Normal and Impaired Mobility of the Glenohumeral Joint

Anatomical, biomechanical and clinical aspects

Normale en beperkte beweeglijkheid van het schoudergewricht

Anatomische, biomechanische en klinische aspecten

Arthur de Gast

CIP DATA KONINKLIJKE BIBLIOTHEEK, DEN HAAG

de Gast, Arthur
Normal and impaired mobility of the glenohumeral joint.
Thesis, Rotterdam. -With ref.- With Summary in Dutch
ISBN 90-9011711-3
NUGI 742
E-mail: a.degast@wxs.nl

Subjects headings: Human
Joint Instability/pathology
Ligament, Articular/physiology
Shoulder Joint/anatomy
Shoulder Joint/injuries/physiology
Range of Motion

Omslagontwerp: A. de Gast
Omslagfoto: Paula van Alphen
Lijntekeningen: A. de Gast
Digitale bewerking tekeningen: W.H. Groeneveld

Druk: Copynomie®

The publication of this thesis was kindly supported by:
Dupuy-Van Straten Orthopaedische Techniek, Howmedica Nederland, Mathys Medical Nederland B.V., Nederlandse Orthopaedische Vereniging, Orthomed B.V., Oudshoorn Chirurgische Techniek B.V., Smith & Nephew Nederland B.V., Somas Orthopaedie, Stichting Anna-Fonds, Stichting Onderwijs en Onderzoek Opleiding Orthopaedie Rotterdam, Stryker B.V.

Normal and Impaired Mobility of the Glenohumeral Joint

Anatomical, biomechanical and clinical aspects

Normale en beperkte beweeglijkheid van het schoudergewricht

Anatomische, biomechanische en klinische aspecten

PROEFSCHRIFT

ter verkrijging van de graad van doctor
aan de Erasmus Universiteit te Rotterdam
op gezag van de Rector Magnificus
Prof.dr P.W.C. Akkermans M.A.
en volgens het besluit van het College voor Promoties.

De openbare verdediging zal plaatsvinden op
woensdag 17 juni 1998 om 13.45 uur

door

Arthur de Gast

geboren te 's Gravenhage

PROMOTIECOMMISSIE

PROMOTORES:	Prof.dr ir C.J. Snijders Prof.dr J.A.N. Verhaar
CO-PROMOTOR:	Dr R. Stoeckart
LEDEN:	Prof.dr J.Voogd Prof.dr H.J. Stam Prof.dr P.M. Rozing

voor mijn ouders, broers en Dana-Anne

CONTENTS

ABBREVIATIONS

A	*A*cromion
ACJ	*A*cromio-*C*lavicular *J*oint
AxR	*Ax*illary *R*ecess
BG	*B*icipital *G*roove
BT	*B*iceps *T*endon (long head)
C	*C*lavicula
CAA	*C*oraco*A*cromial *A*rc
CAL	*C*oraco*A*cromial *L*igament
CHL	*C*oraco*H*umeral *L*igament
CT	*C*omputerized *T*omography
Dm	*D*eltoid muscle
G	*G*lenoid (fossa)
GH	*G*leno*H*umeral
GHJ	*G*leno*H*umeral *J*oint
GHJC	*G*leno*H*umeral *J*oint *C*apsule
GT	*G*reater *T*uberosity
H	*H*umeral
HH	*H*umeral *H*ead
HR	*H*umeral *R*otation
IGHL(C)	*I*nferior *G*leno*H*umeral *L*igament (*C*omplex)
ISP_m	*I*nfra*sp*inatus *m*uscle
ISP_t	*I*nfra*sp*inatus *t*endon
LT	*L*esser *T*uberosity
MGHL	*M*iddle *G*leno*H*umeral *L*igament
MRI	*M*agnetic *R*esonance *I*maging
PRC	*C*oracoid *Pr*ocess
RC	*R*otator *C*uff
RI	*R*otator *I*nterval
ROM	*R*ange *O*f *M*otion
SA	*S*ub*A*cromial
SASDB	*S*ub*A*cromial-*S*ub*D*eltoid *B*ursa
SC	*Sc*apula
ScS	*Sc*apular *S*pine
SD	*S*ub*D*eltoid
SGHL	*S*uperior *G*leno*h*umeral *L*igament
SHBB	*S*hort *H*ead of the *B*iceps *B*rachii
SL	*S*uperior part of the *G*lenoid labrum
SSC_m	*S*ubs*c*apularis *m*uscle
SSC_t	*S*ubs*c*apularis *t*endon
SSP_m	*S*upra*sp*inatus *m*uscle
SSP_t	*S*upra*sp*inatus *t*endon
TM	*T*eres *M*inor
TT	*T*endon *T*riceps brachii (long head)

GLOSSARY

Frontal plane elevation

Abduction: lateral movement of the arm in the frontal plane about a sagittal axis

Adduction: medial movement of the arm in the frontal plane about a sagittal axis

Sagittal plane elevation

Flexion: forward movement of the arm in the sagittal plane about a frontal axis

Extension: backward movement of the arm in the sagittal plane about a frontal axis

Scapular plane elevation

GH elevation: lateral movement of the arm in the scapular plane about an axis perpendicular to the scapular plane

GH adduction: medial movement of the arm in the scapular plane about an axis perpendicular to the scapular plane

Elevation perpendicular to the scapular plane

GH Flexion: forward movement of the arm in the plane perpendicular to the scapular plane about an axis parallel to the scapular plane

GH Extension: backward movement of the arm in the plane perpendicular to the scapular plane about an axis parallel to the scapular plane

Humeral Rotation

Internal rotation: inward rotation of the humerus about its longitudinal axis

External rotation: outward rotation of the humerus about its longitudinal axis

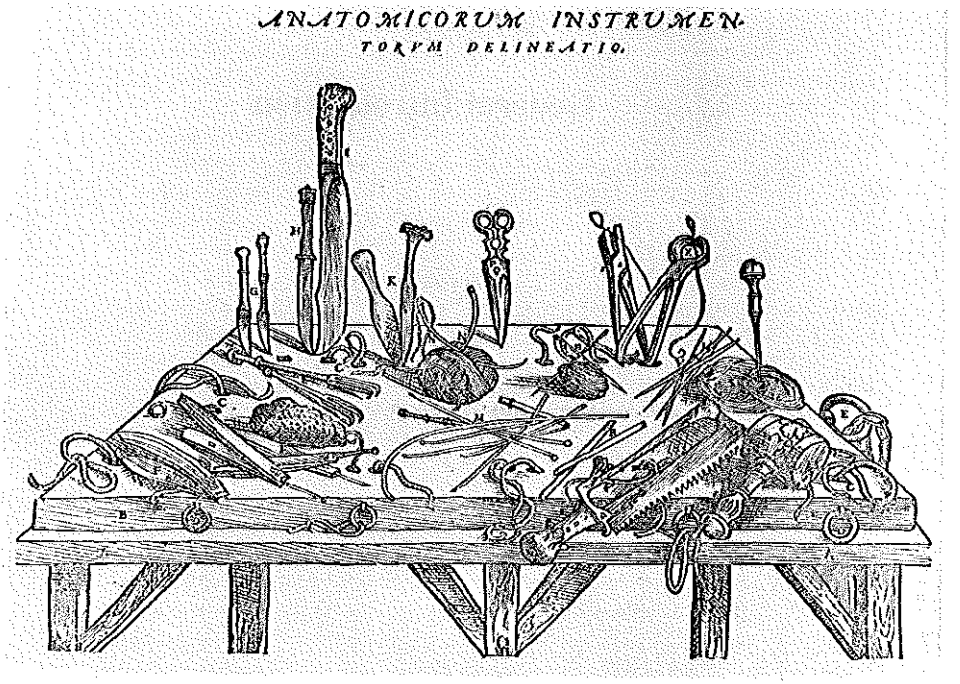
CHAPTER

1

General Introduction

ARTHUR DE GAST, MD¶

¶*Dept. of Anatomy, Faculty of Medicine, Erasmus University Rotterdam*


ANATOMICORUM INSTRUMEN-
TORVM DELINEATIO.

CHAPTER

1

General Introduction

In the field of orthopaedics, extensive knowledge exists on the diagnosis and treatment of skeletal diseases, e.g. developmental anomalies, fractures and osteoarthritis. Less knowledge is available on soft tissue diseases. The main reason for this difference of knowledge concerns the imaging techniques. First, the introduction of clinical radiography at the end of the last century made it possible to obtain images of the human skeleton *in vivo*. However, the technique was unsuitable for the soft tissues. About 80 years later, imaging of soft tissue structures *in vivo* with satisfactory image resolution became possible after the introduction of sonography and magnetic resonance imaging. A second reason for the difference in knowledge is that load transfer in bone is easier comprehensible than in soft tissue.

Certain soft tissue regions of the body have been studied in more detail than others. Compared to the knee joint,[33] less clinical and experimental knowledge is available on the role of the articular soft tissues in normal and pathological movement of the glenohumeral joint (GHJ). This has two reasons. First, the GHJ joint is relatively difficult to access at clinical examination. Second, assessment of GHJ motion is relatively difficult because of the multiple degrees of freedom of motion due to the large number of shoulder girdle joints.

The GHJ is one of the principal sites of pathology in sports injuries,[8, 16, 19, 20, 23, 26, 28, 31] work-related injuries,[10, 11] osteoarthritis and age-related degeneration.[3] During the last two decades important progress has been made in the understanding of the mechanisms of GHJ stability and rotator cuff degeneration, and of the basic functions of the GHJ. In spite of these advances, the following statement made by Burns and Ellis in 1937 still holds today: 'Painful shoulders form an important part of orthopaedic practice, but their obscurity, uncertain prognosis and the fact that they present so few definite signs and symptoms,

render their classification into types difficult on clinical grounds."[1] In our opinion the main reason for this difficulty is the lack of adequate anatomical and biomechanical modeling of the normal shoulder and of the shoulder in pathological conditions. It is generally accepted that thorough knowledge of the anatomy forms the basis for understanding shoulder physiology and pathology. Anatomical studies provide useful data for better insight in the etiology, diagnosis, treatment and prevention of shoulder diseases. However, it can be questioned whether traditional topographical anatomy alone offer these insights. Topographical anatomy provides descriptions of soft tissue structures based on their shape (rhomboid muscle), size (latissimus dorsi muscle), position (plantaris muscle), and their supposed function (abductor hallucis muscle). Furthermore, descriptions of *movement* derived from topographical anatomy are mostly based on one *position*: the anatomic position. Symptoms of shoulder pathology that are usually provoked by certain motion patterns,[36] cannot be fully understood on the basis of topographical anatomy alone.

Pain, limited range of motion and lack of joint stability are the major symptoms in shoulder diseases. Historically, one single structure at a time was held responsible for a painful shoulder. To give an example, Duplay introduced the term *periarthitis humeroscapularis* (PHS) to describe a disease entity characterized by pain and stiffness of the shoulder following trauma.[4] Based on a single patient, he assumed the cause was destruction or fusion of the subacromial bursa. According to his opponents PHS should be regarded as a rheumatic affection or neuritis.[21, 29] Application of X-rays showed soft tissue calcifications between the greater tuberosity and the acromion, which were termed *bursitis calcarea subacromialis*.[32] Later, these soft tissue calcifications were completely identified with PHS.[21] Nowadays PHS usually refers to a painful condition of the shoulder concerning either the subacromial-subdeltoid bursa, the rotator cuff or the tendon of the long head of the biceps brachii muscle (biceps tendon), without emphasis on one of these structures.

During the late 1970s and the 1980s, there was an explosion of interest in diseases of the shoulder and important advances were made in the treatment of pathologic conditions such as GHJ instability, rotator cuff disease and osteoarthritis of the GHJ. However, despite the fact that these treatments, especially surgical procedures, were explicitly designed to restore normal shoulder mechanics, little is known of normal GH and subacromial articular geometry and kinematics. Even less is known of the biomechanics present in disease states or the alteration in biomechanics produced by surgical reconstruction. In pathologic conditions, such as a contracture (of a part) of the joint

capsule, the anatomy of the joint changes and, consequently, new mechanical properties are expected to develop.

For performing the present study we had three reasons. First, topographical anatomy reduces the morphological and the functional properties of the periarticular soft tissues of the GHJ too strongly. Many conclusions concerning the role of soft tissue elements in the normal motion of the GHJ *in vivo* are derived from this 'component' anatomy. As a result, reduction of the functional properties cannot be avoided, but there is another, less obvious problem. The properties of the single components do not apply to the GHJ as a whole. For instance, the lack of GHJ stability is frequently attributed to the marked lack of congruence between the subchondral bone surfaces of the humeral head and the glenoid fossa. However, in the presence of articular cartilage and the glenoid labrum, the actual articulating surfaces do conform.

Second, since mechanical functions of GH soft tissue structures are strongly GHJ position-dependent, their role in normal mobility and stability of the GHJ can only be appreciated when analyzed through a complete range of motion. Earlier studies were carried out either over a limited range of motion or in a limited number of GHJ positions.[2, 9, 12, 13, 15, 22, 24, 25, 30, 34, 35]

Third, theoretical studies, with the use of biomechanical computer models,[5-7, 14, 17, 27] address the shoulder in a sophisticated way, and yield valuable data on muscle forces and joint load, but they need to be validated with anatomical data and biomechanical experiments.

The present study deals with the anatomy and biomechanics of the GHJ and is primarily an experimental approach with the use of embalmed and unembalmed human specimens. The use of unembalmed human specimens has several restrictions (pressure of time, possibility of infectious disease etc.). The use of embalmed specimens has the advantage that the mechanical properties remain constant over a long period, which enables for repeated measurements and time-consuming complex modifications. Disadvantages of the use of embalmed specimens, such as decreased tissue elasticity, are of less significance when the kinematic role of nerves, tendons, ligaments and joint capsule are studied. For example, it has been demonstrated recently that the tensile forces due to joint motion in peripheral nerves obtained from embalmed specimens are positively correlated with those of unembalmed specimens.[18] An obvious advantage of the use of human specimens over mathematical models is that, the real situation of load transfer and mobility can be approximated more closely.

The study confines to the subacromial-subdeltoid bursa, the biceps tendon and the joint capsule. We focused on glenohumeral (GH) elevation in the scapular plane, and humeral

rotation (HR: either internal or external rotation of the humerus), which are generally accepted as the most functional directions. These choices of directions of movement and of the above mentioned structures are justified by the aim of this study.

The aim of this study is threefold. First, to assess the functional anatomy of the subacromial-subdeltoid bursa in relation to the following questions. How are bursal transformations controlled during movements of the GHJ and what are the consequences of these transformations for the evaluation and treatment of GHJ disorders involving the subacromial-subdeltoid bursa? Second, to determine how articular soft tissue structures contribute to the normal pattern of scapular plane elevation and HR. Third, to assess the effects of (surgical) modification of GHJ capsule length on the range of GH elevation and HR.

1.1 Outline of the thesis

In *Chapter 2*, a comprehensive outline of shoulder anatomy is provided, based on a survey of the literature.

In *Chapter 3* the results of an anatomical and kinematic study of the subacromial-subdeltoid mechanism are presented.

Chapter 4 deals with aspects of normal mobility of the GHJ. With the use of a custom-made device and human shoulder specimens, the range of HR through a complete arc of GH elevation in the scapular plane is measured. Special attention is paid to the relationship between GH elevation and HR.

In *Chapter 5* the results of a study on the position-dependent function of GH periarticular soft tissue structures is presented. In this study we assessed the role of the tendon of the long head biceps brachii muscle in the control of HR and guidance of GH elevation to the position of maximal GH elevation.

Chapter 6 addresses the topic of limited GH range of motion in disease states and as a result of surgical intervention. To assess the potential effects of capsular reconstruction on GH elevation and HR, the length of five regions of the GHJ capsule was altered systematically.

In *Chapter 7* the results of the anatomical-biomechanical studies their practical are discussed in view of their consequences.

REFERENCES

1. **Burns BH, Ellis VH.** Recent advances in orthopaedic surgery. London: J and A Churchill Ltd, 1937:151.
2. **Cain PR, Mutschler TA, Fu FH, Lee SK.** Anterior stability of the glenohumeral joint. A dynamic model. *Am J Sports Med* 1987;15(2):144-8.
3. **Cofield RH.** Degenerative and arthritic problems of the glenohumeral joint. In: Rockwood CA, Matsen FA, eds. The shoulder. Philadelphia London Toronto: W.B. Saunders Company, 1990:678-749. vol 2.
4. **Duplay S.** De la peri-arthrite scapulohumerale et de raideurs de l'epaule qui en sont la consequence. *Arch Gen Med* 1872;20:513-25.
5. **Engin AE, Chen SM.** Statistical database for the biomechanical properties of the human shoulder complex--I: Kinematics of the shoulder complex. *J Biomech Eng* 1986;108(3):215-21.
6. **Engin AE, Peindl RD.** On the biomechanics of human shoulder complex--I. Kinematics for determination of the shoulder complex sinus. *J Biomech* 1987;20(2):103-17.
7. **Engin AE, Tumer ST.** Three-dimensional kinematic modelling of the human shoulder complex-- Part I: Physical model and determination of joint sinus cones. *J Biomech Eng* 1989;111(2):107-12.
8. **Fleisig GS, Andrews JR, Dillman CJ, Escamilla RF.** Kinetics of baseball pitching with implications about injury mechanisms. *Am J Sports Med* 1995;23(2):233-9.
9. **Gagey O, Bonfait H, Gillot C, Hureau J, Mazas F.** Anatomic basis of ligamentous control of elevation of the shoulder (reference position of the shoulder joint). *Surg Radiol Anat* 1987;9(1):19-26.
10. **Hagberg M.** ABC of work related disorders. Neck and arm disorders. *Bmj* 1996;313(7054):419-22.
11. **Hagberg M, Kvarnstrom S.** Muscular endurance and electromyographic fatigue in myofascial shoulder pain. *Arch Phys Med Rehabil* 1984;65(9):522-5.
12. **Harryman DTd, Sidles JA, Harris SL, Matsen FAd.** The role of the rotator interval capsule in passive motion and stability of the shoulder. *J Bone Joint Surg [Am]* 1992;74(1):53-66.
13. **Helmig P, Sojbjerg JO, Kjaersgaard-Andersen P, Nielsen S, Ovesen J.** Distal humeral migration as a component of multidirectional shoulder instability. An anatomical study in autopsy specimens. *Clin Orthop* 1990(252):139-43.
14. **Hogfors C, Sigholm G, Herberts P.** Biomechanical model of the human shoulder--I. Elements. *J Biomech* 1987;20(2):157-66.
15. **Howell SM, Galinat BJ, Renzi AJ, Marone PJ.** Normal and abnormal mechanics of the glenohumeral joint in the horizontal plane. *J Bone Joint Surg [Am]* 1988;70(2):227-32.
16. **Johnson JE, Sim FH, Scott SG.** Musculoskeletal injuries in competitive swimmers. *Mayo Clin Proc* 1987;62(4):289-304.
17. **Karlsson D, Peterson B.** Towards a model for force predictions in the human shoulder. *J Biomech* 1992;25(2):189-99.
18. **Kleinrensink GJ, Stoeckart R, Vleeming A, Snijders CJ, Mulder PGH, Wingerden van JP.** Peripheral nerve tension due to joint motion. A comparison bewteen embalmed and unembalmed human bodies. *Clin Biomech* 1995;10(5):235-39.
19. **Kocher MS, Feagin JA, Jr.** Shoulder injuries during alpine skiing. *Am J Sports Med* 1996;24(5):665-9.

20. **Maffet MW, Gartsman GM, Moseley B.** Superior labrum-biceps tendon complex lesions of the shoulder. *Am J Sports Med* 1995;23(1):93-8.
21. **Matsen FA, Arntz CT.** Subacromial impingement. In: Rockwood CA, Matsen FA, eds. The shoulder. Philadelphia: W.B. Saunders, 1990:623-46. vol 2.
22. **Neer CSd, Satterlee CC, Dalsey RM, Flatow EL.** The anatomy and potential effects of contracture of the coracohumeral ligament. *Clin Orthop* 1992(280):182-5.
23. **Neviaser TJ.** Weight lifting. Risks and injuries to the shoulder. *Clin Sports Med* 1991;10(3):615-21.
24. **O' Brien S, Neves MC, Arnoczky SP, et al.** The anatomy and histology of the inferior glenohumeral ligament complex of the shoulder. *Am J Sports Med* 1990;18(5):449-56.
25. **Ovesen J, Nielsen S.** Anterior and posterior shoulder instability. A cadaver study. *Acta Orthop Scand* 1986;57(4):324-7.
26. **Payne LZ, Altchek DW.** The surgical treatment of anterior shoulder instability. *Clin Sports Med* 1995;14(4):863-83.
27. **Peindl RD, Engin AE.** On the biomechanics of human shoulder complex--II. Passive resistive properties beyond the shoulder complex sinus. *J Biomech* 1987;20(2):119-34.
28. **Penny JN, Welsh RP.** Shoulder impingement syndromes in athletes and their surgical management. *Am J Sports Med* 1981;9(1):11-5.
29. **Pettersson G.** Rupture of the tendon aponeurosis of the shoulder joint in antero-inferior dislocation. *Acta Orthop Scan (Suppl.)* 1942;77:1-182.
30. **Rodosky MW, Harner CD, Fu FH.** The role of the long head of the biceps muscle and superior glenoid labrum in anterior stability of the shoulder. *Am J Sports Med* 1994;22(1):121-30.
31. **Smith AM, McCauley TR, Jokl P.** SLAP lesions of the glenoid labrum diagnosed with MR imaging. *Skeletal Radiol* 1993;22(7):507-10.
32. **Stieda A.** Zur Patholgie de Schultergelenkschleimbeutel. In: Langenbeck B, ed. Archiv fur Klinische Chirurgie. Berlin: Verlag von August Hirschwald, 1908:910.
33. **Strobel M, Stedtfeld HW.** Diagnostic evaluation of the knee. First ed. Berlin Heidelberg New York: Springer Verlag, 1990.
34. **Turkel SJ, Panio MW, Marshall JL, Girgis FG.** Stabilizing mechanisms preventing anterior dislocation of the glenohumeral joint. *J Bone Joint Surg [Am]* 1981;63(8):1208-17.
35. **Warner JJ, McMahon PJ.** The role of the long head of the biceps brachii in superior stability of the glenohumeral joint. *J Bone Joint Surg [Am]* 1995;77(3):366-72.
36. **Zuckerman JD, Mirabello SC, Newman D, Gallagher M, Cuomo F.** The painful shoulder: Part II. Intrinsic disorders and impingement syndrome. *Am Fam Physician* 1991;43(2):497-512.

