

0 normal and stable shoulder,
1 abnormal anterior displacement but the head does not pass over the anterior glenoid rim,
2 the head passes medially to the anterior glenoid rim,
3 the head dislocates anteriorly in abduction and external rotation but returns to its normal position when the arm is left free,
4 permanent dislocation even with the arm free.

This protocol allowed the creation of an unstable shoulder using only capsular damage.

After a stage 4 instability was obtained, a lengthening plasty of the capsule was performed on the dislocated joint in order to reproduce the ligament laxity required for an unstable shoulder. This plasty was performed with a piece of fascia lata fixed to the humerus with staples and to the genuine capsule with stitches. After the plasty had been completed the shoulder was reduced and the reproducibility of the dislocation in the same position as above was checked. The "stretched capsule" was then harvested and measurements of the whole capsule and of the genuine capsule were performed. Three measurements were done perpendicularly to the major axis of the sample at the level of its first quarter, of its center and of its third quarter (Fig. 2).

In group 2 (20 shoulders), the protocol was the same until the completion of the capsular plasty. The capsule was then reduced by 2 mm steps (Fig. 3). After each step stability of the joint was checked in abduction and full external rotation and ranked according the classification. The shortening was performed until a stage 0 was reached. After complete stabilization the capsule was harvested and measured according the same protocol as in group 1.

In group 3 (12 shoulders), after dissection as indicated for group 1 the capsule was harvested and measured as described. In addition the vertical and horizontal greater diameters of the humeral head were recorded.


**Fig. 2** Measurement protocol of the harvested capsule. The first, second and third quarters of the major axis are marked; measurements are done perpendicularly to the axis at the level of these three points

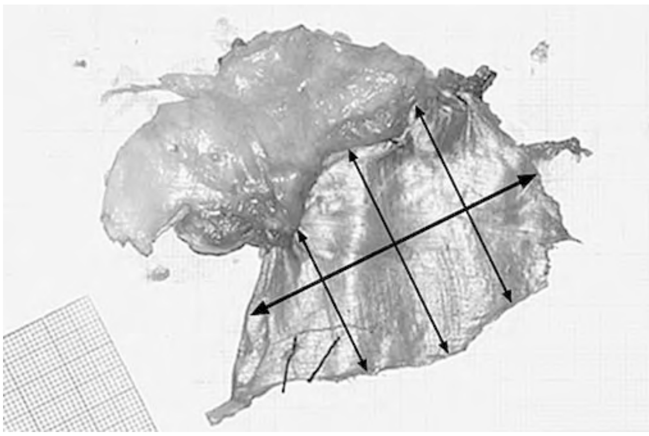

**Fig. 3** Lengthening plasty performed on the dislocated shoulder. *1*, Humeral head covered by the enlarged capsule; *2*, latissimus dorsi tendon; *3*, genuine capsule

## Results

In groups 1 and 2, 38 of 40 shoulders required a section of the inferior middle and superior glenohumeral ligaments to reach stage 4 instability. In the two other cases an additional cut of the latissimus dorsi tendon was necessary to pass from a stage 3 to a stage 4 instability.

Measures performed in group 1 are presented in Table 1. The lengthening of the capsule required to ensure a stage 4 instability was between 240% and 250%.

Table 2 presents the results of group 2. Residual lengthening of the capsule after return to a stage 0 instability was between 175% and 185%.

In all cases stabilization was reached by simple capsular reduction. Comparison of the results in group1 and 2 indicates that a 65% mean shortening of the capsule was required to return from stage 4 to stage 0 instability (Table 3).

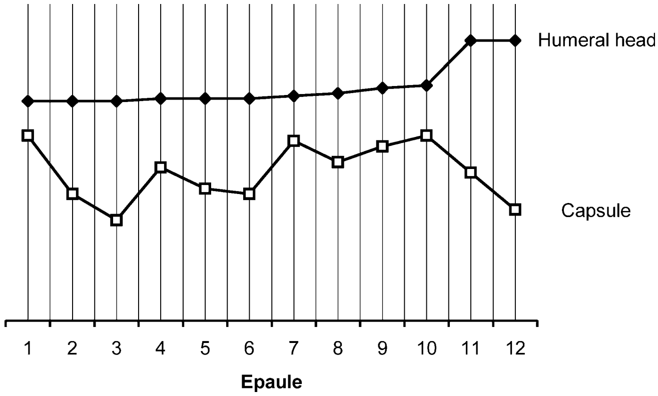


**Fig. 4** Measurements of humeral head diameters and capsule size

The shortening rate varied a great deal from one shoulder to another, but a 10 mm lower threshold was recorded. Two shoulders required a 20 mm capsular reduction, while 15 of 20 required a reduction between 16 and 18 mm.