CHAPTER

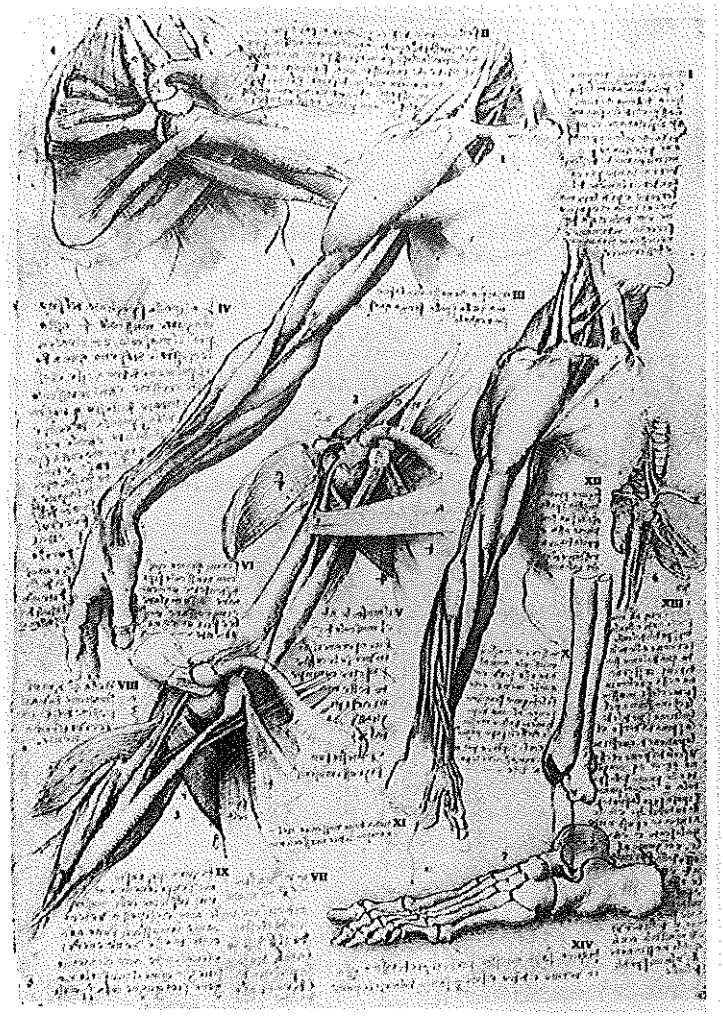
2

Anatomy of the Glenohumeral Joint

DESCRIPTIVE, APPLIED, AND FUNCTIONAL ANATOMY

ARTHUR DE GAST, MD¶ AND ROB STOECKART, PH.D.¶

¶Dept. of Anatomy, Faculty of Medicine, Erasmus University Rotterdam



CHAPTER

2

Anatomy of the Glenohumeral Joint

2.1 Introduction

For a detailed knowledge of the shoulder we are largely indebted to the early investigators Galen and Vesalius. Modern investigations have deepened the understanding of this earlier research, rather than totally altering it. Anatomy (of the shoulder) serves as a tool for the interpretation of clinical sign and symptoms. So, to make a correct diagnosis in patients with complaints in the shoulder region, adequate knowledge of the composing structures is essential. However, mostly this knowledge concerns descriptive anatomy. This component anatomy is based mostly on differences in tissue characteristics. Physical tests based merely on topographical anatomy easily result in oversimplification of patient examination. This may be useful for daily practice but will inevitably lead to the appreciation of only a limited number of signs and symptoms. Still, topographical anatomy is necessary for a reliable three-dimensional representation of the composing structures. Otherwise, it is not possible to comprehend the more complex functional interrelations.

The stimulus for shoulder research comes from four sources at least: 1) the discovery of a new disease (posterior superior glenoid impingement [44]), 2) the invention of a new treatment (arthroscopic acromioplasty [21, 22, 29]), 3) technical advances in imaging techniques (sonography [54] and magnetic resonance imaging [47, 48]), and 4) the arrival of a new method of studying anatomy (rapid-sequence photography, selective cutting studies [17]). For example, the introduction of arthroscopic shoulder surgery has sent a 'generation of surgeons' back to the anatomy laboratory (or at least back to books dealing with this subject).

Although we assume that the readers know a great deal about the anatomy of the shoulder in this chapter, its descriptive anatomy will be reviewed. For a better understanding of the functional connection of the composing structures, detailed interrelationships are presented. This chapter focuses primarily on those structures that are subject of the experimental studies as described in chapter 3 to 6. The nerves and blood vessels will not be considered in this chapter.

2.2 Descriptive and Applied Anatomy

2.2.1 Anatomical planes, axes and directions

Three major anatomical planes are used in the description of (shoulder) anatomy. These are the frontal, sagittal and horizontal planes. The anatomic position is used as a standard for the description of directions and main axes of movement (Figure 2.1).

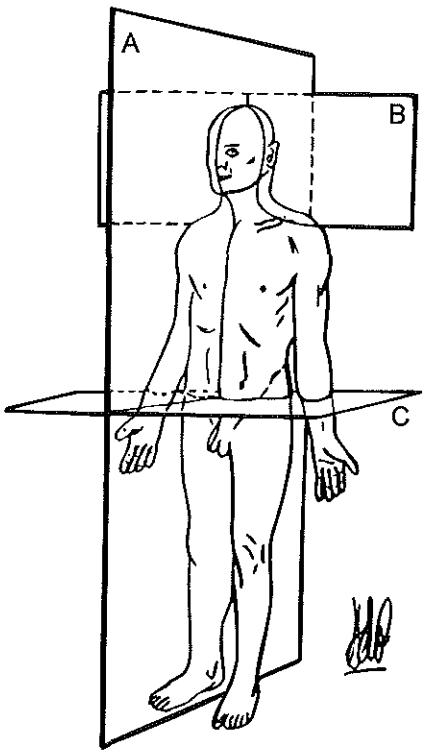


Figure 2.1 Anatomical planes and main axes of movement, referring to the anatomic position (Modified and redrawn from Morris' Anatomy, 1966) A=sagittal plane; B=frontal plane; C=horizontal plane

2.2.2 Bony anatomy

Three bones make up the osseous anatomy of the shoulder girdle; scapula, clavicle and humerus. Together they form three diarthrodial joints: the glenohumeral, acromioclavicular and sternoclavicular. In combination with the fascial spaces of the scapulothoracic mechanism they account for the great range of motion (ROM) of the shoulder. In the framework of this thesis, special emphasis is placed here upon the gross anatomy of the scapula, coracoacromial arc, and proximal humerus.

2.2.2.1 Scapula

The scapula is a thin triangular bone, which is suitable for the attachment of the many scapular muscles and for moving along the curved chest wall. It has ventral and dorsal surfaces, lateral and medial borders, inferior, superior and lateral angles, and three bony processes, the scapular spine, its continuation the acromion, and the coracoid process. The lateral border ends superiorly at the glenoid fossa for articulation with the head of the humerus. The shape of the glenoid fossa compares to an inverted comma: its superior portion is narrow and its inferior portion broad. The dorsal surface of the scapula is interrupted by the scapular spine. The coracoid process projects from the base of the glenoid neck in a lateral and anterior direction. It serves as the origin of the coracobrachialis and short head of the biceps brachii tendons, the coracoacromial and coracoclavicular ligaments and, additionally, it provides an insertion site for the pectoralis minor muscle.

2.2.2.2 Coracoacromial arc and acromioclavicular joint

Typically, descriptions of the coracoacromial arc refer to the acromion, coracoid process and coracoacromial ligament. Since most structural pathology of the shoulder involves the subacromial soft tissues, the acromion is the most studied process of the scapula.[2, 6, 19, 60, 87, 96] Lesions of the subacromial soft tissues, due to compression and friction against parts of the coracoacromial arc, relate to an area called the supraspinatus outlet (Figure 2.2ab).[58] The acromion forms a large part of the roof of this outlet. The remainder of the supraspinatus outlet is composed of the coracoacromial ligament and coracoid process. The medial border of this outlet is defined at the supraglenoid tubercle. Laterally of the supraglenoid tubercle, the proximal humerus forms the floor of the supraspinatus outlet. The term supraspinatus outlet may be confusing since in addition to the supraspinatus, also the tendon of the long head of the biceps brachii muscle (biceps tendon), portions of the subacromial-subdeltoid bursa (SASDB) and the glenohumeral joint capsule (GHJC) occupy the supraspinatus outlet. Medially in the

supraspinatus outlet, abundant fat tissue protects the suprascapular neurovascular bundle.[90] Most likely, it also cushions the supraspinatus muscle-tendon unit, allowing muscle expansion during contraction. Laterally in the supraspinatus outlet, less fat is present, limiting volume compensation in case of inflammatory soft tissue swelling. Due to the morphology of the greater tuberosity, the volume of the supraspinatus outlet increases on internal rotation of the humerus and decreases on external rotation.[60, 71]

The coracoacromial ligament covers a large part of the inferior surface of the acromion.[63] Although the coracoacromial ligament connects two parts of the same bone, it has a distinct role in transferring forces through the scapula, acting as a brace between the acromion and coracoid process (Figure 2.2a).[72]

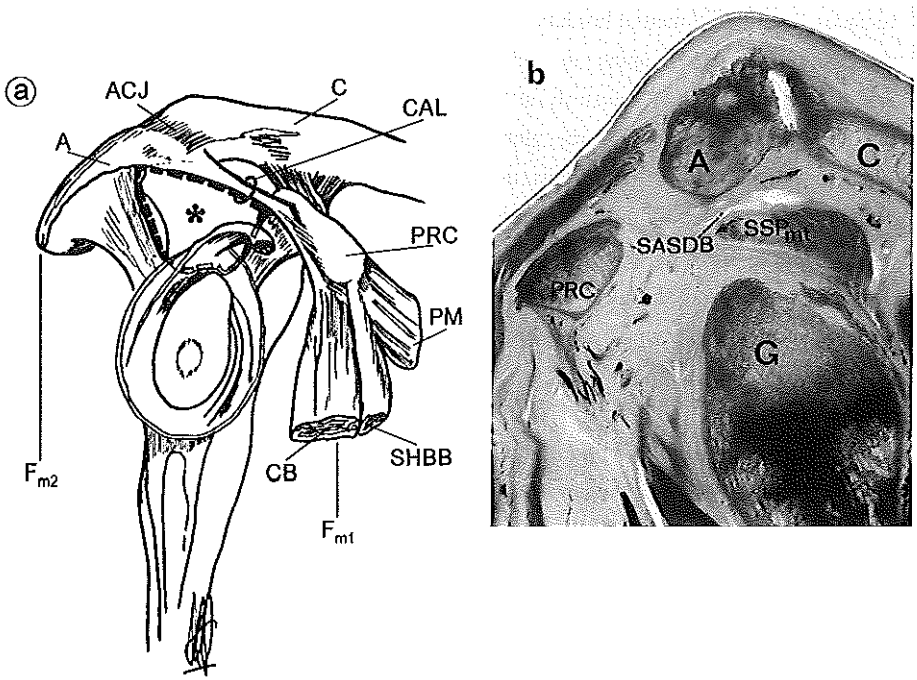


Figure 2.2a Supraspinatus outlet of the right shoulder, anterolateral view. *Supraspinatus outlet, boundaries marked with heavy dotted line; A=acromion; ACJ=acromioclavicular joint; C=clavicle; CAL=coracoacromial ligament; CB=coracobrachialis muscle; PRC=coracoid process; PM=pectoralis minor muscle; SHBB=short head of the biceps brachii muscle; F_{m1}=muscle action representing the line of action of the coracobrachial, pectoralis minor and short head of the biceps muscles forming a force couple with F_{m2} representing the line of action of the deltoid and lower trapezius muscles. The coracoacromial ligament serves as a brace between F_{m1} and F_{m2}.

Figure 2.2b Plastinated coronal cross-section through the supraspinatus outlet of the right shoulder at the level of the acromioclavicular joint. It shows the intimate spatial relationship between the acromioclavicular joint, subacromial-subdeltoid bursa and supraspinatus tendon-muscle unit. G=glenoid; SASDB=subacromial-subdeltoid bursa; SSP_{m1}=supraspinatus muscle-tendon unit. For additional legend see Figure 2.2a. For information on the method of plastination see Chapter 3, section 3.2.1.

With few exceptions, the acromioclavicular joint (ACJ) is the only articulation between the clavicle and the scapula. In about 1% of individuals a coracoclavicular joint or bony connection exist.[31] Since no muscles cross the ACJ, its stability depends on the integrity of the acromioclavicular and coracoclavicular ligaments.[27, 75] Clinically, it is important to recognize the role of the ACJ in patients with impingement syndrome. The ACJ has an intimate spatial relation with the rotator cuff and parts of the SASDB (Figure 2.3). Therefore, swelling of the ACJ, hypertrophy of its inferior capsule, and inferior osteophyt formations (see Figure 2.3) are well-established causes of acquired narrowing of the supraspinatus outlet. It has been emphasized that failure to recognize this role of the ACJ in the impingement syndrome can compromise the result of surgical subacromial decompression.[33, 67, 86, 94]

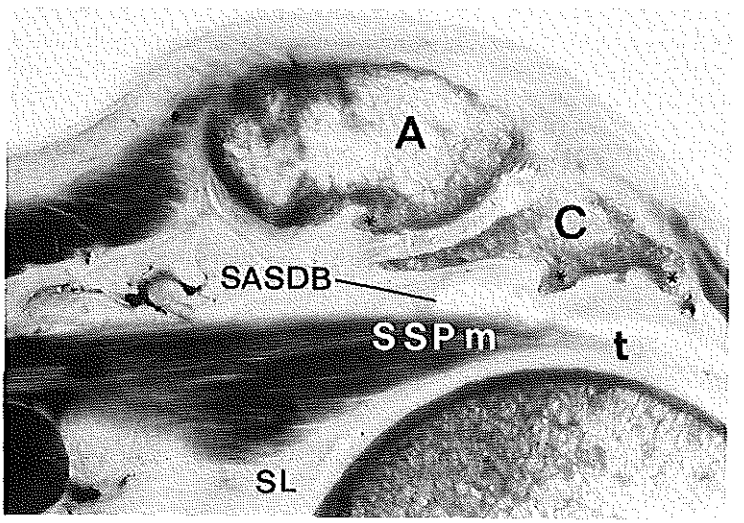


Figure 2.3 Plastinated frontal section through the left acromioclavicular joint (ACJ). It shows the intimate spatial relationship between the ACJ, supraspinatus muscle tendon unit, and parts of the subacromial-subdeltoid bursa. Osteofyt formation at the inferior surface of the ACJ compromises the supraspinatus outlet. A=acromion; C=clavicula; HH=humeral head; SASDB=subacromial-subdeltoid bursa; SL=superior part of the glenoid labrum; SSP_m=supraspinatus muscle; SSP_t=supraspinatus tendon; *=osteophyt formation.

2.2.2.3 Proximal humerus

The subchondral bone surface of the humerus is an irregular spheroid with a mean radius of curvature of approximately 2.3-2.5 cm [40, 82] that is directed medially, superiorly and posteriorly. The posterior torsion of the humeral head compared to the epicondylar axis of the distal humerus is called retroversion (Figure 2.4). Although the retroversion angle is supposed to be 30 to 45°, considerable variation of this angle has been reported using plain radiographs or computerized tomography (CT).[51, 85]

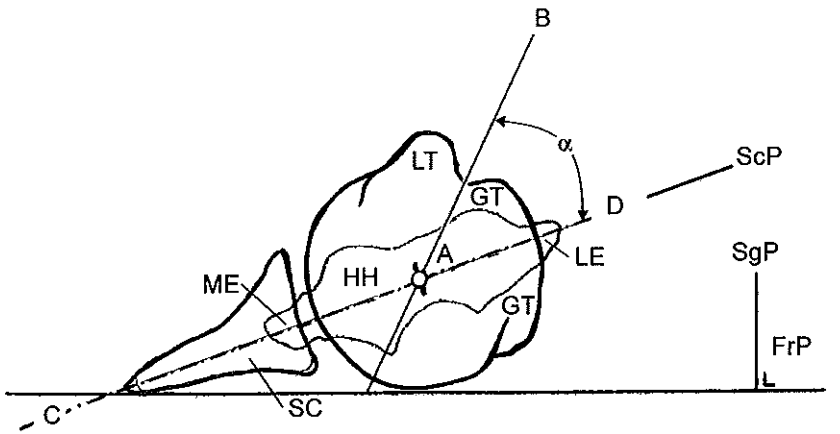


Figure 2.4 Superior view of the humeral head projected over the distal humeral condyles. A line (A-B) drawn through the center of the humeral head to the center of the bottom of the bicipital groove, intersecting the interepicondylar line (C-D) accurately depicts the retroversion (α) of the humeral head. C-D coincides with the scapular plane. ScP indicates the scapular plane; FrP the frontal plane; SgP the sagittal plane. GT=greater tuberosity; HH=humeral head; LE=lateral epicondyl; LT=lesser tuberosity; ME=medial epicondyl; SC=scapula.

The anatomical neck separates the articular cartilage of the humeral head from the attachments of the rotator cuff tendons and GHJC. It varies largely in width from about 1 cm on the anterior, medial and posterior side of the humerus to being hardly detectable on the superior surface. Normal variations such as the anatomic bare area[α] can easily be mistaken for erosions or superficial impression fractures (Hill-Sachs lesion[α] or reversed Hill-Sachs lesion).[63] Traditionally, descriptions of the bicipital groove have focused on its anterior-posterior depth,[16] and on the variability of medial wall angulation (Figure 2.5).[32, 37]

2.2.3 Glenohumeral joint

The glenohumeral joint (GHJ) is the synovial articulation between the humeral head and the glenoid fossa. Earlier studies have suggested lack of congruency of the glenoid fossa and the humeral head (Figure 2.6a).[76, 77] Although clear differences exist between the curvatures of the macerated humeral head and the glenoid fossa, these differences only

[α] The humeral head is characteristically round and shows posteriorly a 'bald spot,' which represents an area of bare bone between the attachment of the posterior capsule and the edge of the articular surface.[18] It is thought to have little functional significance. Hill-Sachs lesion is defined as a humeral head fracture created by dislocation of the humeral head with impression of the anterior glenoid rim on the posterior aspect of the humeral head. This defect characteristically lies in the posterior superior surface of the humeral head and does not have the foramina typically seen in the anatomic bare area.[63]

relate to the subchondral surfaces of macerated bones.[82] Anatomic cross-sections, stereophotogrammetry, CT and magnetic resonance imaging (MRI) show congruency of the articular surfaces of the humerus and glenoid (Figure 2.6b). Hence, instability attributed to the joint based on the shape of the 'naked' bones is not correct. The lack of stability of the GHJ in comparison with the hip, for example, can be attributed to the relatively small surface area of the glenoid fossa that does not enclose the humeral head. Kinematic analyses using subchondral bone contours, such as obtained from plain radiographs, significantly overestimate the actual translations of the humeral head.[5] Therefore, MRI studies enabling visualization of articular cartilage provide a better appreciation of glenohumeral (GH) congruency, and will allow for a more correct description of *in vivo* GH kinematics.[5]

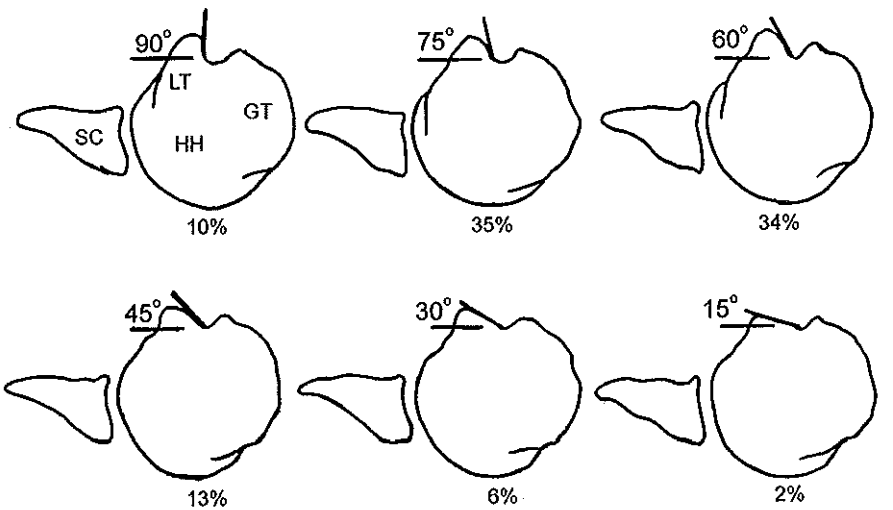


Figure 2.5 Humans are unique in having variations in the medial wall angle of the bicipital groove. The medial wall angle in other primates is constant within the species. Variability of the lateral wall angle has not been studied in detail. HH=humeral head; GT=greater tuberosity; LT=lesser tuberosity; SC=scapula. (Modified and redrawn from Hitchcock HH and Bechtol CO, 1948)

The glenoid labrum, a rim of fibrous tissue that is triangular in cross-section,[95] and the articular surface combine to create a socket that is approximately 9 mm deep in the superior inferior direction and 5 mm deep in the anterior posterior direction.[53] The circular, pliable, glenoid labrum contributes approximately 50% of the total depth of the articular glenoid socket.[38, 53] Its relationship with the origin of the biceps tendon will be discussed in section 2.2.7.

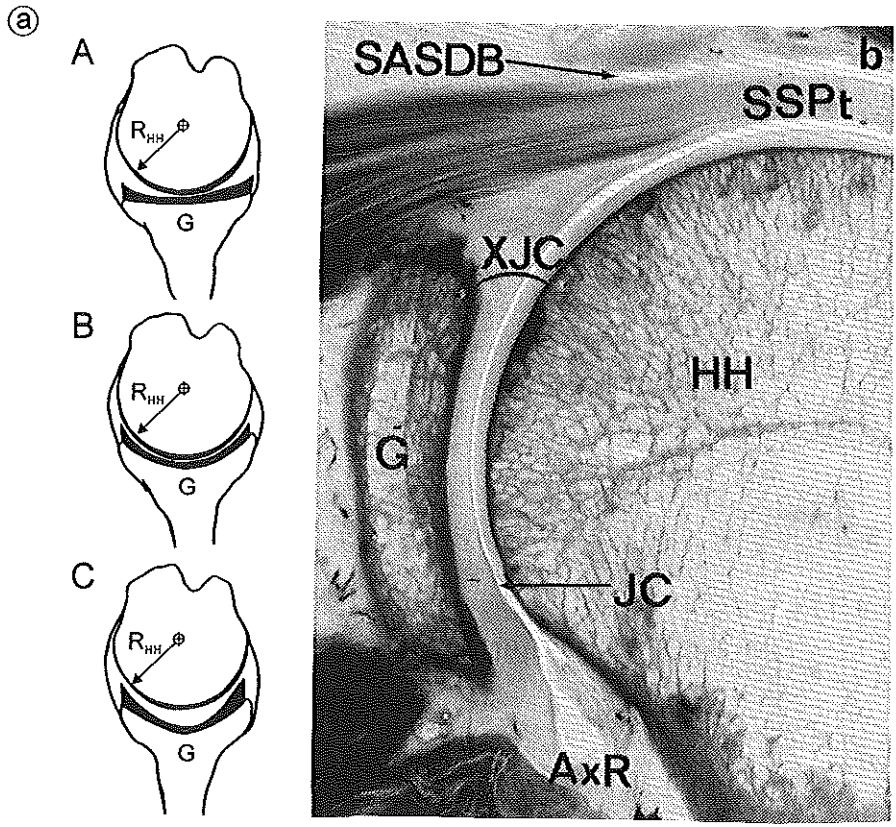


Figure 2.6a. Based on studies on the radius of curvature of the humeral head and the glenoid fossa in 20 shoulders, Saha [76] classified the GH articular surface into three types: Type A, in which the humeral surface has a radius of curvature smaller than that of the glenoid fossa; Type B, in which the humerus and glenoid fossa have similar curves; and Type C, in which the humerus has a radius of curvature larger than that of the glenoid fossa. R_{HH}=radius of curvature of the humeral head; G=glenoid. (Modified and redrawn from Saha, 1967)

Figure 2.6b. Anterior view of a frontal cross-section through the left glenohumeral joint.
AxR=axilary recess; G=glenoid fossa; HH=humeral head; JC=joint cavity; SASDB=subacromial-subdeltoid bursa; SSP_t=supraspinatus tendon; XJC=radiological 'joint space'.

The humeral head is partly covered by the structures that make up the coraco-acromial arc. The GHJC originates from the border of the glenoid cavity and adjacent glenoid rim, covers the humeral head and inserts near the anatomical neck of the humerus. The spheroid geometry of the articular surfaces, their humeral head-to-glenoid surface area ratios (3:1 to 4:1),[82, 71] and the loose GHJC account for the large mobility of the GHJ. The superior, anterior and inferior regions of the GHJC are consolidated by fibrous bands usually called capsular ligaments: the coracohumeral and GH ligaments.[25, 95] These

ligaments were first described as mere thickenings in the GHJC.[78] Clinical and experimental studies showed the functional importance of these structures in maintaining GHJ joint stability.[88, 95] The GH ligaments show great variation in size, shape, thickness, and attachment site (Figure 2.7).[25, 64, 92]

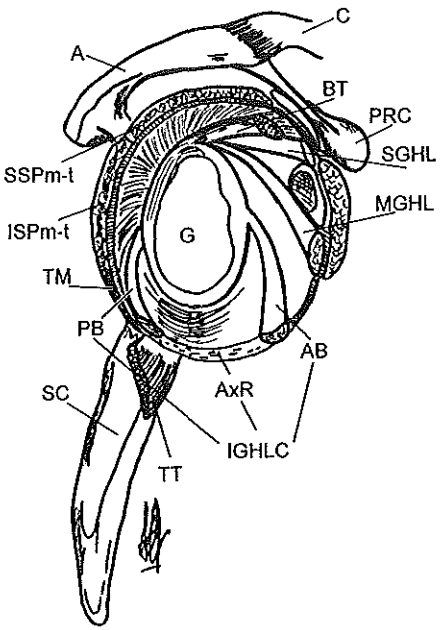


Figure 2.7 Lateral view of the right GHJ shows the most common arrangement of the origins of the GH ligaments. A=acromion; AB=anterior band of the IGHLC; AxR=axillary recess; BT=biceps tendon; C=clavicula; G=glenoid fossa; IGHLC=inferior glenohumeral ligament complex; ISP_{m-t}=infraspinatus muscle-tendon unit; MGHL=middle glenohumeral ligament; PB=posterior band of the IGHLC; PRC=coracoid process; SC=scapula; SGHL=superior glenohumeral ligament; SSP_{m-t}=supraspinatus muscle-tendon; TM=teres minor; TT=origin tendon of the triceps brachii muscle (long head). (Modified and redrawn after O'Brien, 1990)

2.2.3.1 Coracohumeral and superior glenohumeral ligaments

The coracohumeral and superior glenohumeral ligaments are considered the most constant ligamentous structures of the GHJC.[20, 36, 61, 62] Each of these ligaments has a separate origin and insertion.[65]

The *coracohumeral ligament* (CHL) originates from the base and lateral border of the coracoid process, passes the humeral head and inserts into the greater and lesser tuberosities. Its role in limiting external rotation of the humerus, and preventing inferior and posterior dislocation of the humeral head, has been established.[20, 36, 61]

The *superior glenohumeral ligament* (SGHL) originates anterior of the supraglenoid tubercle and the adjacent glenoid labrum, crosses the floor of the rotator interval (see section 2.2.5) and inserts into the superior surface of the lesser tuberosity (fovea capitis). Its presence in shoulder specimens ranges from 90 to 100%.[18, 25, 65] The size of the SGHL is quite small, and its role in stabilizing the GHJ is modest. However, the SGHL has an important role in guiding GH elevation.[28]

2.2.3.2 Middle glenohumeral ligament

The middle glenohumeral ligament (MGHL) shows the most variation in size and is not present as frequently as the other GH ligaments.[18, 56] In the young, the MGHL can be thick as the biceps tendon (5 to 8 mm);[8] it becomes thinner with age.[25] Absence has been reported in as much as 27% of cases.[65] It originates from the glenoid labrum below the SGHL, passes the humeral head in an anterior to inferior direction and inserts into the lesser tuberosity together with the subscapularis tendon. Its variable appearance makes its contribution to static GH stability likewise variable. Selective cutting studies suggest that it acts as a secondary restraint to anterior translation of the humeral head in case of an attenuated or torn anterior band of the inferior glenohumeral ligament complex (IGHLC; see section 2.2.3.3).[17] The MGHL also restrains external rotation, mainly between 60 to 90° GH abduction.[25, 66]

2.2.3.3 Inferior glenohumeral ligament complex

According to O'Brien et al.(64) the inferior glenohumeral ligament (IGHL) includes an anterior band, posterior band and interposed axillary pouch (Figure 2.7). Therefore, the denomination *inferior glenohumeral ligament complex* (IGHLC) has been proposed.[64] Original descriptions of the IGHL mention a triangular shaped structure with its apex at the anterior glenoid labrum, its base blending with the capsule between the subscapularis tendon and the origin of the long head of the triceps brachii.[18] The anterior band of the IGHLC has been observed by Turkel et al.[88] The IGHLC originates from either the glenoid labrum or adjacent glenoid neck and inserts into the inferior portions of anatomical neck of the humerus. The IGHLC acts as a GH elevation-dependent restraint. At 90° GH elevation, the IGHLC 'cradles the humeral head like a hammock' with internal and external rotation causing tightening of the anterior and posterior band, respectively.[92]

2.2.4 The scapulohumeral muscle-tendon units

In addition to the GH ligaments, the insertion tendons of the four rotator cuff muscles (subscapularis, supraspinatus, infraspinatus and teres minor tendons) provide further consolidation of the GHJC (Figure 2.8ab).

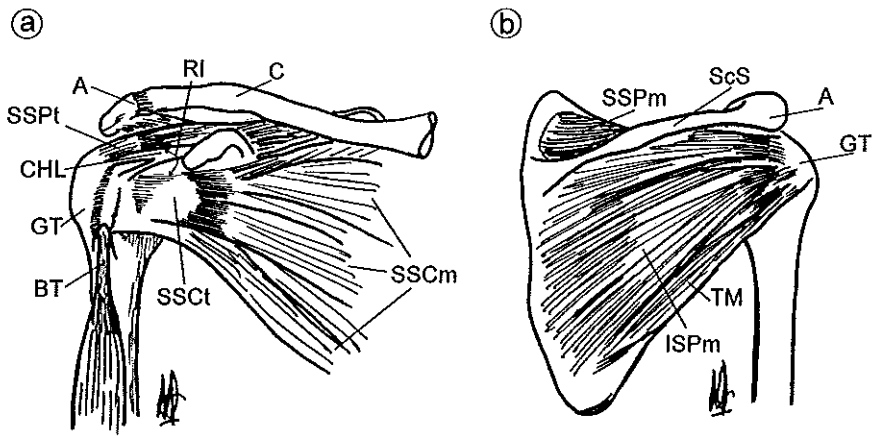


Figure 2.8a. Anterior view of the right shoulder. A=acromion; BT=biceps tendon (long head); C=clavicula; CHL=coracohumeral ligament; GT=greater tuberosity; RI=rotator interval; SSC_m=subscapularis muscle; SSC_t=subscapularis tendon; SSP_m=supraspinatus muscle; SSP_t=supraspinatus tendon. (Modified and redrawn after Neer, 1991)
Figure 2.8b. Posterior view of the right shoulder. ISP_m=infraspinatus muscle; ScS=scapular spine; TM=teres minor muscle. For additional legend see 2.8a

The *subscapularis muscle* is the largest member of the rotator cuff muscles.[46] It is a broad flat muscle that arises from the anterior surface of the scapula and converges laterally to insert into the lesser tuberosity. Its deep muscle and tendon fibers merge anteriorly with the GHJC. The lateral 15 mm of the subscapularis tendon consists of tendon fibers only; distinct tendinous bands are apparent in each pennate unit as they converge to the glenoid neck.[49] The subscapularis tendon continues as a tendinous band over the bicipital groove.[83] This tendinous band is traditionally referred to as the transverse humeral ligament. The superior border of the subscapularis lies directly under the base of the coracoid process. Here, the coracoid process separates the subscapularis tendon from the supraspinatus tendon. The subscapularis muscle is a powerful internal rotator of the humerus and a depressor of the humeral head. In addition, it has a role in the prevention of anterior dislocations of the humeral head.[66, 88]

The fusiform *supraspinatus muscle* originates from the supraspinatus fossa. Its muscle fibers pass laterally atop the GHJC and the bulk of its tendon fibers insert into the anterior facet of the greater tuberosity. A portion of these tendon fibers blends with the neighbouring fibers of other rotator cuff tendons. On its course laterally, the supraspinatus muscle tendon unit passes through the supraspinatus outlet (section 2.2.4 and Figure 2.2). The supraspinatus muscle has a role in active GH elevation, compressive stabilization of the humeral head, and forming a restraint against superior and inferior dislocations of the humeral head.[15, 40]

The flat *infraspinatus muscle* originates from the infraspinatus fossa at the posterior side of the scapula. It has superior and inferior portions that are divided by a tendinous raphe, which can be used for surgical access to the posterior side of the GHJ without jeopardizing the muscle's neurovascular bundles.[79] Approximately 1.5 cm medial from its insertion into the posterior facet of the greater tuberosity, the infraspinatus tendon blends superiorly with the posterior border of the supraspinatus tendon, and inferiorly with the superior border of the teres minor tendon. The infraspinatus is the most powerful external rotator of the GHJ.[34] Furthermore, it acts as an active restraint to humeral head dislocation assisting the posterior and superior regions of the GHJC.[35]

The fusiform *teres minor muscle* originates along the inferior lateral border of the scapula and inserts into the inferior facet of the greater tuberosity. Although it is a small muscle compared to the size of the infraspinatus muscle, it is positioned favorably to generate up to 40% of the total external rotation torque.[14] Coupling its action to the other rotator cuff muscles, the teres minor generates a compressive joint force to stabilize the GHJ; together with the subscapularis muscle it resists the upward pull of the deltoid muscle.[14]

Other scapulohumeral muscles, such as the *deltoid* and *teres major* muscles, do not take part in the formation of the rotator cuff, and will not be considered here.

2.2.5 Rotator Cuff

The tendons of the supraspinatus, subscapularis, infraspinatus and teres minor muscles form a conjoint tendon near their insertion sites: the rotator cuff (Figure 2.9ac). Lateral of the glenoid fossa, the individual cuff tendons can only be appreciated from the articular side (as seen in arthroscopy). Although most anatomy texts [18, 81] indicate separate insertion facets for each rotator cuff tendon, only a portion of each muscle-tendon unit appears to attach at each facet.[34] A thin synovial lining separates the inferior surface of the rotator cuff tendons from the joint cavity. The coracoid process separates the subscapularis tendon from the supraspinatus tendon, creating the triangular rotator

interval, which is bridged by the superior region of the GHJC. The base of the rotator interval is formed by the coracoid process, and the apex by the superior border of the transverse humeral ligament (see section 2.2.4).[36, 83]

For a thorough description of the microstructure of the rotator see Clark et al.[13] In the region of the supraspinatus and subscapularis tendons, the rotator cuff and GHJC, have an average thickness of 9-12 mm; they are clearly delineated into five layers. The articular side of the rotator cuff-GHJC complex shows a transverse band of fibers. This band extends in continuity with the coracohumeral ligament from the rotator interval into the plane between the GHJC and rotator cuff tendon fibers (Figure 2.9a).[12] It has been suggested that this transverse band serves as a stress-shielding 'cable' for the rotator cuff tendon insertions; it frequently coincides with the free margin of a rotator cuff tear (Figure 2.9b).[7]

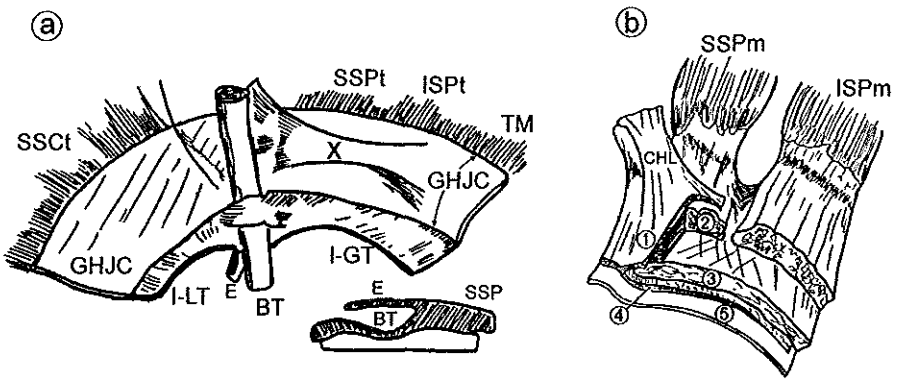


Figure 2.9a The four scapulohumeral muscle-tendon units form a continuous tissue sheet which is shown here from the articular side after it was dissected from the humerus and the rim of the glenoid fossa. A band of fibers (X) extends transversely from the rotator interval capsule (i.e. GHJC bridging the rotator interval) in continuity with the coracohumeral ligament into the plane between the joint capsule and the tendon fibers. The inset shows a cross-section of the bicipital groove and related structures. BT=biceps tendon (long head); GHJC=glenohumeral joint capsule; E=a fibrous extension of the supraspinatus tendon forms a part of the roof over the biceps tendon; I-GT= insertion side to the greater tuberosity; I-LT= insertion side to the lesser tuberosity; ISP_t=infraspinatus tendon; SSC_t=subscapularis tendon; SSP_t=supraspinatus tendon. (Modified and redrawn after Clark, 1992)

Figure 2.9b Diagram of the superior portions of the rotator cuff and GHJ capsule showing the five layers (1-5) of the rotator cuff capsule complex. CHL=coracohumeral ligament. For additional legend see figure 2.9a. (Modified and redrawn after Clark, 1992)

2.2.6 Bursae and potential spaces

Including the biceps tendon sheath, about 15 synovial bursae have been described around the GHJ.[9, 91] They occur between the most unyielding tissues: tendon and bone, skin and bone, and occasionally between muscle and bone near a tendon insertion. The subacromial-subdeltoid bursa (SASDB) is considered the most important bursa of the shoulder region.[10, 24, 26, 30, 42, 50, 57, 73, 89] It is located between the deltoid muscle, the acromion, the coracoacromial ligament and the coracoid process superficially and the rotator cuff deeply. In line with its function, the SASDB frequently has been referred to as the subacromial or secondary 'joint' of the shoulder.[69] Frequently, the subacromial and subdeltoid portions of the SASDB are described as separate entities (Figure 2.10a).[3] Although normally the SASDB is a potential space, it has a capacity of 5 to 10 ml when not reduced by adhesions or edema.[84] The SASDB communicates with the GHJ in case of a full thickness tear of the rotator cuff also involving the deep wall of this bursa.[84] Functional anatomic descriptions of transformation of the SASDB during movements of the arm are very confusing. *Chapter 3* deals with this topic.

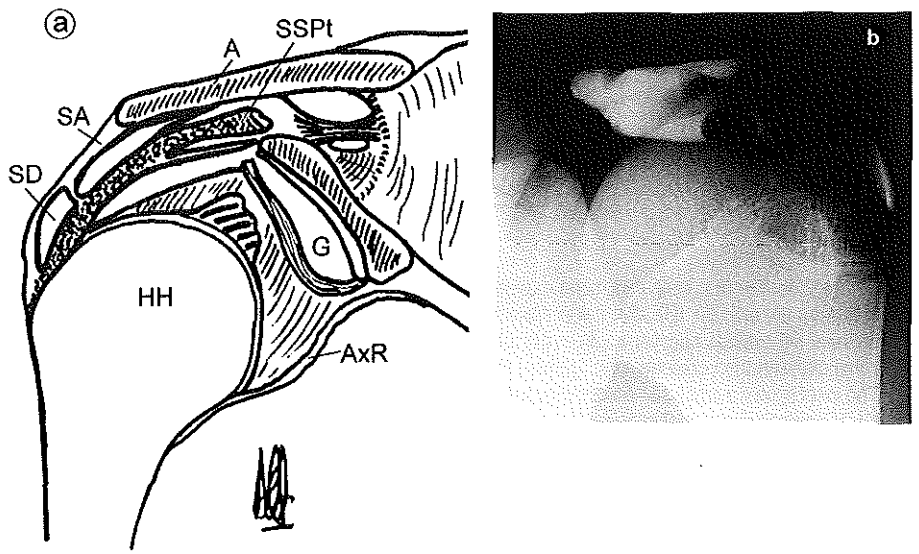


Figure 2.10a Typical representation of the bursae in a frontal section through the left shoulder. Posterior view. Separate subacromial (SA) and subdeltoid (SD) bursae. A=acromion; AxR=axillary recess; G=glenoid fossa; HH=humeral head. (Redrawn from Morris', 1966)
Figure 2.10b Normal bursogram of the right GHJ showing the SASDB. (Courtesy of dr AZ Ginai, University Hospital Rotterdam, Dijkzigt)

Another frequently (80-85%) encountered bursa is the *subscapular bursa*.[18, 65] It is located between the upper border of the subscapularis tendon and the neck of the glenoid fossa. In a vast majority of cases, this bursa communicates with the GHJ, and therefore, it can be considered as a recess rather than a true bursa.

The *subcoracoid bursa* is located between the subscapularis tendon on the one side, and the coracoid process and coracobrachialis tendon on the other. In 20% this bursa is an extension of the SASDB.[84] In such cases, the undersurface of the tip of the coracoid process may be visualized through an arthroscope placed in the SASDB (Figure 2.10b).[55] Between the superior aspect of the coracoid process and the overlying deltoid muscle another bursa may develop (about 1%). The presence of this *supracoracoid bursa* seems to be related to the size and orientation of the coracoid process in relation to the clavicle.[31]

2.2.7 Biceps tendon

The origin tendon of the long head of the biceps brachii muscle (biceps tendon) originates from the supraglenoid tubercle and adjacent glenoid rim and glenoid labrum. The biceps tendon is approximately 9 cm long and can be bifurcated or trifurcated at its origin.[18] The extent of its attachment to the glenoid labrum shows large variability.[18, 32, 83] It passes over the superior aspect of the humeral head and bends anteromedially to descend in the bicipital groove. On entering the groove, the biceps tendon is covered by a tendon sheath, an extension of the synovial layer of the GHJC that accompanies the tendon over a variable distance.[1] Distally in the bicipital groove, the superficial layer of synovial sheath reflects back on itself to form a deep layer. As a result, the biceps tendon is intra-articular but extrasynovial.[8] Consequently, filling of the biceps tendon sheath is a normal finding in an arthrogram. The length of the intra-articular portion depends on the position of the GHJ. With maximal GH elevation, about 1.5 cm of the biceps tendon lies intra-articular. In a position of GH adduction and extension, about 5.5 cm of the biceps tendon has an intra-articular course.[18] The physiologic angulation of the biceps tendon on entering the bicipital groove requests restraint mechanisms that provide protection against medial dislocation of the biceps tendon. Such protection is primarily provided by the coracohumeral ligament and, as a secondary line of defense, by the superior tendon fibers of the subscapularis.[11, 68, 80] According to traditional anatomic teaching the biceps tendon is held in the bicipital groove by means of the transverse humeral ligament.

All movements in the GHJ, regardless of the plane or direction of movement, are accompanied by gliding movements of the bicipital groove along the tendon.[18] Classically, the long head of the biceps brachii muscle is regarded a weak flexor and internal rotator in the GHJ.[4] In addition it has been shown that the biceps tendon can function as a dynamic anterior stabilizer [43, 52, 74, 92, 93] Furthermore, the biceps tendon functions as a depressor of the humeral head.[52, 92, 93]

2.3 Shoulder movements and Glenohumeral stability

2.3.1 Definition of glenohumeral planes, axes and directions

When movements of the multi-axial GHJ are being analyzed, it is preferable and easier to consider the movements of the humerus in relation to the scapula than to the frontal and sagittal planes. For the relevant axes, see Figure 2.11

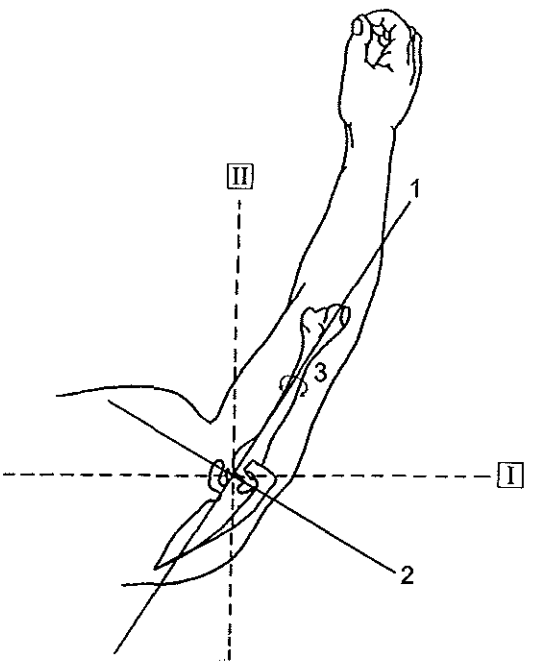


Figure 2.11 The multi-axial GHJ possesses three degrees of freedom. The GH axes (1,2) differ from those normally used in the clinical evaluation of the shoulder (I-II). I. Flexion and extension of the GHJ are forward and backward movements of the arm about a horizontal axis parallel to the plane of the scapula. II. GH abduction and adduction are outward and inward movements about a horizontal axis perpendicular to the plane of the scapula. Humeral rotation are inward and outward rotations about the longitudinal axis (3) of the humerus.
All movements that occur involving intermediate planes can always be mathematically reduced into components related to the three GH axes, which are illustrated here.

2.3.2 Movements of the shoulder

A normal shoulder girdle mechanism is essential for a physiological range of motion of the arm and depends on three synovial joints (the glenohumeral, acromioclavicular, and sternoclavicular joints) and the sliding mechanism of the scapula along the rib cage (the scapulothoracic sliding mechanism). Sliding of subacromial soft tissues under the coracoacromial arc is important too.[69] Additionally, form and function of the thoracic spine and rib cage play an important role in the total ROM of the arm.[23, 59, 97] The scapulothoracic sliding mechanism refers to the sliding of the fascial layers that cover the subscapularis and serratus anterior muscles.

During elevation of the arm, scapular movements are mainly controlled by the action of the rhomboid, trapezius, serratus anterior and levator scapulae muscles (obviously, they for their part are controlled by the central nervous system). The levator scapulae and trapezius muscles bear most of the weight of the upper extremity.[59] The coracoclavicular ligaments and the acromioclavicular joint capsule play an important role in obtaining stability within the acromioclavicular joint (Figure 2.12).[27, 75]

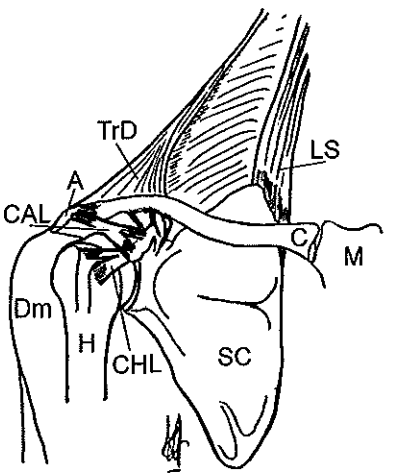


Figure 2.12 Anterior view of the right shoulder region shows the suspensory mechanism of the upper extremity in the erect position. The main active components are the levator scapula and upper trapezius muscles. The coracoclavicular ligaments are important passive suspensory components. A=acromion; C=clavicula; CAL=coracoacromial ligament; CHL=coracohumeral ligament; Dm=contour of the deltoid muscle; H=humerus; LS=levator scapulae muscle; M=manubrium sterni; SC=scapula; TrD=descending part of the trapezius muscle. (Modified and redrawn after Neer, CS 1990)

Movements in the joints of the shoulder girdle mechanism occur synchronously [41] and are essential to provide the upper limb its normal ROM. Movement of the scapula and humerus are coupled in a complex way (scapulo-humeral rhythm, glenohumeral-to-

scapulothoracic ratio). For full elevation of the arm, the coordinate action of the upper part of the trapezius (lateral rotation of the scapula), serratus anterior (protraction of the scapula), the anterior part of the deltoid (GH elevation), supraspinatus (GH elevation and depression of the humeral head) and infraspinatus (external humeral rotation) muscles is important.

Scapular movement is essential for correct positioning of the glenoid fossa in relation to the humeral head.[53] These movements, analogous to the balancing of a ball on the tip of a seal's nose, play an elementary role in obtaining GHJ stability.

2.3.3 Glenohumeral stability

Despite the limited bony coverage of the humeral head by the glenoid fossa, the humeral head is centered in the glenoid fossa, both in rest and throughout most of its movements.[39, 70] While the physician worries about the relative high incidence of GHJ dislocations compared to other joints of the body, the anatomist wonders how this seemingly unstable joint can be so stable. How to resist the pull of gravitational forces over long periods of time, permit lifting heavy loads, and to cause tennis balls to travel with speeds of over 200 kilometers per hour? Due to their laxity, necessary to allow normal motion, the GH ligaments cannot prevent humeral head translation throughout the complete ROM. Therefore, the GH ligaments can prevent excessive humeral head translation only at the extremes of motion.[64, 88, 92] Obviously, passive and active restraints must work together to couple biomechanical stability with the exceptional mobility of the GHJ. *Concavity compression* and *scapulohumeral balance* turned out to be important in the static and the dynamic restraint mechanisms.[53] Concavity compression refers to GHJ stability due to compression of the humeral head into the concave glenoid fossa. Increasing the magnitude of the compressive load by dynamic muscle contraction enhances concavity compression stabilization (Figure 2.13a). The related scapulo-humeral balance refers to the positioning of the GHJ so that the net joint reaction force is balanced within the glenoid fossa. The greater the arc provided by the glenoid fossa and glenoid labrum, the larger the range of joint force angles acting through the humeral head that must be stabilized (Figure 2.13bc).