Table 4 and Fig. 4 present the measures of humeral head diameters and size of the capsule. Despite its small size the sample strongly indicated that there is only weak correlation between the size of the humeral head and that of the capsule [Pearson coefficient: −0.17, Spearman coefficient: 0.17 ($p=0.59$)].

## Discussion

The aim of the present study was to build and validate an experimental model of the unstable shoulder. It is possible to create a stage 4 unstable shoulder with a continuous capsule and to return progressively toward a stable shoulder by successive reduction of the enlarged capsule.

In a previous paper [6] a traumatic model of shoulder dislocation has been described. In the present study we dislocated the shoulder using the same maneuver, which is the most recognized position in which dislocation occurs in chronic anterior shoulder instability. It should be emphasized that no data are available to precisely describe the different positions in which initial or recurrent dislocation occur. To reproduce exactly the same maneuver a machine should have been used. We did not use this technique for an important reason. Using a machine, the head would have followed a constant trajectory that would likely have been too far from the anatomic conditions. Therefore we chose to be as close as possible to the anatomy at the expense of a less precise position of dislocation.

The present study provides for the first time a quantitation of the ligament laxity required to create an experimental shoulder anterior instability. It suggests that a pure capsular lesion may provide shoulder instability.

**Table 1** Experimental values in group 1. Parts *a*, *b* and *c* are superior, middle and inferior capsule measurements, respectively. *Capsule*, normal capsule measurement (mm); *graft*, measurement of the fascia lata graft (mm); *C+G/C*, percentage capsular increase after grafting

| Shoulder no. | Part | Capsule | Graft | C+G/C | Part | Capsule | Graft | C+G/C | Part | Capsule | Graft | C+G/C |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | a | 28 | 43 | 2.54 | b | 22 | 54 | 3.45 | c | 22 | 44 | 3.00 |
| 2 | a | 32 | 46 | 2.44 | b | 31 | 49 | 2.58 | c | 22 | 34 | 2.55 |
| 3 | a | 27 | 47 | 2.74 | b | 28 | 47 | 2.68 | c | 20 | 42 | 3.10 |
| 4 | a | 22 | 51 | 3.32 | b | 28 | 44 | 2.57 | c | 26 | 30 | 2.15 |
| 5 | a | 22 | 50 | 3.27 | b | 43 | 45 | 2.05 | c | 25 | 47 | 2.88 |
| 6 | a | 16 | 29 | 2.81 | b | 28 | 25 | 1.89 | c | 12 | 33 | 3.75 |
| 7 | a | 37 | 43 | 2.16 | b | 31 | 50 | 2.61 | c | 33 | 28 | 1.85 |
| 8 | a | 23 | 32 | 2.39 | b | 23 | 33 | 2.43 | c | 25 | 30 | 2.20 |
| 9 | a | 24 | 40 | 2.67 | b | 28 | 42 | 2.50 | c | 24 | 46 | 2.92 |
| 10 | a | 24 | 43 | 2.79 | b | 41 | 62 | 2.51 | c | 41 | 46 | 2.12 |
| 11 | a | 31 | 34 | 2.10 | b | 46 | 54 | 2.17 | c | 32 | 59 | 2.84 |
| 12 | a | 24 | 29 | 2.21 | b | 27 | 34 | 2.26 | c | 21 | 26 | 2.24 |
| 13 | a | 32 | 39 | 2.22 | b | 32 | 36 | 2.13 | c | 39 | 27 | 1.69 |
| 14 | a | 27 | 33 | 2.22 | b | 28 | 38 | 2.36 | c | 27 | 27 | 2.00 |
| 15 | a | 28 | 34 | 2.21 | b | 24 | 35 | 2.46 | c | 26 | 29 | 2.12 |
| 16 | a | 31 | 34 | 2.10 | b | 36 | 47 | 2.31 | c | 32 | 37 | 2.16 |
| 17 | a | 30 | 38 | 2.27 | b | 35 | 43 | 2.23 | c | 31 | 36 | 2.16 |
| 18 | a | 39 | 41 | 2.05 | b | 32 | 45 | 2.41 | c | 34 | 37 | 2.09 |
| 19 | a | 31 | 34 | 2.10 | b | 39 | 49 | 2.26 | c | 37 | 38 | 2.03 |
| 20 | a | 23 | 31 | 2.35 | b | 23 | 36 | 2.57 | c | 25 | 29 | 2.16 |
| Mean | | 27.55 | 38.55 | 2.45 | | 31.25 | 43.40 | 2.42 | | 27.70 | 36.25 | 2.40 |
| SD | | 5.51 | 6.79 | 0.38 | | 6.86 | 8.75 | 0.32 | | 7.12 | 8.78 | 0.52 |

Validating this model required it to be demonstrated that the reduction of the capsular laxity led to a return to a stable shoulder. Therefore our results suggest that capsular laxity may be the main factor of chronic anterior instability of the shoulder.