In order to profit from the extreme mobility that the GHJ offers, a concerted action of many shoulder muscles is essential for maintaining GH stability.[5] Furthermore, passive guidance of the GH movements is necessary to keep articular motion within its physiological limits.[45]

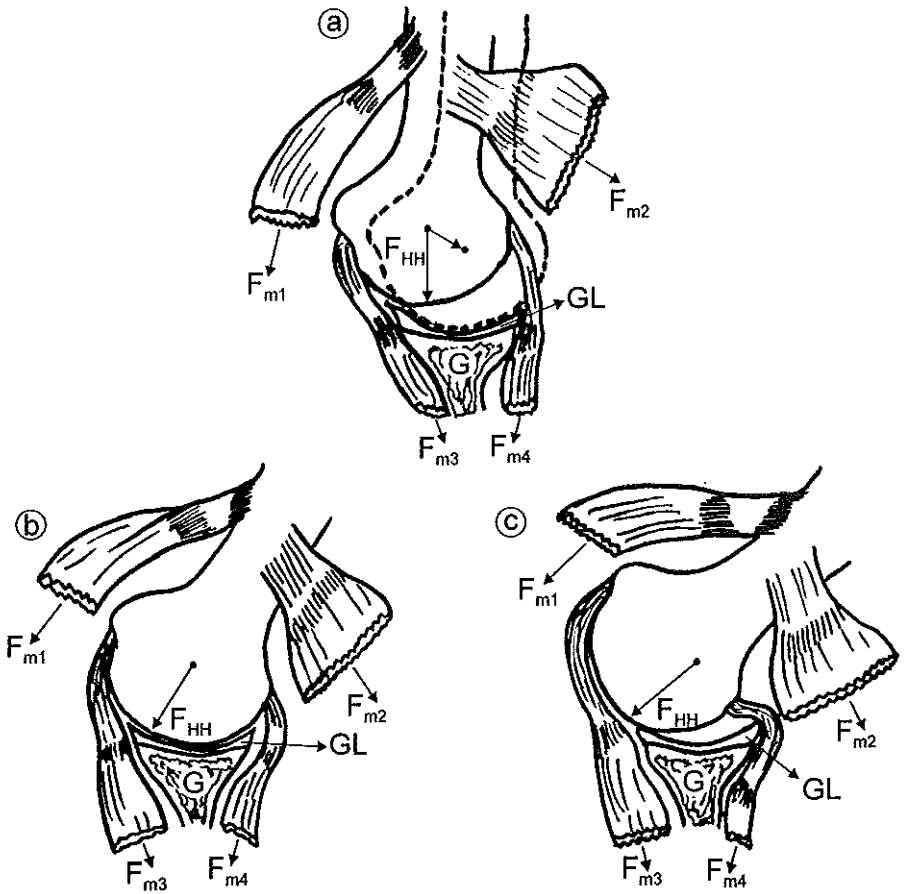


Figure 2.13a Concavity compression refers to the increasing resistance to lateral displacement of a convex object that is pressed into a concave surface. F_{HH}=net muscle force acting on the humeral head; F_{m1}-F_{m4}=muscle forces; G=glenoid fossa; GL=glenoid labrum; HH=humeral head

Figure 2.13b Scapulohumeral balance refers to the principle that the humeral head is balanced in the glenoid fossa if the net reaction force caused by compression of the humeral head in the glenoid fossa passes between the boundaries of the glenoid fossa. For legend see figure 2.13a.

Figure 2.13c In case the net muscle force projects outside the boundaries of the glenoid fossa, subluxation or dislocation occurs. For legend see figure 2.13a.

The presence of an intact glenoid labrum is important for both mechanisms. (Figures modified and redrawn from Lippitt, 1993)

REFERENCES

1. **Ahovuo J, Linden H, Hovi I, Paavolainen P, Bjorkenheim JM, Slatis P.** Arthrography of the biceps tendon. *Eur J Radiol* 1988;8(3):196-8.
2. **Andrews JR, Byrd JW, Kupferman SP, Angelo RL.** The profile view of the acromion. *Clin Orthop* 1991(263):142-6.
3. **Anson BJ,** ed. Morris' Human Anatomy. 12th ed. New York Toronto London: McGraw-Hill book company, 1966.
4. **Basmajan JV, Latif MA.** Integrated actions and functions of the chief flexors of the elbow. *J Bone Joint Surg [Am]* 1957;39 A:1106-18.
5. **Bigliani LU, Kelkar R, Flatow EL, Pollock RG, Mow VC.** Glenohumeral stability. Biomechanical properties of passive and active stabilizers. *Clin Orthop* 1996(330):13-30.
6. **Bigliani LU, Ticker JB, Flatow EL, Soslowsky LJ, Mow VC.** The relationship of acromial architecture to rotator cuff disease. *Clin Sports Med* 1991;10(4):823-38.
7. **Burkhart SS.** A unified biomechanical rational for the treatment of rotator cuff tears: Debridement versus repair. In: Burkhead WZ, ed. Rotator cuff disorders. 1st ed. Baltimore Philadelphia London: Williams and Wilkins, 1996:293-312.
8. **Burkhead WZ.** The biceps tendon. In: Rockwood CA, Matsen FA, eds. The shoulder. Philadelphia: W.B. Saunders, 1990:793-99. vol 2.
9. **Bywaters EGL.** The bursae of the body. Editorial. *Ann Rheum Dis* 1965;24:215-18.
10. **Calvert PT, Packer NP, Stoker DJ, Bayley JI, Kessel L.** Arthrography of the shoulder after operative repair of the torn rotator cuff. *J Bone Joint Surg [Br]* 1986;68(1):147-50.
11. **Chan TW, Dalinka MK, Kneeland JB, Chervrot A.** Biceps tendon dislocation: evaluation with MR imaging. *Radiology* 1991;179(3):649-52.
12. **Clark J, Sidles JA, Matsen FA.** The relationship of the glenohumeral joint capsule to the rotator cuff. *Clin Orthop* 1990(254):29-34.
13. **Clark JM, Harryman DT, 2nd.** Tendons, ligaments, and capsule of the rotator cuff. Gross and microscopic anatomy. *J Bone Joint Surg [Am]* 1992;74(5):713-25.
14. **Colachis Jr S, Strohm BR, Brechner VL.** Effects of axillary nerve block on muscle force in the upper extremity. *Arch Phys Med Rehabil* 1969;50:647-54.
15. **Colachis Jr SC, Strohm BR.** Effects of suprascapular and axillary nerve block on muscle forces in the upper extremity. *Arch Phys Med Rehabil* 1971;52:22-9.
16. **Cone RO, Danzig L, Resnick D, Goldman AB.** The bicipital groove: radiographic, anatomic, and pathologic study. *AJR Am J Roentgenol* 1983;141(4):781-8.
17. **Curl LA, Warren RF.** Glenohumeral joint stability. Selective cutting studies on the static capsular restraints. *Clin Orthop* 1996(330):54-65.
18. **DePalma AF.** Surgery of the shoulder. 2nd ed. Philadelphia Toronto: J.B. Lippincott Company, 1972.
19. **Edelson JG, Taitz C.** Anatomy of the coraco-acromial arch. Relation to degeneration of the acromion. *J Bone Joint Surg [Br]* 1992;74(4):589-94.
20. **Edelson JG, Taitz C, Grishkan A.** The coracohumeral ligament. Anatomy of a substantial but neglected structure. *J Bone Joint Surg [Br]* 1991;73(1):150-3.
21. **Ellman H.** Arthroscopic subacromial decompression: analysis of one- to three-year results. *Arthroscopy* 1987;3(3):173-81.
22. **Ellman H.** Arthroscopic treatment of impingement of the shoulder. *Instr Course Lect* 1989;38:177-85.
23. **Emery RJ, Mullaji AB.** Glenohumeral joint instability in normal adolescents. Incidence and significance. *J Bone Joint Surg Br* 1991;73(3):406-8.

24. **Farin PU, Jaroma H, Harju A, Soimakallio S.** Shoulder impingement syndrome: sonographic evaluation. *Radiology* 1990;176(3):845-9.
25. **Ferrari DA.** Capsular ligaments of the shoulder. Anatomical and functional study of the anterior superior capsule. *Am J Sports Med* 1990;18(1):20-4.
26. **Fukuda H, Mikasa M, Yamanaka K.** Incomplete thickness rotator cuff tears diagnosed by subacromial bursography. *Clin Orthop* 1987(223):51-8.
27. **Fukuda K, Craig EV, An KN, Cofield RH, Chao EY.** Biomechanical study of the ligamentous system of the acromioclavicular joint. *J Bone Joint Surg [Am]* 1986;68(3):434-40.
28. **Gagey O, Bonfait H, Gillot C, Hureau J, Mazas F.** Anatomic basis of ligamentous control of elevation of the shoulder (reference position of the shoulder joint). *Surg Radiol Anat* 1987;9(1):19-26.
29. **Gartsman GM, Blair ME, Jr., Noble PC, Bennett JB, Tullos HS.** Arthroscopic subacromial decompression. An anatomical study. *Am J Sports Med* 1988;16(1):48-50.
30. **Griffith JF, Peh WC, Evans NS, Smallman LA, Wong RW, Thomas AM.** Multiple rice body formation in chronic subacromial/subdeltoid bursitis: MR appearances. *Clin Radiol* 1996;51(7):511-4.
31. **Haas de WHD, Drucker F.** Gewone en ongewone betrekkingen tussen processus coracoideus en de clavicula. *Ned Tijdschr Geneesk* 1966;110:1640-6.
32. **Habermeyer P, Kaiser E, Knappe M, Kreusser T, Wiedemann E.** Zur funktionellen Anatomie und Biomechanik der langen Bicepssehne. *Unfallchirurg* 1987;90(7):319-29.
33. **Ha'eri GB, Wiley AM.** Shoulder impingement syndrome. Results of operative release. *Clin Orthop* 1982(168):128-22.
34. **Harryman DT, Clarck Jr JM.** Anatomy of the rotator cuff. In: Burkhead Jr WZ, ed. Rotator cuff disorders. 1st ed. Baltimore: Williams & Wilkins, 1996:23-35.
35. **Harryman DTd, Sidles JA, Clark JM, McQuade KJ, Gibb TD, Matsen FAd.** Translation of the humeral head on the glenoid with passive glenohumeral motion. *J Shoulder Elbow Surg* 1995;4(3):199-208.
36. **Harryman DTd, Sidles JA, Harris SL, Matsen FAd.** The role of the rotator interval capsule in passive motion and stability of the shoulder. *J Bone Joint Surg [Am]* 1992;74(1):53-66.
37. **Hitchcock HH, Bechtol CO.** Observations on the role of the long head of the biceps brachii in its causation. *J Bone Joint Surg [Am]* 1948;30:263-73.
38. **Howell SM, Galinat BJ.** The glenoid-labral socket. A constrained articular surface. *Clin Orthop* 1989(243):122-5.
39. **Howell SM, Galinat BJ, Renzi AJ, Marone PJ.** Normal and abnormal mechanics of the glenohumeral joint in the horizontal plane. *J Bone Joint Surg [Am]* 1988;70(2):227-32.
40. **Howell SM, Imobersteg AM, Seger DH, Marone PJ.** Clarification of the role of the supraspinatus muscle in shoulder function. *J Bone Joint Surg [Am]* 1986;68(3):398-404.
41. **Inman VT, Saunders JB, Abbott LC.** Observations of the function of the shoulder joint. 1944 [classical article]. *Clin Orthop* 1996(330):3-12.
42. **Ishii H, Brunet JA, Welsh RP, Ulthoff HK.** "Bursal reactions" in rotator cuff tearing, the impingement syndrome, and calcifying tendinitis. *J Shoulder Elbow Surg* 1997;6(2):131-6.
43. **Itoi E, Kuechle DK, Newman SR, Morrey BF, An KN.** Stabilising function of the biceps in stable and unstable shoulders [published erratum appeared in J Bone Joint Surg Br 1994 Jan;76(1):170]. *J Bone Joint Surg Br* 1993;75(4):546-50.
44. **Jobe CM.** Superior glenoid impingement. Current concepts. *Clin Orthop* 1996(330):98-107.

45. **Jobe CM, Ianotti JP.** Limits imposed on glenohumeral motion by joint geometry. *J Shoulder Elbow Surg* 1995;4:281-85.
46. **Keating JF, Waterworth P, Shaw-Dunn J, Crossan J.** The relative strengths of the rotator cuff muscles. A cadaver study. *J Bone Joint Surg [Br]* 1993;75(1):137-40.
47. **Kieft GJ, Bloem JL, Obermann WR, Verbout AJ, Rozing PM, Doornbos J.** Normal shoulder: MR imaging. *Radiology* 1986;159(3):741-5.
48. **Kieft GJ, Bloem JL, Rozing PM, Obermann WR.** Rotator cuff impingement syndrome: MR imaging. *Radiology* 1988;166(1 Pt 1):211-4.
49. **Klapper RC, Jobe FW, Matsuura P.** The subscapularis muscle and its glenohumeral ligament-like bands. A histomorphologic study. *Am J Sports Med* 1992;20(3):307-10.
50. **Konrath GA, Nahigian K, Kolowich P.** Pigmented villonodular synovitis of the subacromial bursa. *J Shoulder Elbow Surg* 1997;6(4):400-4.
51. **Kronberg M, Brostrom LA, Posch E.** Stability in relation to humeral head retroversion after surgical treatment of recurrent anterior shoulder dislocations. *Orthopedics* 1993;16(3):281-5.
52. **Kumar VP, Satku K, Balasubramaniam P.** The role of the long head of biceps brachii in the stabilization of the head of the humerus. *Clin Orthop* 1989(244):172-5.
53. **Lippitt S, Matsen F.** Mechanisms of glenohumeral joint stability. *Clin Orthop* 1993(291):20-8.
54. **Mack LA, Matsen FAd, Kilcoyne RF, Davies PK, Sickler ME.** US evaluation of the rotator cuff. *Radiology* 1985;157(1):205-9.
55. **Matthews LS, Fadale PD.** Subacromial anatomy for the arthroscopist. *Arthroscopy* 1989;5(1):36-40.
56. **Matthews LS, Terry G, Vetter WL.** Shoulder anatomy for the arthroscopist. *Arthroscopy* 1985;1(2):83-91.
57. **Mitchell MJ, Causey G, Berthoty DP, Sartoris DJ, Resnick D.** Peribursal fat plane of the shoulder: anatomic study and clinical experience. *Radiology* 1988;168(3):699-704.
58. **Neer C, Poppen NK.** Supraspinatus outlet. *Orthop Trans* 1987;11:234-37.
59. **Neer CS.** Shoulder reconstruction. 1st ed. Philadelphia London Tokyo: W.B. Saunders Company, 1990.
60. **Neer CSd.** Anterior acromioplasty for the chronic impingement syndrome in the shoulder: a preliminary report. *J Bone Joint Surg [Am]* 1972;54(1):41-50.
61. **Neer CSd, Satterlee CC, Dalsey RM, Flatow EL.** The anatomy and potential effects of contracture of the coracohumeral ligament. *Clin Orthop* 1992(280):182-5.
62. **Nobuhara K, Ikeda H.** Rotator interval lesion. *Clin Orthop* 1987(223):44-50.
63. **Nottage WM.** Arthroscopic anatomy of the glenohumeral joint and subacromial bursa. *Orthop Clin North Am* 1993;24(1):27-32.
64. **O'Brien S, Neves MC, Arnoczky SP, et al.** The anatomy and histology of the inferior glenohumeral ligament complex of the shoulder. *Am J Sports Med* 1990;18(5):449-56.
65. **O'Brien SJ, Arnoczky DVM, Warren RF, Rozbruch SR.** Developmental anatomy of the shoulder and anatomy of the glenohumeral joint. In: Rockwood CA, Matsen FA, eds. The shoulder. Philadelphia: W.B. Saunders, 1990:15-16. vol 1.
66. **Ovesen J, Nielsen S.** Stability of the shoulder joint. Cadaver study of stabilizing structures. *Acta Orthop Scand* 1985;56(2):149-51.
67. **Penny JN, Welsh RP.** Shoulder impingement syndromes in athletes and their surgical management. *Am J Sports Med* 1981;9(1):11-5.

68. **Petersson CJ.** Spontaneous medial dislocation of the tendon of the long biceps brachii. An anatomic study of prevalence and pathomechanics. *Clin Orthop* 1986(211):224-7.
69. **Pfuhl W.** Das subakromiale Nebengelenk de Schultergelenks. *Morph JB* 1934;73:300-46.
70. **Poppen NK, Walker PS.** Forces at the glenohumeral joint in abduction. *Clin Orthop* 1978(135):165-70.
71. **Putz R.** Topographie und Funktionelle Anatomie des Schulterguertels und des Schultergelenks. In: Habermeyer P, Krueger P, Scheiberer L, eds. Schulterchirurgie. Muenchen Wien Baltimore: Urban & Schwarzenberg, 1990:3-18.
72. **Putz R, Liebermann J, Reichelt A.** Funktion des Ligamentum Coracoacromiale. *Acta Anat (Basel)* 1988;131(2):140-5.
73. **Rahme H, Nordgren H, Hamberg H, Westerberg CE.** The subacromial bursa and the impingement syndrome. A clinical and histological study of 30 cases. *AJR Am J Roentgenol* 1993;160(3):561-4.
74. **Rodosky MW, Harner CD, Fu FH.** The role of the long head of the biceps muscle and superior glenoid labrum in anterior stability of the shoulder. *Am J Sports Med* 1994;22(1):121-30.
75. **Rosenorn M, Pedersen EB.** The significance of the coracoclavicular ligament in experimental dislocation of the acromioclavicular joint. *Acta Orthop Scand* 1974;45(3):346-58.
76. **Saha AK.** Anterior recurrent dislocation of shoulder. *Acta Orthop Scand* 1967;68:479-93.
77. **Saha AK.** The classic. Mechanism of shoulder movements and a plea for the recognition of "zero position" of glenohumeral joint. *Clin Orthop* 1983(173):3-10.
78. **Schlemm F.** Ueber die Verstarkungsbaender am Schultergelenk. *Arch Anat* 1853;10:45-8.
79. **Shaffer BS, Conway J, Jobe FW, Kvitne RS, Tibone JE.** Infraspinatus muscle-splitting incision in posterior shoulder surgery. An anatomic and electromyographic study. *Am J Sports Med* 1994;22(1):113-20.
80. **Slatis P, Aalto K.** Medial dislocation of the tendon of the long head of the biceps brachii. *Acta Orthop Scand* 1979;50(1):73-7.
81. **Sobotta.** Atlas of human anatomy. Munich: Urban & Schwarzenberg, 1982:311. vol I.
82. **Soslowsky LJ, Flatow EL, Bigliani LU, Mow VC.** Articular geometry of the glenohumeral joint. *Clin Orthop* 1992(285):181-90.
83. **Steiner D, Hermann B.** Zur Topographie des oberen Bicepssehnenabschnittes. *Langenbecks Arch Chir* 1990;375(1):19-23.
84. **Strizak AM, Danzig L, Jackson DW, Resnick D, Staple T.** Subacromial bursography. An anatomical and clinical study. *J Bone Joint Surg [Am]* 1982;64(2):196-201.
85. **Symeonides PP, Hatzokos I, Christoforides J, Pournaras J.** Humeral head torsion in recurrent anterior dislocation of the shoulder. *J Bone Joint Surg [Br]* 1995;77(5):687-90.
86. **Thorling J, Bjerneld H, Hallin G, Hovelius L, Hagg O.** Acromioplasty for impingement syndrome. *Acta Orthop Scand* 1985;56(2):147-8.
87. **Toivonen DA, Tuite MJ, Orwin JF.** Acromial structure and tears of the rotator cuff. *J Shoulder Elbow Surg* 1995;4(5):376-83.
88. **Turkel SJ, Panio MW, Marshall JL, Girgis FG.** Stabilizing mechanisms preventing anterior dislocation of the glenohumeral joint. *J Bone Joint Surg [Am]* 1981;63(8):1208-17.
89. **Uhthoff HK, Sarkar K.** Surgical repair of rotator cuff ruptures. The importance of the subacromial bursa. *J Bone Joint Surg Br* 1991;73(3):399-401.

90. **Vahlensieck M, Wiggert E, Wagner U, Schmidt HM, Schild H.** Subacromial fat pad. *Surg Radiol Anat* 1996;18(1):33-6.
91. **von Lanz T, Wachsmuth W.** Praktische Anatomie. 2nd ed. Berlin Goettingen Heidelberg: Springer Verlag, 1959. vol 1.
92. **Warner JJ, Deng XH, Warren RF, Torzilli PA.** Static capsuloligamentous restraints to superior-inferior translation of the glenohumeral joint. *Am J Sports Med* 1992;20(6):675-85.
93. **Warner JJ, McMahon PJ.** The role of the long head of the biceps brachii in superior stability of the glenohumeral joint. *J Bone Joint Surg [Am]* 1995;77(3):366-72.
94. **Watson M.** The refractory painful arc syndrome. *J Bone Joint Surg [Br]* 1978;60-B(4):544-6.
95. **Zlatkin MB, Bjorkengren AG, Gylys-Morin V, Resnick D, Sartoris DJ.** Cross-sectional imaging of the capsular mechanism of the glenohumeral joint. *AJR Am J Roentgenol* 1988;150(1):151-8.
96. **Zuckerman JD, Kummer FJ, Cuomo F, Greller M.** Interobserver reliability of acromial morphology classification: an anatomic study. *J Shoulder Elbow Surg* 1997;6(3):286-7.
97. **Zuckerman JD, Matsen FA, de Gast A.** Biomechanica van de schouder. In: Snijders CJ, Nordin M, Frankel VH, eds. Biomechanica van het spierskeletstelsel: Grondslagen en toepassingen. Utrecht: Lemma BV, 1995:337-59.

CHAPTER

3

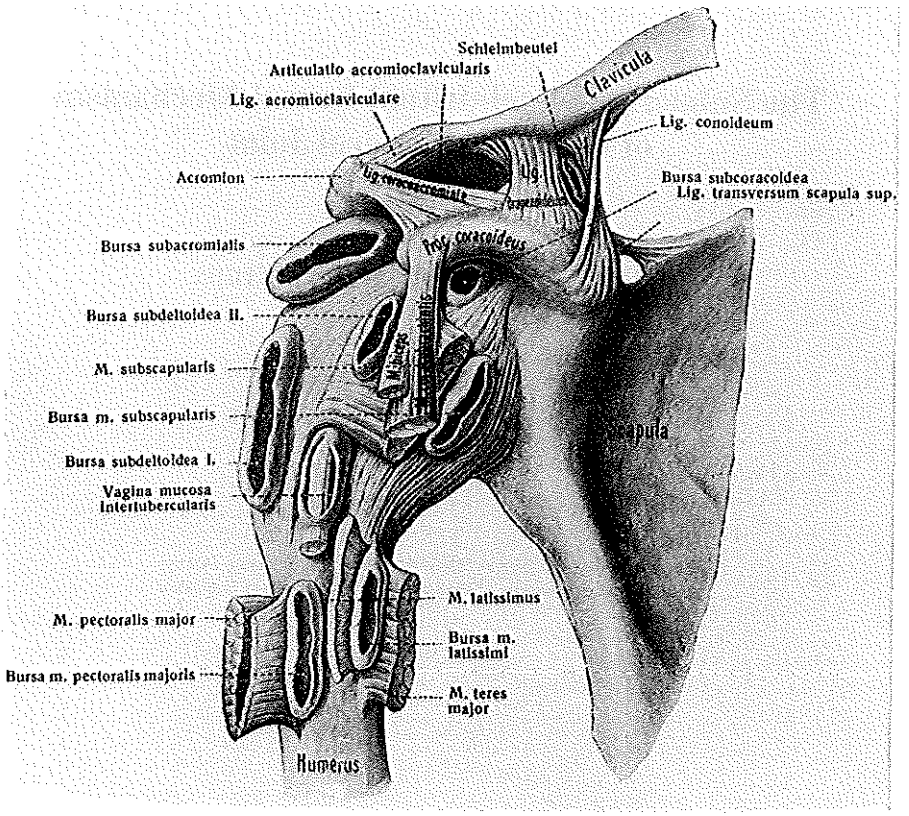
The Subacromial-subdeltoid Bursal Mechanism of the Glenohumeral Joint

AN ANATOMICAL AND KINEMATIC STUDY

ARTHUR DE GAST, MD§,¶ , ROB STOECKART, PH.D.¶,

§Dept of Orthopaedic Surgery, University Hospital Rotterdam, Dijkzigt
¶Dept of Anatomy, Faculty of Medicine, Erasmus University Rotterdam

Submitted for Publication


Schleimbeutel
Articulatio acromioclavicularis
Lig. acromioclaviculare
Clavicula
Lig. conoideum
Acromion
Lig. coracoacromiale
Lig. trapezoideum
Bursa subcoracoidea
Lig. transversum scapulae sup.
Bursa subacromialis
Proc. coracoideus
Bursa subdeltoidea II.
M. subscapularis
Bursa m. subscapularis
Scapula
Bursa subdeltoidea I.
Vagina mucosa intertubercularis
M. latissimus
M. pectoralis major
Bursa m. latissimi
Bursa m. pectoralis majoris
M. teres major
Humerus

CHAPTER

3

The Subacromial-subdeltoid Bursal Mechanism of the Glenohumeral Joint

Abstract

Clinically, the subacromial-subdeltoid bursa is the most important bursa in the shoulder region, but descriptions of its functional anatomy and pathomechanics are confusing. Therefore, the anatomy of unembalmed and embalmed human shoulder specimens (n=63) was studied in relation with their function. Forty-seven specimens were used for kinematic studies and cross sectioning. It turned out that various structures control transformation of the bursa during movement in the glenohumeral joint: rotator cuff tendons, parts of the coracobrachialis muscle and the short head of the biceps brachii. Furthermore, for smooth elevation of the arm it is essential that the accumulation of bursal tissue of the superficial wall of the subdeltoid portion is taken care of. We could show that specific wrinkling-patterns of the bursal wall guarantee smooth transformation of the bursa and deal with the theoretical surplus of bursal tissue. Pathomechanically, impingement related lesions of the walls of the subacromial-subdeltoid bursa specifically relate to the superficial wall of the subacromial portion and the deep wall of the subdeltoid portion. A better understanding of the functional anatomy of the subacromial-subdeltoid bursa will add to the diagnosis and treatment of patients with complaints of the shoulder.

3.1 Introduction

To allow for the large range of glenohumeral joint (GHJ) movement with least resistance, bursae are needed. A bursa is a closed and flattened sac lined with synovium that contains fluid and is found in areas subject to friction. The main bursa of the shoulder region is the subacromial-subdeltoid bursa (SASDB), which is located in the secondary 'joint' of the shoulder: the subacromial space. A distinction can be made between the superficial wall and the deep wall of the SASDB (Figure 3.2b). In human embryos of 12 weeks the SASDB is already present as a well-defined structure (Figure 3.1).[41]

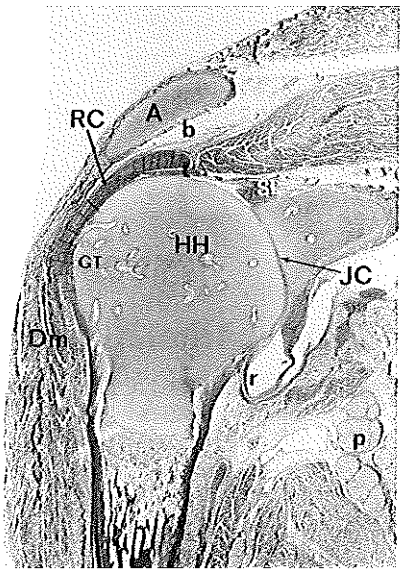


Figure 3.1 Frontal section of the right shoulder of a human fetus of 12.5 weeks, 86 mm with subacromial-subdeltoid bursa. Humeral head diameter approximately 5 mm. Azan. A=acromion; b=subacromial-subdeltoid bursa; Dm=deltoid muscle; GT=greater tuberosity; HH=humeral head; JC=joint cavity; p=brachial plexus; r=axillary recess; SL=superior part of the glenoid labrum; RC=rotator cuff; t=muscle-tendon transition of the supraspinatus. (Reproduced from Uthoff, 1990)

Descriptions and illustrations of the SASDB are confusing. For instance, according to *Gray's Anatomy* the 'sub*acromial* bursa' is located between the *deltoid* muscle and the joint capsule.[15] In *Sobotta's Atlas*, illustrations show a small 'subacromial bursa' that covers exclusively the superior portion of the supraspinatus tendon passing under the acromion.[38] Also, structural adaptations of the SASDB during position changes in the GHJ are not well documented. According to Katthagen [23] and Habermeyer et al.[17] the SASDB slides completely under the coracoacromial arc during abduction of the arm (Figure 3.2ab). However, since the superficial wall of the SASDB is firmly attached to the fascia of the deltoid muscle,[39] this part of the SASDB cannot be expected to slide under the coracoacromial arc during abduction of the arm.

In subacromial impingement the SASDB is frequently involved,[8, 13, 36, 40] but mostly the discussion on the structural pathology focuses on changes in the rotator cuff (RC).[2, 4, 34, 44] In studies dealing with the diagnosis and treatment of subacromial impingement, only few authors pay attention the pathology of the SASDB [35, 39] and its transformations during movement of the arm.[13]

The aim of the present study is to assess the functional anatomy of the SASDB and to provide a better basis for diagnosis and treatment of patients with shoulder pain.

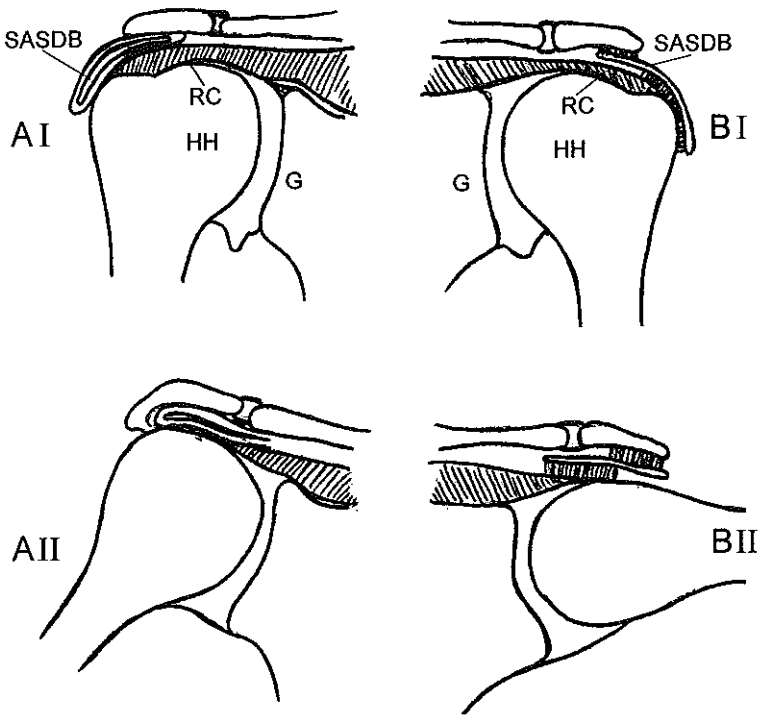


Figure 3.2. Examples of former illustrations drawn to show structural transformation of the SASDB during (full) GH abduction (I=0° GH elevation; II=GH elevation). A. The GHJ did not notably change position (II). GH elevation seems to have taken place through scapular movement alone. Nevertheless, the SASDB has moved medially and lies completely under the acromion. (Redrawn from Habermeyer and Wiedemann, 1990) B. In the original drawing the attachment of the SD portion of the SASDB to the deltoid muscle is not drawn. Obviously, it would imply that the deltoid muscle slides completely under the acromion during GH abduction. (Redrawn from Katthagen 1989). G=Glenoid; HH=humeral head; RC=rotator cuff; SASDB=subacromial-subdeltoid bursa.

3.2 Materials and Methods

3.2.1 Anatomic study

Dissection was performed on 4 unembalmed shoulder specimens from 2 human specimens (both male) age 71 and 67 years, 2 right and 2 left shoulders, and 59 embalmed shoulder specimens (30 male, 29 female) age 75.6 $\pm$ 5.3 years of age, 28 left and 31 right shoulders. The 4 unembalmed shoulder specimens were frozen at about 28 hours after death at -40° C and thawed 12 hours before dissection at room temperature. Forty-eight to 60 hours postmortem the other specimens were embalmed by vascular perfusion with a medium containing: 50g phenol 99%, 20g $MgSO_4$, 20g

$NaSO_4$, 10g NaCl, 60ml formaldehyde 37%, 60ml glycerin, H_2O ad 1000ml. The specimens were kept in containers filled with phenol (30 g/l) for 6 weeks. Subsequently, the specimens were stored in phenoxy-ethanol (10ml/l) at a temperature of 14° C for three months. Two embalmed shoulder specimens were deep-frozen (-80°C) to make cryosections and plastinated cross-sections. Plastination was performed using the E12 technique [18] as described in a previous study.[11] Forty-five specimens were specifically used to study the anatomic connections between the SASDB, the coracoacromial ligament, the coracobrachialis muscle (CB) and the short head of the biceps brachii muscle (SHBB).

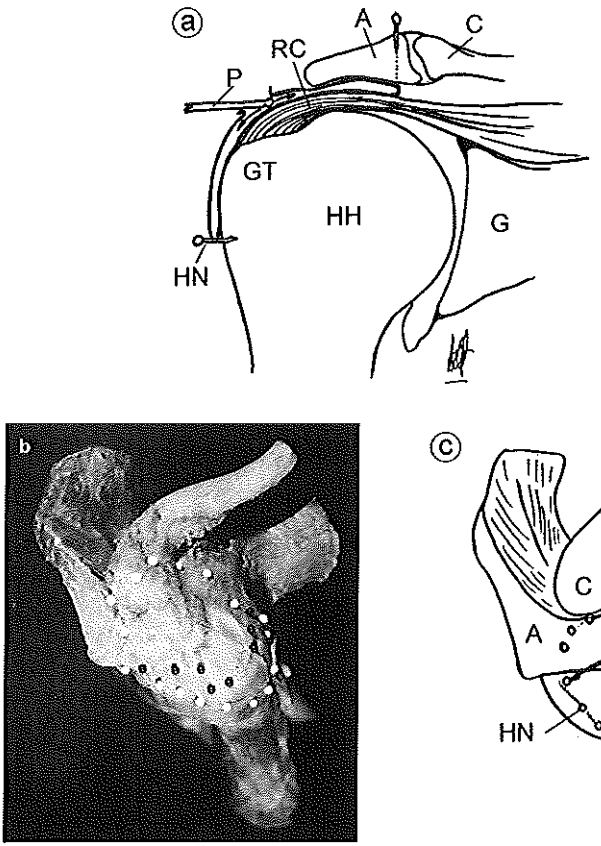


Figure 3.3a Schematic drawing to show how the interior of the SASDB is probed with a flexible plastic rod through an opening created in the lateral part of the superficial wall. A=acromion; C=clavicula; G=glenoid fossa; GT=greater tuberosity; HH=humeral head; HN=household needle; P=plastic rod; RC=rotator cuff.
Figures 3.3b Latero-superior view of the right shoulder showing the white needles that mark the edges of the SASDB. In this particular specimen black needles mark two bursal septa.
Figure 3.3c Reference lines used to estimate the surface area of the SASDB. Small circles represent the household needles as shown in figure 3.3b. A=acromion; CAL=coracoacromial ligament; PRC=coracoid process.

After removing the skin and subcutaneous fat, a five-step dissection was carried out to investigate the SASDB. First, the major shoulder muscles were stripped of the shoulder specimens, preserving the proximal one-third of the SHBB, the deltoid and CB muscles, the RC muscle-tendon units, the SASDB and, the tendon of the long head of the biceps brachii (biceps tendon). Blunt splitting of the deltoid muscle fibers between its clavicular and acromial parts created an opening extending to the superficial wall of the SASDB. Through the opening, movements of superficial wall could be observed during elevation of the arm in various planes. Second, the deltoid muscle was removed completely. The anatomic relationships of the superficial wall of the SASDB, coracoacromial ligament, SHBB and CB muscle were documented with the use of photographs and schematic drawings. The width of the conjoint tendon of the SHBB and CB muscles was measured with a caliper 1 cm proximal to the tip of the coracoid process. Third, in 20 specimens (10 left and 10 right shoulders) a small opening was made in the lateral aspect of the superficial layer of the SASDB to measure the size of the SASDB by probing the bursa from the inside (Figure 3.3a). The bursal margins and intrabursal septa were marked with household needles (Figure 3.3b). The surface area of the SASDB was estimated by measuring the anterior to posterior and the medial to lateral length of the SASDB (Figure 3.3c). Also, an estimation was made of the surface-to-surface ratio of the subacromial (SA) and subdeltoid (SD) portions of the SASDB. Fourth, to assure maximal GHJ mobility, the RC muscles were incised perpendicular to the muscle fiber direction, two centimeters medially of the glenoid rim. To preclude impairment of the joint capsule, the part of the RC muscles located medially to the incision were freed subperiostally from the scapula. In this step of the dissection, the glenohumeral joint capsule (GHJC) and incorporated GH ligaments were spared. The joints were not vented, maintaining negative intra-articular pressure, and hence glenohumeral joint stability.[6, 14, 27] Fifth, the superficial wall of the SD portion of the SASDB was incised to assess visually the contact areas between the superficial and deep walls of the SASDB in different GHJ positions (see section 3.2.2). The bursal cavity was inspected for macroscopic changes, such as fraying or disruption of the bursal walls. After dissection all specimens displayed a full range of motion on manual examination and were without signs of GHJ instability. Finalizing the experiments, the shoulder specimens were inspected for abnormalities of the RC, the GHJC or joint surfaces. All specimens revealed congruent joint surfaces without signs of osteoarthritis.

3.2.2 Kinematic study

A custom-made three-dimensional positioning device was developed for the kinematic tests. 10 Embalmed and 4 unembalmed shoulder specimens were mounted on this device

(Figure 3.4ab). The scapula of each specimen was anchored with clamps, fixing the scapular margins to a reference plate. The medial (vertebral) border of the scapula was oriented parallel to the vertical axis of the reference plate, that in turn was perpendicular to the ground. Thus, the scapula and humerus were placed in the correct anatomical position. In this study, rotation about the longitudinal axis of the humerus is referred to as humeral rotation (HR; either internal or external rotation). A metal socket contained the humeral shaft. HR was measured with a goniometer attached to the metal socket. A telescopic device between metal socket and frame, a universal (Hookes) joint and a low-friction ball bearing provided complete freedom of humeral head translation and HR. An adjustable clamp limited caudal translation of the humerus to 1.0 cm, necessary because of the absence of deltoid muscle force. Experiments were conducted at room temperature. The specimens were kept moist throughout the experiment. Since it has been suggested that 'true abduction' of the arm should not be in the frontal plane but in the plane of the scapula, movements were defined with respect to the plane of the scapula. So, the scapular planes and axes differ from those, normally used in the clinical evaluation of shoulder movements.[16]

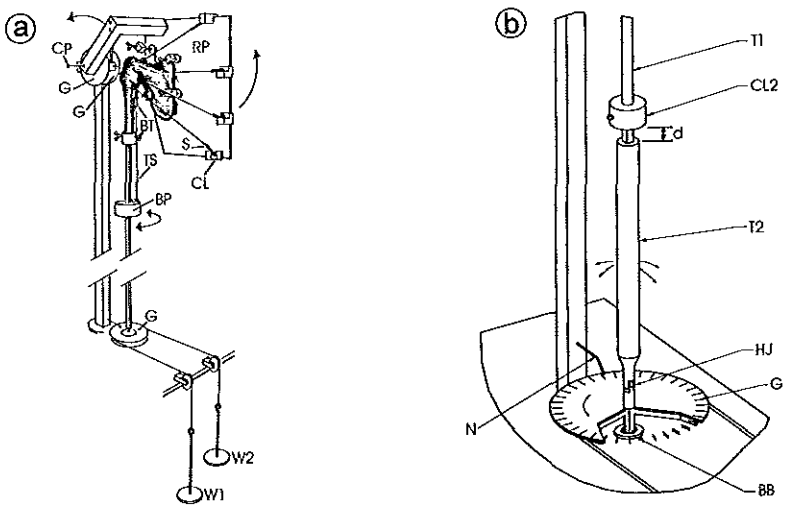


Figure 3.4a Custom made device for the kinematic tests. Scapula fixed to the reference plate. The humerus is free to rotate about its longitudinal axis. Biceps tendon load applied by weights on base plate. Pre- load applied to the rotator cuff tendons by means of isotonic spring devices (S) fixed to suture wires. Rotational torque applied by weights W1 and W2. BP=base plate; BT=biceps tendon; CL=clamps; CP=centre pin; G=goniometer; MS=metal socket; RP=reference plate; TS=Ticron suture.
Figure 3.4b Detail of the telescopic device and Hookes joint that allowed for translations of the humeral head. An adjustable clamp limited caudal translation of the humerus to 1.0 cm, necessary because of the absence of deltoid muscle force. BB=ball bearing; CL_2=special clamp limiting caudal translation (d=1.0 cm); HJ=Hookes joint; N=Needle;T1/2=telescopic device. For additional legend see Figure 3.4a.

Loading of the biceps tendon (2.25 N) and RC tendons (2.0 N) prevented slack during the experiment and provided a compressive joint force, centering the humeral head with respect to the glenoid fossa.[29] RC pre-load was applied by means of nylon sutures (Ethylon 1.O, a-traumatic), attached to isotonic spring devices at the border of the reference plate. Loading of the biceps tendon occurred by means of a similar nylon suture, woven through the biceps tendon using a modified Bunell technique. Distally, the nylon suture was attached to a base plate that slid along the telescopic device. This design allowed for biceps tendon tension in the direction of the humeral shaft. The weight of the base plate was 2.25 N.

The following definitions were used: 1) the neutral position of the GHJ refers to a vertical position of the medial (vertebral) border of the scapula (0° GH elevation) and the rotatory position of the humerus with the earlier mentioned pre-load on the RC muscles and biceps tendon (0° HR); 2) passively moving the scapula with 15°-increments from -15° to maximal elevation simulated GH elevation in the scapular plane (referred to as GH elevation); 3) GH flexion and GH extension were defined, respectively, as anterior and posterior elevation of the humerus perpendicular to the scapular plane. The specimens were photographed through arcs of motion in the above mentioned directions. Stops were made at 30°-intervals. The pictures were used to study the anatomic relationships between SASDB and related structures.

3.3 Results

3.3.1 Anatomic study

Subacromial (SA) and subdeltoid (SD) portions were present in all specimens. These portions were completely continuous in all but four specimens (6.3%); here one or two septa partially separated these two portions (Figure 3.5ab). In two specimens (3.2%) a distinct subcoracoid extension of the SASDB was present. Extending more inferiorly than the rest of the SASDB, this portion was located anteriorly to the subscapularis tendon, separate from the subscapularis recess. In the anatomic position about one-third of the SASDB was located under the coracoacromial arc, two-thirds under the deltoid muscle. In all specimens the SASDB extended in superior and medial direction under the acromion and coracoacromial ligament, its medial margin located laterally of the acromioclavicular joint (ACJ). At the anterior side of the GHJ, the SASDB extended in inferior direction, superficial to the biceps tendon sheath. In addition it extended medially between the subscapularis muscle and the coracoid process, deep to the origins of the CB and the SHBB muscles. The SASDB covered about 20% of the superior part

of the subscapularis tendon. At the posterior side of the GHJ, the SASDB extended over the anterior part of the infraspinatus tendon, covering about 25% of its surface. In all specimens, the lateral margin of the SASDB extended 1 to 1½ cm over the lateral edge of the greater tuberosity. Superiorly, the superficial wall of the SASDB was firmly attached to the inferior surface of acromion and coracoacromial ligament, anteriorly to the posterior surface of the CB and SHBB muscles (in 89% of the specimen), and anteriorly and laterally to the subdeltoid fascia. Superiorly, the deep wall of the SASDB was firmly attached to the superior surface of the RC, extending over the infraspinatus and subscapularis tendons. In all specimens, the bursal margins were loosely attached to fat tissue. The anterior to posterior length of the SASDB was 6.5 ± 2.1 cm (mean and standard deviation), the medial to lateral length 6.2 ± 1.9 cm. The surface area of the SASDB was 36 ± 6.3 cm^2 (range 30-43 cm^2). So, roughly the SASDB-mesothelium had a total surface area of 60 to 85 cm^2. The cross-sections of the GHJ showed that muscle fibers of the RC were attached to the medial margin of the SASDB.

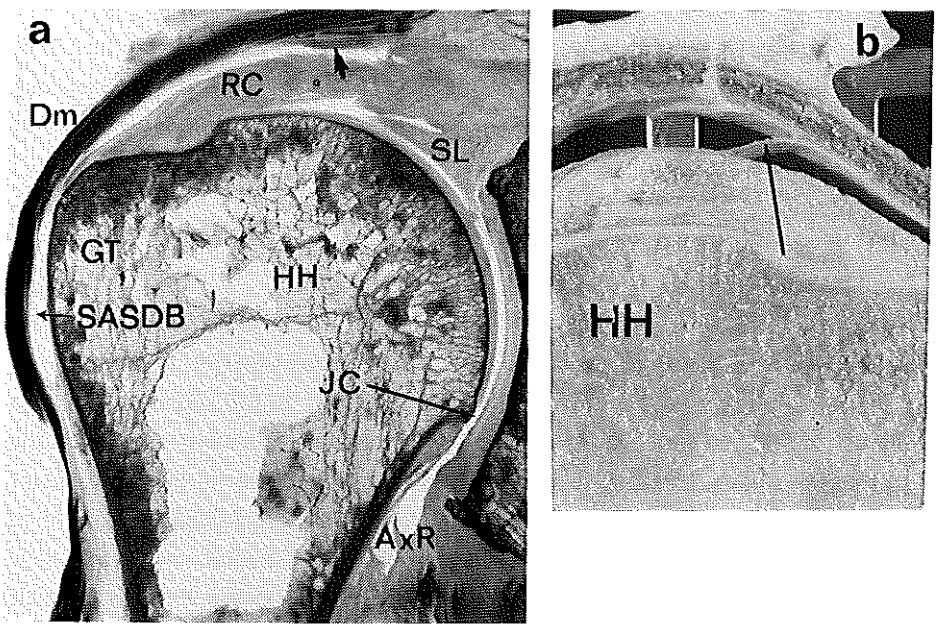


Figure 3.5a Frontal section through the right shoulder specimen of an eighty-year old male. Most likely age-related thinning of the rotator cuff has caused superior subluxation of the humeral head. AxR= axillary recess; CAL=coracoacromial ligament (arrow); Dm=deltoid muscle; GT=greater tuberosity; HH=humeral head; JC=joint cavity; SASDB=subacromial-subdeltoid bursa; SL=superior part of the glenoid labrum; RC=rotator cuff

Figure 3.5b Frontal section through the left shoulder specimen of a sixty-year old female. The intrabursal septum is indicated with an arrow. For additional legend see 3.5a

A band of fibrous tissue passed laterally along the coracoid process and followed the lateral margin of the coracoacromial ligament into the acromion. Distal to the coracoid process this band was continuous with the origin of the SHBB. The lateral margin of this band merged with the superficial layer of the SASDB. Proximal to the coracoid process, the medial margin of the band merged with the coracoacromial ligament. The mean width of this band in 45 specimens was 5.5 ± 3.3 mm (range 0.5-16 mm) (Figure 3.6). In 5/45 specimens (11%) the SHBB was not attached at all to the superficial layer the SASDB. Here, a band of muscle fibers of the CB attached to the SASDB. The width of this band measured 4.2 ± 2.2 mm (range 2-7 mm).

In 8/61 specimens (13%) inspection of the bursal cavity showed an area of fraying () of the deep wall over the supraspinatus tendon. This area of fraying of about 1-2 cm^2 was located over the critical zone[α] of the supraspinatus tendon as described by Mosely and Goldie (32) and others.(5, 28, 30) This area fraying was always associated with another area of fraying (about 1 cm^2) located at the superficial wall of the SA portion at the inferior surface of the anterior third of the acromion. In 6/61 specimens (10%), a partial tear of the RC was noted, in 2/61 (3%) a full thickness rupture, all involving the supraspinatus tendon. Their location resembled the location of the earlier mentioned area of fraying of the deep wall of the SD portion.