Nevertheless this model has two main limitations. The forces applied to the joint after capsular plasty were necessarily limited in order to avoid tearing the capsule. The natural elasticity of the capsule was not taken into account in the present results. The size of the plasty also depends on the final position of the dislocated head. Unfortunately no data are available to quantify the position of the dislocated head. Consequently these results are likely overestimated; this may explain the results presented in Table 1.

The absence of a Bankart lesion should be taken into account. The Bankart lesion may participate in the dislocation in two ways. The labral lesion may facilitate the dislocation by diminishing the height of the anterior glenoid rim, and a "bucket-handle" of the labrum may participate in the laxity of the capsule. Therefore the present model is of use only for studying atraumatic chronic anterior instability. This might also contribute to explaining the high rate of laxity recorded in the study.

**Table 2** Experimental values in group 2. Parts *a*, *b* and *c* are superior, middle and inferior capsule measurements, respectively. *Capsule*, normal capsule measurement (mm); *graft*, measurement of the fascia lata graft (mm); *C+G/C*, percentage of capsular increase after grafting

| Shoulder | Part | Capsule | Graft | C+G/C | Part | Capsule | Graft | C+G/C | Part | Capsule | Graft | C+G/C |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | a | 18 | 30 | 2.67 | b | 20 | 31 | 2.55 | c | 20 | 33 | 2.65 |
| 2 | a | 26 | 22 | 1.85 | b | 31 | 25 | 1.81 | c | 25 | 24 | 1.96 |
| 3 | a | 29 | 25 | 1.86 | b | 35 | 30 | 1.86 | c | 32 | 25 | 1.78 |
| 4 | a | 42 | 35 | 1.83 | b | 41 | 29 | 1.71 | c | 31 | 11 | 1.35 |
| 5 | a | 24 | 20 | 1.83 | b | 21 | 17 | 1.81 | c | 28 | 21 | 1.75 |
| 6 | a | 16 | 13 | 1.81 | b | 15 | 10 | 1.67 | c | 20 | 22 | 2.10 |
| 7 | a | 31 | 22 | 1.71 | b | 36 | 25 | 1.69 | c | 34 | 28 | 1.82 |
| 8 | a | 23 | 20 | 1.87 | b | 34 | 21 | 1.62 | c | 27 | 17 | 1.63 |
| 9 | a | 24 | 19 | 1.79 | b | 35 | 21 | 1.60 | c | 23 | 20 | 1.87 |
| 10 | a | 35 | 22 | 1.63 | b | 33 | 30 | 1.91 | c | 31 | 17 | 1.55 |
| 11 | a | 34 | 20 | 1.59 | b | 35 | 27 | 1.77 | c | 29 | 21 | 1.72 |
| 12 | a | 21 | 11 | 1.52 | b | 24 | 18 | 1.75 | c | 20 | 12 | 1.60 |
| 13 | a | 22 | 13 | 1.59 | b | 19 | 12 | 1.63 | c | 20 | 10 | 1.50 |
| 14 | a | 28 | 21 | 1.75 | b | 25 | 18 | 1.72 | c | 21 | 15 | 1.71 |
| 15 | a | 22 | 22 | 2.00 | b | 28 | 16 | 1.57 | c | 34 | 18 | 1.53 |
| 16 | a | 30 | 25 | 1.83 | b | 34 | 30 | 1.88 | c | 31 | 24 | 1.77 |
| 17 | a | 24 | 21 | 1.88 | b | 21 | 18 | 1.86 | c | 27 | 20 | 1.74 |
| 18 | a | 24 | 21 | 1.88 | b | 29 | 20 | 1.69 | c | 26 | 18 | 1.69 |
| 19 | a | 23 | 20 | 1.87 | b | 24 | 21 | 1.88 | c | 30 | 19 | 1.63 |
| 20 | a | 28 | 25 | 1.89 | b | 30 | 23 | 1.77 | c | 26 | 20 | 1.77 |
| Mean | | 26.20 | 21.35 | 1.83 | | 28.50 | 22.10 | 1.79 | | 26.75 | 19.75 | 1.76 |
| SD | | 6.13 | 5.44 | 0.23 | | 7.03 | 6.18 | 0.21 | | 4.80 | 5.58 | 0.27 |