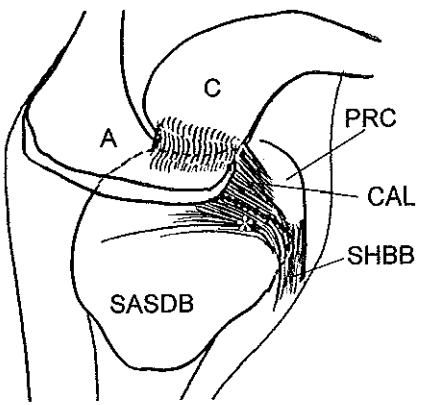


Figure 3.6 Schematic illustration of the right shoulder viewed from the antero-lateral side showing the relationship between the SASDB, SHBB, and CAL. Heavy dotted line (●●●●) represents the lateral border of the coracoacromial ligament. Laterally of this line, tendon fibers of the SHBB (and in some cases muscle fibers of the coracobrachialis muscle) form a (fibrous) band (*) that connect to the superficial wall of the SASDB. A=acromion; C=clavicula; CAL=coracoacromial ligament; PRC=coracoid process; SASDB=subacromial-subdeltoid bursa; SHBB=tendon of the short head of the biceps brachii muscle.

[α] The critical zone is an area of relatively sparse vascularity as compared to other parts of the supraspinatus tendon. It has been frequently associated with the most common site of rotator cuff tendon failure. The zone is located approximately 1-1.5 cm proximal of the supraspinatus tendon insertion.

3.2.2 Kinematic study

During most movements in the GHJ, the SASDB walls showed two distinct wrinkling patterns: either parallel or perpendicular to a vertical plane that parallels the coracoacromial arc. These different patterns are referred to as a parallel or a perpendicular pattern (Figure 3.7a-c). A parallel pattern was seen most in areas of SASDB wall relaxation, and a perpendicular pattern in areas of tensioning of the SASDB walls. Wrinkling of the superficial wall of the SD portion of the SASDB was more pronounced than wrinkling of the deep wall of the SA and SD portions.

Superiorly, a parallel pattern of the superficial wall of the SD portion occurred during GH elevation. Anteriorly, parallel wrinkling of this wall occurred during GH flexion combined with internal rotation of the humerus. During internal rotation the posterior side of the superficial wall of the SD portion tensioned. Anteriorly, a perpendicular pattern was present during external rotation of the humerus. During external rotation of the humerus the deep wall of the SD portion tensioned and a perpendicular wrinkling pattern appeared. During internal rotation of the humerus, a perpendicular pattern occurred at the posterior side of the deep wall of the SD portion. GH extension produced a reversed pattern of that occurring during forward flexion, however, this pattern was less pronounced. The GHJ position of maximal GH flexion combined with (about 15°) internal rotation of the humerus, showed a marked perpendicular wrinkling of the deep wall of the SD portion

At maximal GH elevation the lateral margin of the superficial wall approached the lateral border of the coracoacromial arc but did not slide under it. In contrast, the deep wall of the SD portion completely slid under the coracoacromial arc. From 0° to 45° GH elevation, the medial margin of the deep wall of the SA portion shifted 7.0 $\pm$ 1.3 mm in a medial direction; at maximal GH elevation, this shift was 3.1 $\pm$ 0.6 mm.

In the neutral position the area of the deep wall of the SD portion in connected to the supraspinatus tendon was located consistently anterior to the acromion and inferior and lateral to the coracoacromial ligament. During movements in the GHJ that involve elevation, this area contacted the superficial wall of the SA portion at all aspects of the coracoacromial arc. The area of contact was located anteriorly with GH extension combined with internal rotation of the humerus. Between 60° and 90° GH elevation the area of contact was located more posteriorly at the junction of the coracoacromial ligament and the acromion.

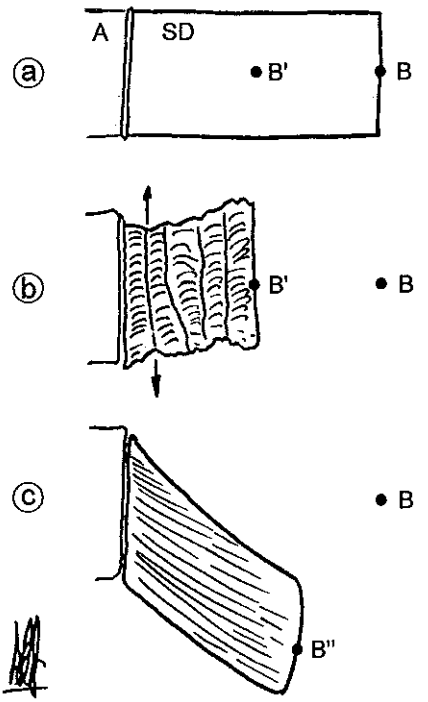


Figure 3.7a Schematic drawing of the wrinkling pattern of the during GH elevation. In the anatomic position this wall is streched over the humeral head and the rotator cuff. SD=superficial wall of the SD portion of the SASDB. A=acromion; B=lateral bursal margin; B'=expected location of B after GH elevation without rotation of the humerus.
Figure 3.7b GH elevation without humeral rotation causes accumulation of bursal near the acromion. Relaxation of the bursal wall is accompanied by wrinkling parallel to the acromion.
Figure 3.7c Due to obligatory external rotation of the humerus B moves in a lateral and posterior direction stretching the bursal wall again and causing wrinkling perpendicular to the acromion.

3.4 Discussion

3.4.1 Morphology and Nomenclature

This study showed that the SASDB is a flattened sac consisting of continuous SA and SD portions. In most specimens (94%) they were not separated by septa. This percentage is somewhat higher than that obtained by Strizak et al. (80-90%) with double-contrast bursography.[39] This method relies on the gravity-dependent distribution of radiopaque fluid or air. In all probability, the higher percentage found by us is more realistic since we probed the interior of the SASDB with a plastic rod and dissected the bursae step-wise.

Interestingly, anatomical nomenclature [1] contributes to the misconceptions that concern the morphology of the SASDB. It should be realized that, division of the SASDB in a SD and SA portion is based on a topography that exclusively relates to the *anatomic position*. Since the deep layer of the SASDB is attached to the RC tendons, parts of the deep wall of the SD portion slide under the acromion during elevation. In this position it could be named a SA portion. In contrast, the firm attachment of the superficial wall of the SASDB to the coracoacromial arc and deltoid muscle prevents this portion of the SASDB to change from a SA to a SD position, or vise versa.

3.4.2 SASDB transformation during movements of the GHJ

During GH elevation the deep wall of the SASDB follows the displacements of the connected portions of the RC. The superficial wall of the SD portion moves medially and wrinkles, but does *not* slide under the coracoacromial arc. In several medical illustrations,[17, 23] however, the superficial wall of the SD portion is shown to slide completely under the coracoacromial arc during GH elevation (Figure 3.2ab). Most likely, the use of two-dimensional drawings attributed to the earlier misleading concepts of 'bursal movements.' With two-dimensional models the transformation of the SASDB, especially at its margins, can not be correctly shown.

Following the GH dimensions,[21] 4 to 5 cm displacement of the SASDB margins occur during maximal elevation. It can be seriously questioned whether such a displacement can be tolerated (Figure 3.8). The present study showed that the displacement was much smaller. From 0° to 45° GH elevation the medial margin of the SASDB shifted about 7 mm medially. From 45° to maximal GH elevation, the medial margin shifted 4 mm laterally. How can this lateral shift be explained? Our cross-sections of the GHJ showed that muscle fibers of the supraspinatus muscle attach to the medial margin of the SASDB. We postulate that these fibers create a dynamic control system pulling the medial margin of the SASDB medially from 0° to 45° GH elevation and reeling it out laterally from 45° to maximal GH elevation. This lengthening, noted by Watson [41] in the past, is caused by the obligatory external rotation of the humerus necessary to reach maximal GH elevation (see Chapters 4 and 5). Since the deep wall of the SASDB is firmly attached to the RC, it has to follow its movements.

3.4.3 Clinical considerations

About 15 synovial bursae, have been described in the region of the shoulder.[7, 26, 42] The SASDB is the largest and the most important due to its involvement in several pathologic processes.[7, 8, 12, 13, 22, 40] Except for primary involvement of the SASDB in rheumatoid

arthritis,[37, 45] infectious disease,[9, 43] osteochondromatosis,[24, 31] and pigmented villonodular synovitis,[25] the majority of SASDB lesions is considered to be secondary to RC disorders.[22, 33] In subacromial impingement, lesions of the SASDB, RC and biceps tendon are closely related. Involvement of SASDB in subacromial impingement varies from 70-100% and mainly concerns fibrosis of its synovial tissue.[3, 36] Our study showed that macroscopic fraying of the SASDB, only relates to the impinging areas and the location of impingement related RC tears (Figure 3.9). This observation confirms the observations made by Ishii:[22] they reported that 'bursal reaction' distant to the RC tendon lesion is minimal and does not correlate with the findings at the site of the associated tendon lesion. We therefore support his term 'localized bursal reaction' as opposed to bursitis, to describe the involvement of the SASDB in subacromial impingement.

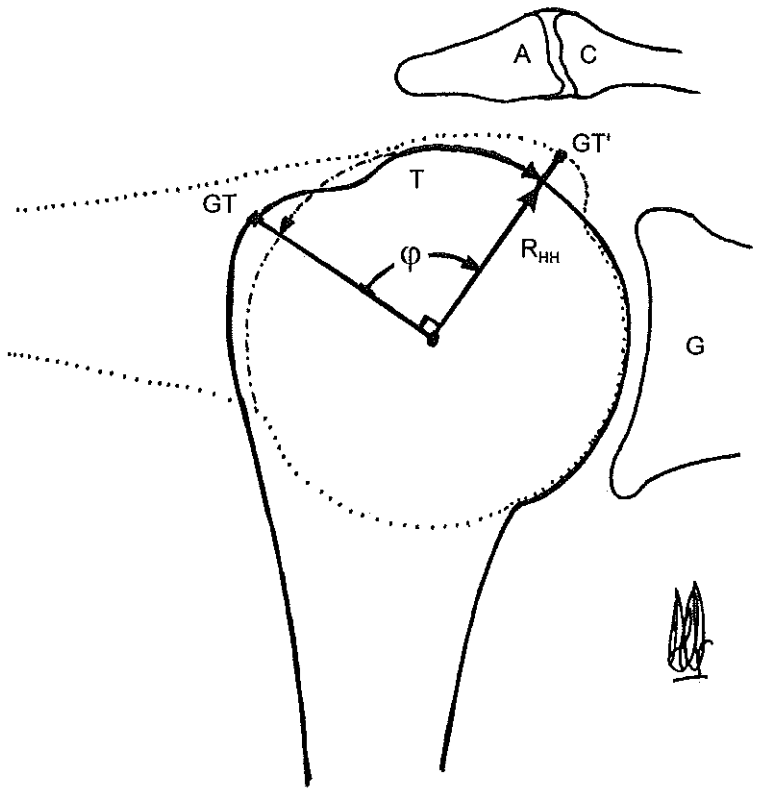


Figure 3.8 Drawing of a frontal section of the right shoulder. Theoretically, the two-dimensional representation of the trajectory of the GT (and, consequently, parts of the deep layer of the SASDB) during elevation of the arm is defined by the following equation: $T = (2\pi r.360^{0}).\varphi^{-1}$. Here, T is the trajectory of the GT (GT-GT') and associated deep layer of the SASDB, R_{HH} is the radius of curvature of the humeral head and φ represents the amount of glenohumeral abduction in the frontal plane in degrees. The radius of curvature averages 2.55 cm (range 1.85 to 2.75 cm).[21] Following the equation, T = 5.4 cm at 120° abduction and T = 4.1 cm at 90°.

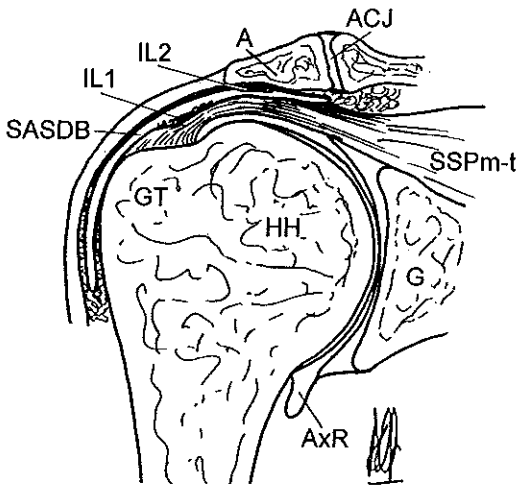


Figure 3.9. Mechanical impingement lesions of the SASDB. There are two impingement lesions; one at the side of the rotator cuff (IL1) and one at the inferior surface of the acromion and coracoacromial ligament (IL2). Actually, IL1 lies laterally and anterior of IL2. These impingement lesions only 'kiss' in the individual's impingement position. A=acromion; ACJ=acromioclavicular joint; AxR=axillary recess; HH=humeral head; G=glenoid fossa; GT=greater tuberosity; SSP_{m-t}=supraspinatus tendon muscle-tendon unit.

In practice, practitioners make a distinction between bursitis and RC tendon lesions. This distinction is based on the implicit assumption that the SASDB is not influenced by isometric contraction of the RC muscles. As a rule, bursitis is diagnosed with the following traction test. In case of pain arising during active elevation (with a painful arc) or with resisted abduction, decreases by longitudinal traction to the humerus the test is positive.[10, 20] In case no decrease of pain occurs during traction, this would favor an affliction of the supraspinatus muscle-tendon unit. However, the present study showed that the deep wall of the SASDB is firmly attached to the RC. Moreover, we showed that of supraspinatus muscle fibers directly attach to the SASDB. So, contraction of the supraspinatus will inevitably tension the adjacent wall of the SASDB. Thus, it can be questioned whether this traction test really makes a distinction between supraspinatus and SASDB lesions.

Hawkins' manoeuver [19] provokes impingement pain with maximal GH flexion and (about 15°) internal rotation of the humerus. In our study, this GHJ position produced marked perpendicular wrinkling of the deep wall of the SD portion, indicating tensioning of this wall. Perpendicular wrinkling of the walls of the SASDB also occurred during certain other clinically applied movements in the GHJ. It was most apparent during external rotation of the humerus and during GH flexion especially when combined with internal rotation. This wrinkling was most pronounced in areas of the SASDB where

impingement lesions are most frequently observed; the area centered over the supraspinatus tendon near its insertion into the greater tuberosity.[34] These findings support our clinical impression that additionally to impingement maneuvers, pain is not only provoked by impingement manoeuvers, but also at the extremes of GH flexion and external rotation of the humerus. Since the SASDB relates so closely to the RC and the coracoacromial arc it is not easy to 'design' a specific bursa-test that can be used during physical examination.

3.4.4 Biopsy of the SASDB for the diagnosis of subacromial impingement

According to Rahme,[36] a strong association exists between the presence of SASDB fibrosis and a favorable outcome of surgical subacromial decompression in patients with impingement syndrome. Lack of bursal fibrosis in these patients indicates the absence of mechanical impingement and would plea against subacromial decompression.[36] So, microscopic examination of bursal tissue specimens obtained by means of percutaneous needle biopsy or during subacromial endoscopy could be helpful in deciding whether or not to perform surgical subacromial decompression. It is important, however, that these biopsies are obtained from areas of the SASDB that are involved in subacromial impingement. The data of the present study indicate that these biopsies should be taken at two areas. First, at the area of the deep wall of the SD portion located over the critical zone of the supraspinatus tendon as described by Mosely and Goldie [32] and others.[5, 28, 30] Second, at the superficial wall of the SA portion at the inferior surface of the anterior third of the acromion.

3.5 Conclusions

1. The SASDB has SA and SD portions that were present in all specimens and fully continuous in about 95%.
2. The usual division of the SASDB in a SA and a SD portion is based on a topographical taxonomy that only relates to the *anatomic position*.
3. Transformation of the SASDB is dynamically controlled by parts of the RC, the CB, deltoid and SHBB muscles.
4. There is no basis for specific 'bursa-tests' during physical examination.

Acknowledgements

The authors thank Chris J. Snijders, Ph.D. for his biomechanical advice, Marieke van Zwienen en Marieke Joosten for their indispensable help with the kinematic study, Cor Goedegebuur for technical assistance, and Cees Entius and Jan Velkers for their assistance with the preparation of the specimens.

REFERENCES

1. **Anatomists TICo.** Nomina Anatomica. Nomina Anatomica. 6th ed. London Melbourne New York: Churchill Livingstone, 1985:
2. **Bergman AG.** Rotator cuff impingement. Pathogenesis, MR imaging characteristics, and early dynamic MR results. *Magn Reson Imaging Clin N Am* 1997;5(4):705-19.
3. **Bigliani LU, DF DA, Duralde XA, McIlveen SJ.** Anterior acromioplasty for subacromial impingement in patients younger than 40 years of age. *Clin Orthop* 1989(246):111-6.
4. **Breazeale NM, Craig EV.** Partial-thickness rotator cuff tears. Pathogenesis and treatment. *Orthop Clin North Am* 1997;28(2):145-55.
5. **Brooks CH, Revell WJ, Heatley FW.** A quantitative histological study of the vascularity of the rotator cuff tendon. *J Bone Joint Surg [Br]* 1992;74(1):151-3.
6. **Brown AO, Hoffmeyer P, An KN.** The influence of atmospheric pressure on shoulder stability. *Orthop Trans* 1990;14:259-61.
7. **Bywaters EGL.** The bursae of the body. Editorial. *Ann Rheum Dis* 1965;24:215-18.
8. **Calvert PT, Packer NP, Stoker DJ, Bayley JI, Kessel L.** Arthrography of the shoulder after operative repair of the torn rotator cuff. *J Bone Joint Surg [Br]* 1986;68(1):147-50.
9. **Co DL, Baer AN.** Staphylococcal infection of the subacromial/subdeltoid bursa. *J Rheumatol* 1990;17(6):849-51.
10. **Cyriax J.** Textbook of orthopaedic medicine. 6th ed. London: Bailliere Tindall, 1975. vol 1.
11. **Entius C, Kuiper J, Koops W, Gast de A.** A new positioning technique for comparing sectional anatomy of the shoulder with sectional diagnostic modalities. *J Int Soc Plastination* 1993;7:23-6.
12. **Farin PU, Jaroma H, Harju A, Soimakallio S.** Shoulder impingement syndrome: sonographic evaluation. *Radiology* 1990;176(3):845-9.
13. **Fukuda H, Mikasa M, Yamanaka K.** Incomplete thickness rotator cuff tears diagnosed by subacromial bursography. *Clin Orthop* 1987(223):51-8.
14. **Gibb TD, Sidles JA, Harryman DTd, McQuade KJ, Matsen FAd.** The effect of capsular venting on glenohumeral laxity. *Clin Orthop* 1991(268):120-7.
15. **Gray H.** Arthrology. In: Williams P, Warwick R, eds. Gray's Anatomy. 36th ed. Edinburgh: Churchill Livingstone, 1980:458.
16. **Greene WB, Heckman JD,** eds. The clinical measurement of joint motion. 1st ed. American Academy Of Orthopaedic Surgeons, 1994.
17. **Habermeyer P, Wiedemann E.** Pathologie und Pathomechanik. In: Habermeyer P, Krueger P, Sweibener D, eds. Schulterchirurgie. 1st ed. Munchen: Urban & Schwarzenberg, 1990:19-31.
18. **Hagens von G, Tiedemann K.** The current potential of plastination. *Anat Embryol* 1987;175:411-21.
19. **Hawkins RJ, Kennedy JC.** Impingement syndrome in athletes. *Am J Sports Med* 1980;8(3):151-8.
20. **Hollingworth G, Ellis R, Hattersly T.** Comparison of injection techniques for shoulder pain: results of a double blind, randomized study. *Br Med J* 1983;287:1339-41.
21. **Iannotti JP, Gabriel JP, Schneck SL, Evans BG, Misra S.** The normal glenohumeral relationships. An anatomical study of one hundred and forty shoulders. *J Bone Joint Surg [Am]* 1992;74(4):491-500.
22. **Ishii H, Brunet JA, Welsh RP, Uhthoff HK.** "Bursal reactions" in rotator cuff tearing, the impingement syndrome, and calcifying tendinitis. *J Shoulder Elbow Surg* 1997;6(2):131-6.

23. **Katthagen B.** Schultersonographie. Technik, Anatomie, Pathologie. Stuttgart: Georg Thieme Verlag, 1988.
24. **Ko JY, Wang JW, Chen WJ, Yamamoto R.** Synovial chondromatosis of the subacromial bursa with rotator cuff tearing. *J Shoulder Elbow Surg* 1995;4(4):312-6.
25. **Konrath GA, Nahigian K, Kolowich P.** Pigmented villonodular synovitis of the subacromial bursa. *J Shoulder Elbow Surg* 1997;6(4):400-4.
26. **Kopsch F.** Rauber's Lehrbuch der Anatomie des Menschen. Abteilung 3:Muskeln, Gefaesse. 11 ed. Leipzig: Georg Thieme, 1919.
27. **Kumar VP, Balasubramaniam P.** The role of atmospheric pressure in stabilising the shoulder. An experimental study. *J Bone Joint Surg [Br]* 1985;67(5):719-21.
28. **Ling SC, Chen CF, Wan RX.** A study on the vascular supply of the supraspinatus tendon. *Surg Radiol Anat* 1990;12(3):161-5.
29. **Lippitt S, Matsen F.** Mechanisms of glenohumeral joint stability. *Clin Orthop* 1993(291):20-8.
30. **Lohr JF, Uhthoff HK.** The microvascular pattern of the supraspinatus tendon. *Clin Orthop* 1990(254):35-8.
31. **Milgram JW, Hadesman WM.** Synovial osteochondromatosis in the subacromial bursa. *Clin Orthop* 1988(236):154-9.
32. **Mosely HF, Goldie I.** The arterial pattern of the rotator cuff of the shoulder. *J Bone Joint Surg [Br]* 1963;45:780-89.
33. **Neer CS.** Shoulder reconstruction. 1st ed. Philadelphia London Toronto Montreal Sydney Tokyo: W.B. Saunders Company, 1990.
34. **Neer CSd.** Impingement lesions. *Clin Orthop* 1983(173):70-7.
35. **Nottage WM.** Arthroscopic anatomy of the glenohumeral joint and subacromial bursa. *Orthop Clin North Am* 1993;24(1):27-32.
36. **Rahme H, Nordgren H, Hamberg H, Westerberg CE.** The subacromial bursa and the impingement syndrome. A clinical and histological study of 30 cases. *AJR Am J Roentgenol* 1993;160(3):561-4.
37. **Ruhoy MK, Tucker L, McCauley RG.** Hypertrophic bursopathy of the subacromial-subdeltoid bursa in juvenile rheumatoid arthritis: sonographic appearance. *Pediatr Radiol* 1996;26(5):353-5.
38. **Sobotta.** Atlas of human anatomy. Munich: Urban & Schwarzenberg, 1982:311. vol I.
39. **Strizak AM, Danzig L, Jackson DW, Resnick D, Staple T.** Subacromial bursography. An anatomical and clinical study. *J Bone Joint Surg [Am]* 1982;64(2):196-201.
40. **Uhthoff HK, Sarkar K.** Surgical repair of rotator cuff ruptures. The importance of the subacromial bursa. *J Bone Joint Surg [Br]* 1991;73(3):399-401.
41. **Uthoff H.** The embryology of the human locomotor system. New York Heidelberg Berlin: Springer Verlag, 1990:73-81.
42. **von Lanz T, Wachsmuth W.** Praktische Anatomie. 2nd ed. Berlin Goettingen Heidelberg: Springer Verlag, 1959. vol 1.
43. **Ward WG, Eckardt JJ.** Subacromial/subdeltoid bursa abscesses. An overlooked diagnosis. *Clin Orthop* 1993(288):189-94.
44. **Watson M.** Rotator cuff function in the impingement syndrome. *J Bone Joint Surg [Br]* 1989;71(3):361-6.
45. **Weston WJ.** The enlarged subdeltoid bursa in rheumatoid arthritis. *Br J Radiol* 1969;42(499):481-6.

CHAPTER

4

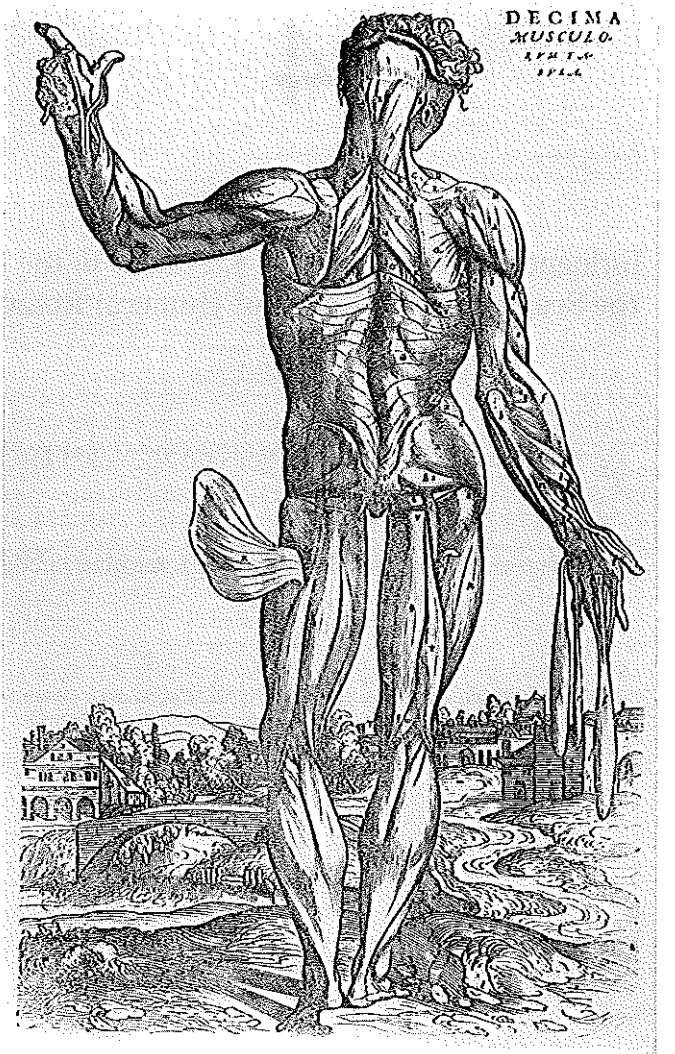
The Influence of Glenohumeral Elevation in the Plane of the Scapula on the Range of Humeral Rotation

AN ANATOMICAL AND BIOMECHANICAL STUDY

ARTHUR DE GAST, MD¶, $, CHRIS J. SNIJDERS, PH.D.¶, $, ROB STOECKART, PH.D.¶

$Dept. of Biomedical Physics and Technology, Faculty of Medicine, Erasmus University Rotterdam
¶Dept. of Anatomy, Faculty of Medicine, Erasmus University Rotterdam

Submitted for Publication


DECIMA
MUSCULO.

CHAPTER

4

The Influence of Glenohumeral Elevation in the Plane of the Scapula on the Range of Humeral Rotation

Abstract

Evaluation of the glenohumeral range of motion is an essential part of the physical examination of patients with shoulder complaints. To assess the range of internal and external rotation of the humerus (humeral rotation) through an arc of glenohumeral elevation in the plane of the scapula, two unembalmed and five embalmed shoulder specimens were used. Between 30° and 45° glenohumeral elevation all specimens reached the maximal range of humeral rotation: 144 ± 4° for the unembalmed and 155 ± 23.7° for the embalmed specimens. At maximal glenohumeral elevation (125° for the unembalmed and 109 ± 13.4° for the embalmed specimens) all humeral rotation was lost. The results were in the range of the physiological glenohumeral range of motion.

Correct diagnosis of shoulder disorders requires knowledge of the normal range of humeral rotation at different levels of glenohumeral elevation.

4.1 Introduction

Clinically, assessment of joint motion is important since the range of joint motion is an essential clue to correct diagnosis. It provides an index to the severity and progression of disorders and it serves as a tool to monitor the results of treatment.[19] Traditionally, clinical evaluation of shoulder motions focuses primarily on two planes of motion; the sagittal for flexion or extension and the coronal for abduction or adduction.[1,24] Orthopaedic surgeons specialized in treating shoulder problems usually limit assessment of shoulder motion to forward elevation (flexion), external rotation with the arm in 0°

abduction (coronal plane elevation), external rotation with the arm in 90° abduction, and posterior reach (internal rotation with the arm at the side).[19]

In patients with glenohumeral joint (GHJ) instability the clinical importance of humeral rotation (HR: either internal or external rotation of the humerus) has been emphasized.[3, 7, 12, 35, 43, 50] Mostly, damage to the glenohumeral (GH) ligaments from trauma is caused by forces that bring about abnormal HR, i.e. more than normal external or internal rotation of the humerus. The combination of abduction, extension and external rotation of the humerus may result in anterior dislocation. Axial loading of the adducted and internally rotated humerus may cause posterior dislocation.[3, 25] Obviously, the trauma mechanisms of these injuries involve HR, yet, classification of the severity of these lesions is limited to the assessment of humeral head translation.[21, 22, 29, 47-49]

Although it is generally accepted that the range of HR depends on GH elevation, this has not been quantified. The purpose of the present study is to provide a clinical guideline for the evaluation of the range HR. Therefore, we assessed the influence of GH elevation on the range of HR in normal shoulder specimens. Since it has been agreed that 'true abduction' of the arm should not be in the frontal plane but in the plane of the scapula,[14, 28, 45] GHJ motions in this study refer to the scapular plane. Consequently, the axes of the GHJ differ from those normally used in the clinical evaluation of arm movements (Figure 4.1).[19]

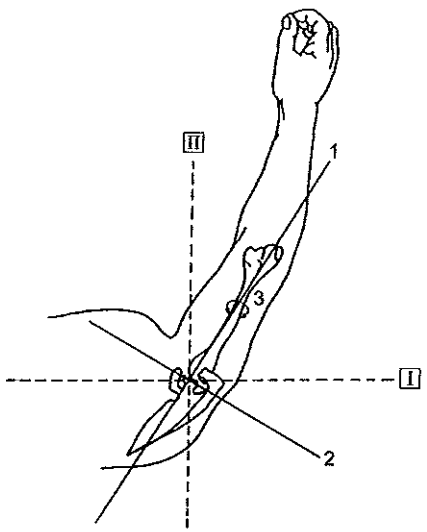


Figure 4.1 Schematic drawing of the right shoulder region and arm, showing the differences between shoulder movements referred to either the anatomical planes (I and II; dotted lines) or the scapular plane (1 and 2; full lines). For additional information see legend figure 2.11, Chapter 2 (Modified from CS Neer, 1990)

4.2 Materials and Methods

4.2.1 Method of Dissection and specimen preparation

Dissection was performed on two unembalmed and five embalmed shoulder specimens (four male, three female; 76.9 ± 6.6 years of age; range 70 - 86 years), four left and three right shoulders. The unembalmed specimens were obtained from one human cadaver twenty-eight hours after death. These shoulders were frozen at -40° C and thawed twelve hours before dissection at room temperature. Forty-eight to sixty hours postmortem the other specimens were embalmed by vascular perfusion with a medium containing: 50g phenol 99%, 20g $MgSO_4$, 20g $NaSO_4$, 10g NaCl, 60ml formaldehyde 37%, 60ml glycerin, H_2O ad 1000ml. The specimens were kept in containers filled with phenol (30 g/l) for six weeks. Subsequently, the specimens were stored in phenoxy-ethanol (10ml/l) at a temperature of 14°C for three months. The shoulder specimens were stripped of all muscles, preserving the rotator cuff muscle-tendon units, GHJ capsule, and the tendon of the long head of the biceps brachii muscle (biceps tendon).

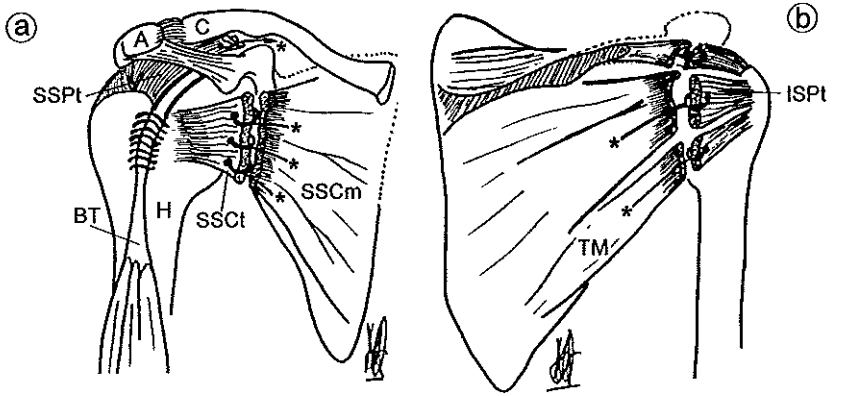


Figure 4.2a Anterior view of the right shoulder, showing the rotator cuff. The subscapularis and supraspinatus muscle-tendon units are reflected. A=acromion; BT=biceps tendon; C=clavicula; H=humerus; ISPt= infraspinatus tendon; SSCm=subscapularis muscle; SSCt=subscapularis tendon; SSPt=supraspinatus tendon.
Figure 4.2b Posterior view of the right shoulder showing the superior and posterior rotator cuff muscle-tendon units and the direction of the suture wires (*) pulling the tendons. TM= teres minor. For additional legend see figure 4.2a.

The joints were not vented, maintaining negative intra-articular pressure, and hence GHJ stability.[4, 17, 30] To assure maximal mobility, the rotator cuff muscles were incised perpendicular to the muscle fiber direction, two centimeters medially of the glenoid rim. To preclude impairment of the joint capsule, the part of the rotator cuff muscles located medially to the incision were freed subperiostally from the scapula (Figure 4.2ab). After

the dissection all specimens displayed a full range of motion (ROM) without signs of GH instability on manual examination. Finalizing the experiments, the shoulder specimens were further dissected and inspected for abnormalities of the rotator cuff, joint capsule, and joint surfaces. All specimens revealed normal soft tissue structures and normal joint surfaces without signs of osteoarthritis.

4.2.2 Instrumentation and Kinematic Tests

For the custom-made three-dimensional positioning device see Figure 4.3ab. The scapula of each specimen was anchored with clamps fixing the scapular margins to a reference plate. The medial (vertebral) border of the scapula was oriented parallel to the vertical axis of the reference plate, which was perpendicular to the ground. Thus, the scapula and humerus were placed in the correct anatomic position. The reference plate was free to rotate about an anterior to posterior axis centered over the center of the humeral head. A specific metal socket contained the humeral shaft in a vertical position. In this study, rotation about the axis of the humerus is referred to as HR. HR was measured with a specially designed goniometer attached to the metal socket. A telescopic device between metal socket and frame, a universal (Hookes) joint and a low-friction ball bearing provided complete freedom of humeral head translation and internal and external rotation with respect to the glenoid cavity. An adjustable clamp limited caudal translation of the humerus to 1.0 cm, necessary because of the absence of deltoid muscle force. Experiments were conducted at room temperature. The specimens were kept moist throughout the experiment.

Loading of the biceps tendon (2.25 N) and rotator cuff tendons (2.0 N) prevented slack during the experiment and provided a compressive joint force, centering the humeral head with respect to the glenoid cavity.[31] Rotator cuff load was applied by means of nylon sutures (Ethylon 1.O, a-traumatic), attached to isotonic springs at the border of the reference plate. Loading of the biceps tendon occurred by means of a similar nylon suture, woven through the tendon using a modified Bunnell technique. Distally, this suture was attached to a base plate that slid along the telescopic device. This design allowed for biceps tendon tension in the direction of the humeral shaft. The weight of the base plate was 2.25 N.

The neutral position of the GHJ refers to a vertical position of the medial (vertebral) border of the scapula (0° GH elevation) and a rotatory position of the humerus with the above mentioned load on the rotator cuff muscles and biceps tendon (0° HR). Torsional rotation was defined as the rotation of the humerus caused by a constant torque of 1.8 Nm applied to the goniometer, causing either internal or external rotation of the

humerus. This torque was used since a preliminary study showed that it caused maximal humeral rotation within 10 seconds. Internal rotation was marked (in degrees) with a positive value, external rotation with a negative value. GH elevation was simulated by moving the scapula through -15°, 0°, 15°, 30°, 45°, 60°, 75°, 90°, 105°, 120° and maximal elevation. At each of these levels, the range of HR was measured.

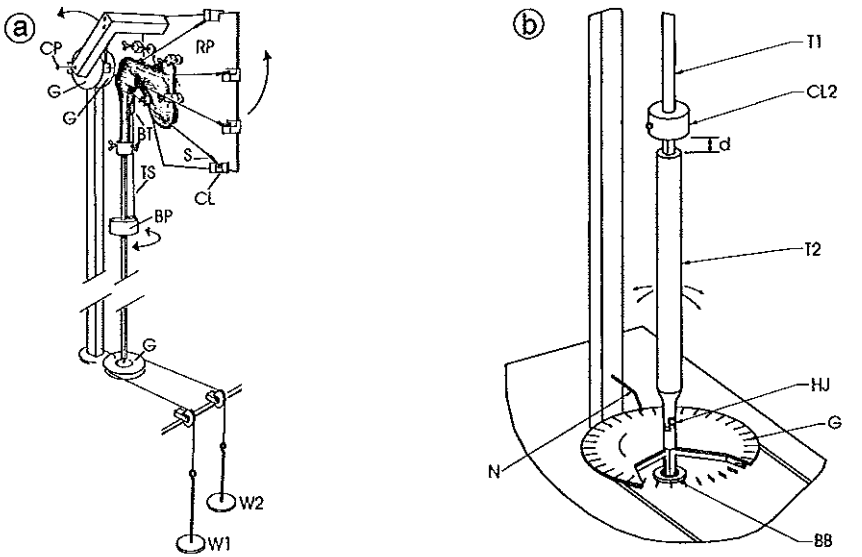


Figure 4.3a Schematic drawing of the custom-made device to measure humeral rotation (HR) through an arc of glenohumeral (GH) elevation. BP=base plate; BT=biceps tendon; CL=adjustable clamps holding the scapula to the reference plate; CP=one of two movable pins to center the humeral head as the scapula was mounted on the reference plate; G=goniometer; *=suture wires; HJ=Hookes joint; RP=reference plate; S=isotonic springs; SC=scapula; TS=ticron suture; $W_{1\text{-}2}$=weights to generate HR.
Figure 4.3b Detail of the telescopic device and Hookes joint that that allowed for translations of the humeral head. An adjustable clamp limiting caudal translation of the humerus (d=1.0 cm), necessary because of the absence of deltoid muscle force. BB=ball bearing; CL_2=special clamp limiting caudal translation; HJ=Hookes joint; N=Needle for reading the goniometer; T1/T2=telescopic device. For additional the legend see Figure 4.3a.

4.3 Results

In all specimens, the relationship between GH elevation and HR showed a similar pattern, although the magnitude varied. For a comparison of an unembalmed and an embalmed specimen, see Figure 4.4. The results of the embalmed specimens are graphically presented in Figure 4.5. In Table 4.1 the data of the embalmed and the embalmed specimens are separately listed.

The mean range of GH elevation of the unembalmed specimens was -15° to 125°. At 30° GH elevation, the mean range of HR of the unembalmed specimens reached a maximum of 144°. At maximal GH elevation both specimens lost all HR, with the humerus in a fixed position of 55° external rotation.

For the embalmed specimens the mean range of GH elevation was -15° to 109 ± 13.4°. At 30° GH elevation four specimens reached their maximal range of HR, one specimen at 45°. The mean maximal range of HR was 155 ± 23.7°. At their individual maximal GH elevation the embalmed specimen lost all HR, with the humerus in a fixed position of 60°-72° (mean 65°) external rotation.

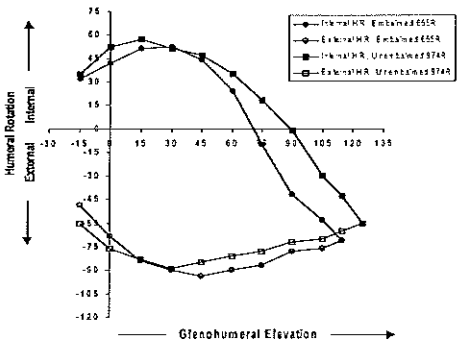


Figure 4.4 Graphical representation of the range of HR of an embalmed specimen (№ 665R) and an unembalmed specimen (№ 974R) showing the resemblance of the kinematic patterns. X-axis represents full-range of GH elevation in the scapular plane. Y-axis represents the amount of HR; internal rotation marked with a positive value, external rotation with a negative value. Ranges of motion in degrees.

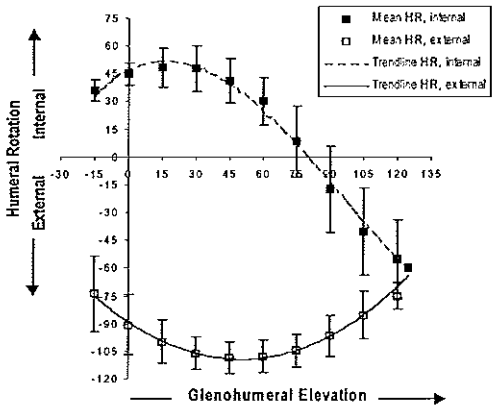


Figure 4.5 Graphical representation of the mean, standard deviation and trend of the range of HR of the embalmed specimens through an arc of GH elevation (see figure 4.4 for explanation of the axes).

Table 4.1. Relationship between Glenohumeral Elevation and Humeral Rotation in Unembalmed and Embalmed Shoulder Specimens

	Humeral Rotation					
	Internal Rotation			External Rotation		
Glenohumeral Elevation§	Mean UE†	Mean (SD) E‡	Median E‡‡	Mean UE†	Mean (SD) E‡	Median E‡‡
-15	32.5	35.8 (5.7)	38	-61.5	-73.8 (20.4)	-80
0	51	45 (6.2)	47	-78.5	-90.6 (16.3)	-94
15	57	48.4 (10.7)	51	-85	-99.8 (11.8)	-98
30	53	47.8 (12.4)	52	-91	-106 (9.0)	-106
45	48.5	41.2 (12.1)	41	-87.5	-108.6 (8.5)	-111
60	38	30 (13.0)	27	-84.5	-107.8 (8.8)	-112
75	21.5	8.2 (19.0)	0	-81.5	-104.8 (8.9)	-108
90	1	-17.6 (23.3)	-23	-75.5	-96.6 (11.1)	-101
105	-26	-40.3 (23.8)	-38.5	-72.5	-85.5 (12.9)	-86
120	-38	-55 (21.2)	-55	-63.5	-75 (7.1)	-75
125	-55	-60		-55	-60	

§ Glenohumeral Elevation in the plane of the scapula, in degrees
† Mean of the two unembalmed (UE) specimens, in degrees
‡ Mean of the five embalmed (E) specimens, in degrees
‡‡ Median and (Standard Deviation) of the five embalmed specimens, in degrees

4.4 Discussion

4.4.1 GH elevation-dependent range of HR

Our data show that the relationship between GH elevation and the range of HR has two distinct characteristics. First, between 30° and 45° GH elevation, the range of HR reached its maximum. Second, above 45° GH elevation, the range of HR strongly diminished and at maximal GH elevation all HR was lost (Figure 4.4). These results parallel the general opinion that at mid-elevation the GHJ capsule and incorporated GH ligaments are slackest, and permit the greatest range of HR.[8, 31] Furthermore, loss of HR at maximal GH elevation agrees with the following observation. At maximal GH elevation, the GHJ reaches its maximally close packed position [18] or 'zero position',[45] which is considered the most stable joint position.[15] New in our study is the

quantification of the 'zero position' of the GHJ and of the relation between HR and GH elevation through a complete arc of scapular plane motion.

The influence of GH elevation on the range of HR can be explained by the GH position-dependent regional tensioning of the passive restraints, i.e. the GHJ capsule and incorporated GH ligaments. According to Ferrari,[13] below 60° GH elevation, the superior region of the GHJ capsule (including the coracohumeral and superior glenohumeral ligaments) limits the range of external rotation of the humerus. Above 60°, this region of the GHJ capsule slackens and allows for further external rotation.[13] We observed that at 0° GH elevation the superior region of the GHJ capsule strongly limited external rotation. Between 0° and 90° GH elevation this superior region got progressively slacker, potentially allowing for a gradual increase of external rotation. In contrast with Ferrari's observation we measured a decrease of the range of external rotation above 45° GH elevation (Figures 4.4 and 4.5). How can this difference be explained? The superior capsule's function in restraining the range of external rotation can be shown *in vivo*. The range of external rotation is limited with the arm at the side, larger at 60° arm elevation, and still larger at 90°.[13, 35] This holds for both arm elevation in the frontal plane and in the scapular plane (Figure 4.6a). It should be noted that the gradual increase of external rotation during elevation of the arm reflects movements in both the GH joint and scapulothoracic mechanism. So, an increase of external rotation of the arm above 45° GH elevation reflects an increase of scapulothoracic movement (Figure 4.6b). Above approximately 45° arm elevation the scapula strongly rotates laterally and shifts anteriorly along the rib cage, contributing to the range of external rotation of the arm. Thus, to get an accurate assessment of the GH range of external rotation above 45° GH elevation, the examiner should either determine scapulothoracic movements by palpation or eliminate scapulothoracic motion by manual fixation of the scapula.

4.4.2 Range of motion of the shoulder specimens

Another important finding of this study was that adequately prepared embalmed specimens are useful for kinematic studies of the GHJ; the relationship between GH elevation and range of HR in the embalmed specimens parallels that of the unembalmed ones. This justifies the use of embalmed human bodies in an experimental design aimed at analyzing GHJ ROM through an arc of GH elevation. Furthermore, the ROM of both the embalmed and unembalmed specimens corresponds well with ranges of motion measured *in vivo*.[19, 23] Freedman and Munro [14] reported a maximal arm elevation of 167 ± 7.6°, in which the GHJ contributed 107.5 ± 9.1°. In the present study, mean

maximal GH elevation of the embalmed specimens was 109 ± 13.4°. *In vitro* ranges of HR are difficult to compare with *in vivo* ranges of HR. Those *in vivo* data [2, 12, 19, 20] that are available are not precise enough to be applied to the data of the present study, designed to measure isolated HR through a complete arc of GH elevation.

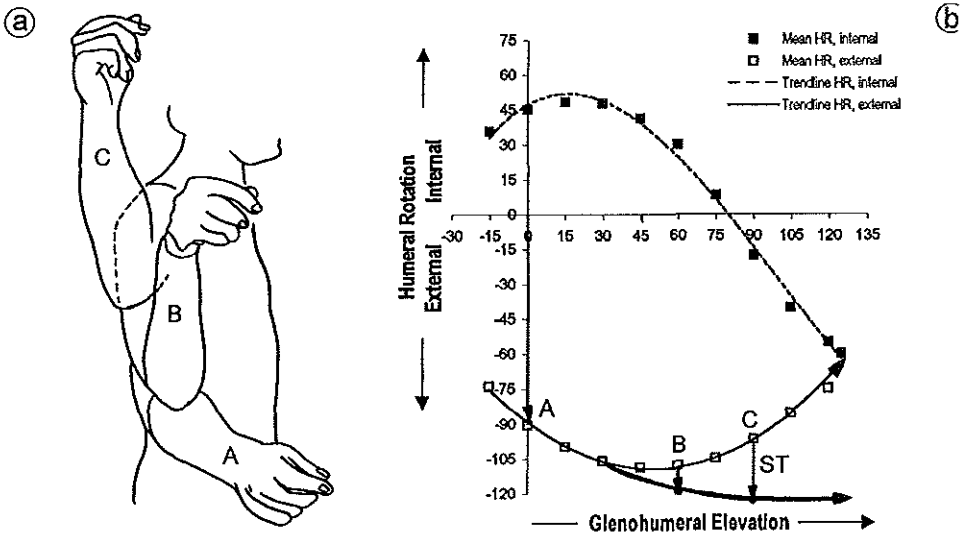


Figure 4.6a Illustration of the superior capsule's restraint on external rotation. A. At 0° abduction the range of external rotation of the humerus arm is limited, a greater range can be obtained at 60° GH abduction (B), and still more at 90° (C). (Modified and redrawn from Ferrari, 1990)

Figure 4.6b Adaptation from figure 4.4 showing the scapulothoracic (ST) contribution to the range of external rotation during elevation of the arm. The amount of external rotation of the arm above 45° GH elevation is estimated on measurements taken from healthy volunteers. Note that although the total range of external rotation of the arm increases, external rotation in the GHJ decreases above 45° GH elevation. Obviously, the contribution of the ST movement increases. Ranges of motion in degrees.