**Table 3** Progressive decrease of instability while lengthening plasty decreases. Data are classified in descending order

| Shoulder no. | Reduction (mm) | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | 2 | 4 | 6 | 8 | 10 | 12 | 14 | 16 | 18 | 20 |
| 10 | 4 | 4 | 3 | 3 | 3 | 3 | 2 | 1 | 1 | 0 |
| 11 | 4 | 3 | 3 | 3 | 2 | 2 | 1 | 1 | 1 | 0 |
| 16 | 4 | 4 | 3 | 3 | 3 | 2 | 2 | 1 | 0 | |
| 9 | 4 | 4 | 3 | 3 | 2 | 2 | 1 | 1 | 0 | |
| 2 | 4 | 4 | 3 | 3 | 2 | 2 | 1 | 1 | 0 | |
| 3 | 4 | 4 | 3 | 3 | 3 | 2 | 1 | 1 | 0 | |
| 6 | 4 | 4 | 3 | 2 | 2 | 2 | 1 | 1 | 0 | |
| 7 | 4 | 3 | 2 | 2 | 2 | 2 | 1 | 1 | 0 | |
| 19 | 4 | 3 | 3 | 2 | 2 | 2 | 1 | 1 | 0 | |
| 15 | 4 | 3 | 3 | 2 | 2 | 2 | 1 | 1 | 0 | |
| 18 | 4 | 4 | 3 | 2 | 2 | 1 | 1 | 1 | 0 | |
| 17 | 4 | 3 | 3 | 2 | 2 | 2 | 1 | 0 | | |
| 5 | 4 | 3 | 2 | 2 | 2 | 2 | 1 | 0 | | |
| 8 | 4 | 3 | 3 | 2 | 2 | 1 | 1 | 0 | | |
| 20 | 4 | 3 | 2 | 2 | 2 | 1 | 1 | 0 | | |
| 1 | 4 | 4 | 3 | 3 | 2 | 1 | 1 | 0 | | |
| 14 | 4 | 4 | 3 | 2 | 2 | 1 | 1 | 0 | | |
| 13 | 4 | 3 | 3 | 3 | 2 | 1 | 0 | | | |
| 12 | 4 | 3 | 3 | 2 | 1 | 0 | | | | |
| 4 | 4 | 3 | 3 | 2 | 1 | 0 | | | | |

**Table 4** Measurements of humeral head diameter and capsule size

| Humeral head diameter | Capsule width |
|---|---|
| 41.5 | 35 |
| 41.5 | 24 |
| 41.5 | 19 |
| 42 | 29 |
| 42 | 25 |
| 42 | 24 |
| 42.5 | 34 |
| 43 | 30 |
| 44 | 33 |
| 44.5 | 35 |
| 53 | 28 |
| 53 | 21 |
| Pearson's coefficient | −0.17 ($p=0.58$) |
| Spearman's coefficient | +0.17 ($p=0.59$) |

The difference between the present results and those in Urayama and Itoi's study [13] should also be discussed. In their study based on arthro-MRI, the length of the glenohumeral ligaments was measured on each slice and compared with the other side. The study focused on traumatic cases and did not take into account the consequences of the Bankart lesion, the exact orientation of the slices regarding the complex shape of the capsule in the rest position cannot be analyzed, and consequently what has been measured remains imprecise.

Only three direct measurements were performed on the harvested capsules. Studying the surface of the capsule would have been of interest but the posterior and anterior limits of the harvested capsule were imprecise and would have induced too large a bias.

The absence of a relationship between the size of the head and the size of the capsule suggests that there is no correlation between the size of the head and the dimensions of the capsular plasty required to obtain an unstable shoulder.

## Conclusion

This first attempt at an experimental anatomic model for anterior chronic shoulder instability raises many questions. This model is suitable only for atraumatic instability [8, 11], which is at present an unsolved therapeutic problem. The present model does not take in account the elasticity of the normal capsule. Regarding the instability, the role of the Bankart lesion compared with the role of the associated capsular laxity remains unknown. This question is of importance since we lack quantitative data that would provide a rational capsular retention during the Bankart procedure. This experimental model may provide useful guidelines in the technique of capsular reduction. Further improvements to the model should allow study of the consequences of laxity limited to the anterior part of the inferior glenohumeral ligament or to the middle glenohumeral ligament. It may also be improved to study the role of thermal capsular plasty.

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