4.4.3 Maximal glenohumeral elevation and obligatory humeral rotation

The results of the present study deepen our understanding of the amount and the direction of HR that is 'obligatory' to reach maximal GH. In the 1930s, investigators stated that external rotation of the humerus is required to reach maximal GH elevation.[32, 34] This theory has been qualitatively [15] and quantitatively [5] confirmed. According to Browne at al.[5] maximal elevation of the humerus requires 35° external rotation. In our study approximately 65° *external* rotation of the humerus had to be made. This difference in the amount of obligatory external rotation can be explained by differences in study design. We studied maximal GH elevation in the plane of the scapula, Brown et al. in several planes of elevation anterior to the scapular plane. Furthermore, we showed that the amount and direction of obligatory HR depends on the starting position of the humerus. For examples see Figure 4.7.

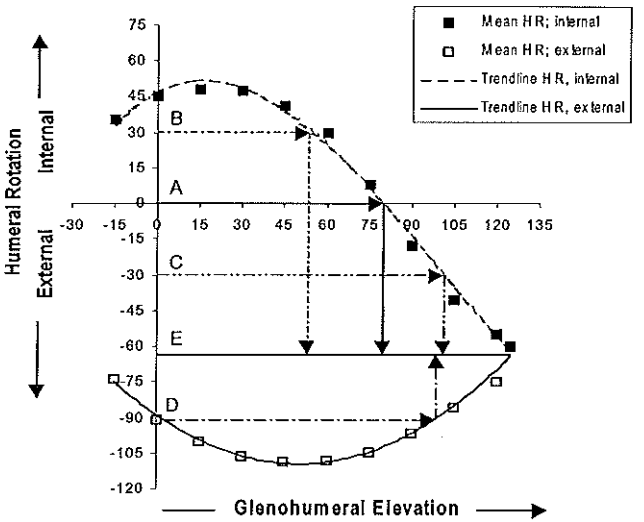


Figure 4.7 Graphical representation showing the mean range of HR of the embalmed specimen, and the relation between rotatory starting position of the humerus and the direction and amount of obligatory HR during GH elevation. Starting from the neutral position (A) allows for approximately 80° GH elevation, here about 60° external rotation of the humerus is necessary for achieving full GH elevation. Starting at (B) will necessitate more than 90° external rotation for achieving full GH elevation, starting from (C) 30° external rotation. However, starting from (D) necessitates 30° *internal* rotation. Starting from (E) allows for full GH elevation without HR. Ranges of motion in degrees.

It can be questioned why HR is obligatory for maximal GH elevation? Several factors can be identified. First, to provide more 'articular cartilage on articular cartilage' contact.[27] Second, to protect soft tissue attachments at the anatomic neck of the humerus from impinging on the glenoid rim (Figure 4.8),[26, 27] and, third, to reach the maximally closed packed position for optimum stability.[18, 31, 45] For maximal GH elevation, several passive factors are of importance: the geometry of the joint surfaces,[27, 42, 46] capsuloligamentous structures,[15] the tendon of the long head of the biceps brachii muscle (biceps tendon),[10] negative intra-articular pressure [4, 17, 30] and the adhesive properties of the synovial fluid.

Since our study showed that ranges of GH elevation and HR influence each other reciprocally, knowledge of this relationship is essential for the (clinical) assessment of GHJ mobility. For example, quantification of HR required for maximal GH elevation helps to explain the relationship between limited GH elevation and limited HR as seen in frozen shoulder and after operations limiting the range of HR.

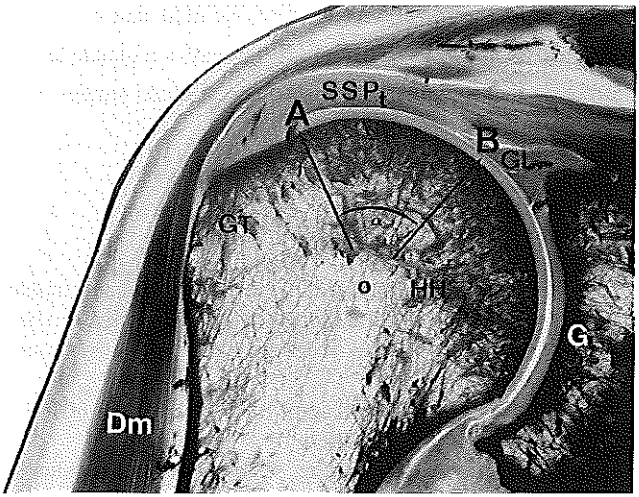


Figure 4.8 Anterior view of a plastinated cross section of the left shoulder, showing an arc (α) of 65° available for abduction before the soft tissue attachments at the anatomical neck of the humerus at point A begin to make contact with the glenoid labrum at point B. Dm=deltoid muscle; G=glenoid fossa; GT=greater tuberosity; GL=glenoid labrum; HH=humeral head; SSP_t=supraspinatus tendon.

4.4.4 Clinical assessment of GHJ instability

HR plays a role in practically all injuries to the shoulder that lead to damage of the GHJ capsule.[37, 39, 40, 48, 49] Since, specific segments of the GHJ capsule act as GH position-dependent ligamentous restraints,[6, 9, 13] certain GHJ positions will jeopardize one segment of the GHJ capsule more than the other.[3, 7, 25, 38-41, 48, 49] In case a particular segment of the GHJ capsule is damaged by excessive HR at a certain degree of GH elevation, excessive HR is expected at that particular degree of GH elevation. This can be easily assessed during physical examination of the shoulder. In contrast, special tests to assess the severity of GHJ capsule damage focus on apprehension and drawer signs.[α 21, 33, 47-49, 51] However, traditional anterior and posterior apprehension and drawer tests have limited success in assessing the severity and direction of GHJ instability.[11, 16, 36, 44] Although drawer tests have been refined,[16] they remain hard to perform, are observer depended and clinically subjective.[50] Furthermore, these tests are *indirect* measures of the actual

[α]Apprehension tests rely on patient 'apprehension', and drawer tests rely on joint laxity and muscle relaxation of the patient. Since laxity of the passive constraints of the GHJ is an essential feature of shoulder motion, it is difficult to draw a line between normal and pathologic laxity. Furthermore, the degree of laxity may vary considerably between individuals [2, 50] and, therefore, a distinction must be made between instability and hyperlaxity. Here GHJ laxity is defined as excessive translation of the humeral head on the glenoid fossa in the absence of clinical symptoms or pathological changes. GHJ instability is defined as asymmetrical laxity associated with clinical symptoms on pathological changes.

position-dependent GHJ hypermobility. Therefore, we think that is worthwhile in case of trauma to the shoulder to assess the range of HR through a complete arc of GH elevation both in the affected and the unaffected shoulder. Future studies will address this supposition.

4.5 Conclusions

1. The mobility of adequately prepared embalmed human shoulder specimens corresponds well with ranges of GHJ motion that are measured *in vivo*.
2. The range of internal and external rotation of the humerus strongly depends on glenohumeral elevation.
3. The amount and direction of HR necessary to reach maximal GH elevation depends on the rotatory position of the humerus.
4. The outcome of the present study is relevant for the clinical evaluation of the range of HR through a complete arc of GH elevation. After all, humeral rotation is an essential component for the clinical assessment of the severity and the direction of GHJ instability.

Acknowledgements

The authors thank Cor Goedegebuur for technical assistance, Cees Entius and Jan Velkers for their assistance with the preparation of the specimens.

References

1. **Basmajan JV.** Muscles alive. Their fuctions revealed by elctromyography. Baltimore London Sidney: Williams & Wilkens, 1985:19-64.
2. **Boone DC, Azen SP.** Normal range of motion of joints in male subjects. *J Bone Joint Surg [Am]* 1979;61(5):756-9.
3. **Branch TP, Lawton RL, Iobst CA, Hutton WC.** The role of glenohumeral capsular ligaments in internal and external rotation of the humerus. *Am J Sports Med* 1995;23(5):632-7.
4. **Brown AO, Hoffmeyer P, An KN.** The influence of atmospheric pressure on shoulder stability. *Orthop Trans* 1990;14:259-61.
5. **Browne AO, Hoffmeyer P, Tanaka S, An KN, Morrey BF.** Glenohumeral elevation studied in three dimensions. *J Bone Joint Surg [Br]* 1990;72(5):843-5.
6. **Clark J, Sidles JA, Matsen FA.** The relationship of the glenohumeral joint capsule to the rotator cuff. *Clin Orthop* 1990(254):29-34.
7. **Cofield RH, Nessler JP, Weinstabl R.** Diagnosis of shoulder instability by examination under anesthesia. *Clin Orthop* 1993(291):45-53.
8. **Curl LA, Warren RF.** Glenohumeral joint stability. Selective cutting studies on the static capsular restraints. *Clin Orthop* 1996(330):54-65.
9. **de Gast A, Raissadat K, Snijders CJ, Stoeckart R.** Effects of glenohumeral capsular contracture on internal and external rotation of the humerus and shoulder elevation. *Submitted for publication* 1997.

10. **de Gast A, Snijders CJ, Stoeckart R.** Role of the tendon of the long head of the biceps brachii muscle in humeral rotation control. An anatomical and biomechanical study. *Accepted for publication J Shoulder Elbow Surg* 1998.
11. **DePalma AF.** Surgery of the shoulder. 2nd ed. Philadelphia Toronto: J.B. Lippincott Company, 1972.
12. **Ellenbecker TS, Roetert EP, Piorkowski PA, Schulz DA.** Glenohumeral joint internal and external rotation range of motion in elite junior tennis players. *J Orthop Sports Phys Ther* 1996;24(6):336-41.
13. **Ferrari DA.** Capsular ligaments of the shoulder. Anatomical and functional study of the anterior superior capsule. *Am J Sports Med* 1990;18(1):20-4.
14. **Freedman L, Munro RR.** Abduction of the arm in the scapular plane: scapular and glenohumeral movements. A roentgenographic study. *J Bone Joint Surg [Am]* 1966;48(8):1503-10.
15. **Gagey O, Bonfait H, Gillot C, Hureau J, Mazas F.** Anatomic basis of ligamentous control of elevation of the shoulder (reference position of the shoulder joint). *Surg Radiol Anat* 1987;9(1):19-26.
16. **Gerber C, Ganz R.** Clinical assessment of instability of the shoulder. With special reference to anterior and posterior drawer tests. *J Bone Joint Surg [Br]* 1984;66(4):551-6.
17. **Gibb TD, Sidles JA, Harryman DTd, McQuade KJ, Matsen FAd.** The effect of capsular venting on glenohumeral laxity. *Clin Orthop* 1991(268):120-7.
18. **Gray H.** Arthrology. In: Williams P, Warwick R, eds. Gray's Anatomy. 36th ed. Edinburgh: Churchill Livingstone, 1980:458.
19. **Greene WB, Heckman JD,** eds. The clinical measurement of joint motion. 1st ed. American Academy Of Orthopaedic Surgeons, 1994.
20. **Gunal I, Kose N, Erdogan O, Gokturk E, Seber S.** Normal range of motion of the joints of the upper extremity in male subjects, with special reference to side. *J Bone Joint Surg [Am]* 1996;78(9):1401-4.
21. **Harryman DTd, Sidles JA, Harris SL, Matsen FAd.** The role of the rotator interval capsule in passive motion and stability of the shoulder. *J Bone Joint Surg [Am]* 1992;74(1):53-66.
22. **Hawkins RJ, Schutte JP, Janda DH, Huckell GH.** Translation of the glenohumeral joint with the patient under anesthesia. *J Shoulder Elbow Surg* 1996;5(4):286-92.
23. **Hoogland PV.** De bovenste extremiteit. In: Lohman AHM, ten Donkelaar HJ, eds. Klinische anatomie en embryologie. 1 st ed. Utrecht: Wetenschappelijke Uitgeverij Bunge, 1997:661-748.
24. **Inman VT, Saunders JB, Abbott LC.** Observations of the function of the shoulder joint. 1944 [classical article]. *Clin Orthop* 1996(330):3-12.
25. **Itoi E, Kuechle DK, Newman SR, Morrey BF, An KN.** Stabilising function of the biceps in stable and unstable shoulders [published erratum appeared in J Bone Joint Surg Br 1994 Jan;76(1):170]. *J Bone Joint Surg [Br]* 1993;75(4):546-50.
26. **Jobe CM.** Superior glenoid impingement. Current concepts. *Clin Orthop* 1996(330):98-107.
27. **Jobe CM, Ianotti JP.** Limits imposed on glenohumeral motion by joint geometry. *J Shoulder Elbow Surg* 1995;4:281-85.
28. **Johnston T.** The movements of the shoulder. A plea for the use of the 'plane of the scapula' as the plane of reference for movements occuring at the humero-scapular joint. *British J Surg* 1937;25:252-60.
29. **Jorgensen U, Bak K.** Shoulder instability. Assessment of anterior-posterior translation with a knee laxity tester. *Acta Orthop Scand* 1995;66(5):398-400.

30. **Kumar VP, Balasubramaniam P.** The role of atmospheric pressure in stabilising the shoulder. An experimental study. *J Bone Joint Surg [Br]* 1985;67(5):719-21.
31. **Lippitt S, Matsen F.** Mechanisms of glenohumeral joint stability. *Clin Orthop* 1993(291):20-8.
32. **Martin CP.** A note on the movements of the shoulder joint. *Brit J Surg* 1932;20:61-6.
33. **McFarland EG, Torpey BM, Curl LA.** Evaluation of shoulder laxity. *Sports Med* 1996;22(4):264-72.
34. **McGregor L.** Rotation at the shoulder. A critical injury. *Brit J Surg* 1937;24:425-38.
35. **Neer CS.** Shoulder reconstruction. 1st ed. Philadelphia: W.B. Saunders Company, 1990.
36. **Neer CSd, Foster CR.** Inferior capsular shift for involuntary inferior and multidirectional instability of the shoulder. A preliminary report. *J Bone Joint Surg [Am]* 1980;62(6):897-908.
37. **O'Brien S, Schwartz RS, Warren RF, Torzilli PA.** Capsular restraints to anterior-posterior motion of the abducted shoulder: a biomechanical study. *J Shoulder Elbow Surg* 1995;4(4):298-308.
38. **Ovesen J, Nielsen S.** Anterior and posterior shoulder instability. A cadaver study. *Acta Orthop Scand* 1986;57(4):324-7.
39. **Ovesen J, Nielsen S.** Posterior instability of the shoulder. A cadaver study. *Acta Orthop Scand* 1986;57(5):436-9.
40. **Ovesen J, Nielsen S.** Stability of the shoulder joint. Cadaver study of stabilizing structures. *Acta Orthop Scand* 1985;56(2):149-51.
41. **Ovesen J, Sojbjerg JO.** Lesions in different types of anterior glenohumeral joint dislocation. An experimental study. *Arch Orthop Trauma Surg* 1986;105(4):216-8.
42. **Rajendran K.** The rotary influence of articular contours during passive glenohumeral abduction. *Singapore Med J* 1992;33(5):493-5.
43. **Rosenberg BN, Richmond JC, Levine WN.** Long-term followup of Bankart reconstruction. Incidence of late degenerative glenohumeral arthrosis. *Am J Sports Med* 1995;23(5):538-44.
44. **Rowe CR, Zarins B.** Recurrent transient subluxation of the shoulder. *J Bone Joint Surg [Am]* 1981;63(6):863-72.
45. **Saha AK.** The classic. Mechanism of shoulder movements and a plea for the recognition of "zero position" of glenohumeral joint. *Clin Orthop* 1983(173):3-10.
46. **Soslowsky LJ, Flatow EL, Bigliani LU, Mow VC.** Articular geometry of the glenohumeral joint. *Clin Orthop* 1992(285):181-90.
47. **Terry GC, Hammon D, France P, Norwood LA.** The stabilizing function of passive shoulder restraints. *Am J Sports Med* 1991;19(1):26-34.
48. **Turkel SJ, Panio MW, Marshall JL, Girgis FG.** Stabilizing mechanisms preventing anterior dislocation of the glenohumeral joint. *J Bone Joint Surg [Am]* 1981;63(8):1208-17.
49. **Warner JJ, Deng XH, Warren RF, Torzilli PA.** Static capsuloligamentous restraints to superior-inferior translation of the glenohumeral joint. *Am J Sports Med* 1992;20(6):675-85.
50. **Warner JJ, Micheli LJ, Arslanian LE, Kennedy J, Kennedy R.** Patterns of flexibility, laxity, and strength in normal shoulders and shoulders with instability and impingement. *Am J Sports Med* 1990;18(4):366-75.
51. **Williams GR.** Multidirectional instability. In: Warner JJP, Ianotti RP, Gerber C, eds. Complex and revision problems in shoulder surgery. Phaladelphia: Lipcott-Raven Publishers, 1997:85-98.

CHAPTER

5

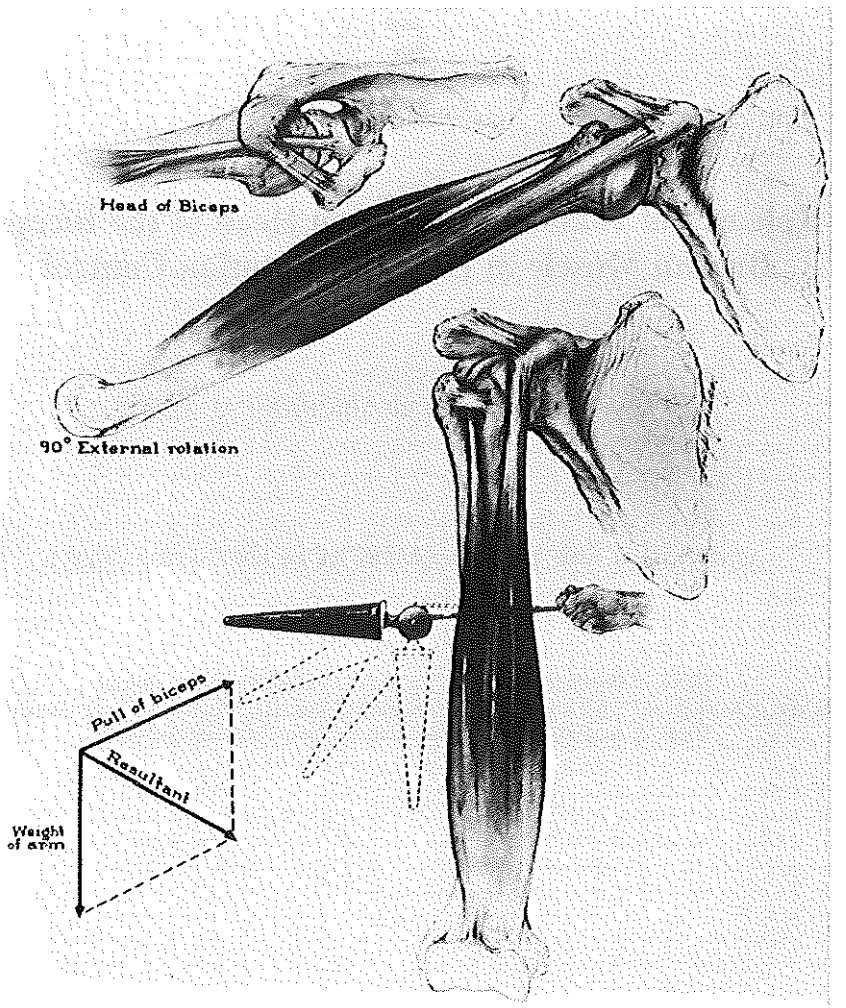
Role of the Tendon of the Long Head of the Biceps Brachii Muscle in Humeral Rotation Control

AN ANATOMICAL AND BIOMECHANICAL STUDY

ARTHUR DE GAST, MD§, ¶, CHRIS J. SNIJDERS, PH.D.$, ROB STOECKART, PH.D.¶

§Dept. of Orthopaedic Surgery, University Hospital Rotterdam, Dijkzigt
$Dept. of Biomedical Physics and Technology, Faculty of Medicine, Erasmus University Rotterdam
¶Dept. of Anatomy, Faculty of Medicine, Erasmus University Rotterdam

In Press
J Shoulder Elbow Surg


Head of Biceps
90° External rotation
Pull of biceps
Resultant
Weight of arm

CHAPTER

5

Role of the Tendon of the Long Head of the Biceps Brachii Muscle in Humeral Rotation Control

Abstract

This anatomical and biomechanical study focuses on the influence of the long head of the biceps brachii muscle on the glenohumeral range of motion during arm elevation. The tendon of the long head of the biceps brachii (biceps tendon) is shown to either facilitate or restrict humeral rotation. Its effect on glenohumeral motion is strongly related to 1) the amount of biceps tendon load, 2) glenohumeral scapular plane elevation and 3) the rotatory position of the humerus. Under 45° of glenohumeral elevation, biceps tendon load facilitates internal and external humeral rotation, actively increasing the rotatory range of motion. Above 45° biceps tendon load restricts internal and external rotation of the humerus, actively increasing joint stability. Furthermore, biceps tendon loads restrict the glenohumeral range of motion mimicking a pattern commonly found in patients with frozen shoulder.

These findings on the function of the biceps tendon have consequences for the clinical interpretation of shoulder pain, shoulder instability, restriction patterns of glenohumeral range of motion and the use and interpretation of tests specifically related to the biceps brachii muscle.

Since the biceps muscle functionally couples the elbow to the shoulder joint, it is emphasized that biceps muscle action on the elbow inevitably influences shoulder function and vice versa.

5.1 Introduction

The role of the tendon of the long head of the biceps brachii (biceps tendon) in the kinematics of the glenohumeral joint (GHJ) has been subject of numerous studies. Experimental data suggest that the long head of the biceps brachii functions as a weak flexor and internal rotator in the GHJ.[1] Both experimentally and clinically it has been shown that the biceps tendon can function as a dynamic anterior stabilizer of the GHJ by

decreasing anterior translation of the humeral head [12, 21, 26] and increasing torsional rigidity.[17, 21, 25, 26] In addition it can function as a depressor of the humeral head.[17, 25, 26]
Effects of biceps tendon tension on internal and external rotation of the humerus through a complete arc of glenohumeral (GH) elevation have not been assessed in detail in the literature. Therefore, this anatomical and biomechanical study was designed to investigate the role of the biceps tendon in controlling humeral rotation (HR). This role is rather complex since the biceps tendon provides both rotational mobility and torsional rigidity and furthermore couples elbow to shoulder function. Better understanding of the biceps tendon function during arm elevation in the scapular plane has implications for the clinical interpretation of shoulder pain, shoulder instability, restriction patterns of GH range of motion (ROM) and for the use and interpretation of tests specifically related to the function of the biceps tendon.
Since elevation in the scapular plane is considered to be the most functional GH movement,[4] this movement is chosen for the testing procedures in this study. This elevation in the scapular plane is indicated here with 'GH elevation'. Rotation about the longitudinal axis of the humerus is HR (either internal or external rotation). The purpose of this study was to define the relationship between HR through a full arc of GH elevation and biceps tendon tension.

5.2 Materials and methods

5.2.1 Dissection and specimen preparation

Dissection was performed on one unembalmed male specimen and five embalmed specimens (three male, two female; 79.2 $\pm$ 5.3 years of age), four left and two right shoulders. The fresh specimen was frozen twenty-six hours after death at -40° C and thawed twelve hours before dissection at room temperature. The other specimens were embalmed by vascular perfusion forty-eight and sixty hours postmortem with a medium containing: 50g phenol 99%, 20g $MgSO_4$, 20g $NaSO_4$, 10g NaCl, 60ml formaldehyde 37%, 60ml glycerin, H_2O ad 1000ml. The specimens were kept in containers filled with phenol (30 g/l) for six weeks. Subsequently, the specimens were stored in phenoxy-ethanol (10ml/l) at a temperature of 14°C for three months. The specimens were stripped of soft tissues, preserving the rotator cuff muscle-tendon units. GHJ capsule and incorporated GH ligaments were spared. The joints were not vented, maintaining negative intra-articular pressure, and hence GHJ stability.[3, 5, 16] To assure maximal mobility, the rotator cuff muscles were incised perpendicular to the muscle fiber direc-

tion, two centimeters medially of the glenoid rim. To preclude impairment of the joint capsule, the part of the rotator cuff muscles located medially to the incision were subperiostally freed from the scapula (Figure 5.1a-b).

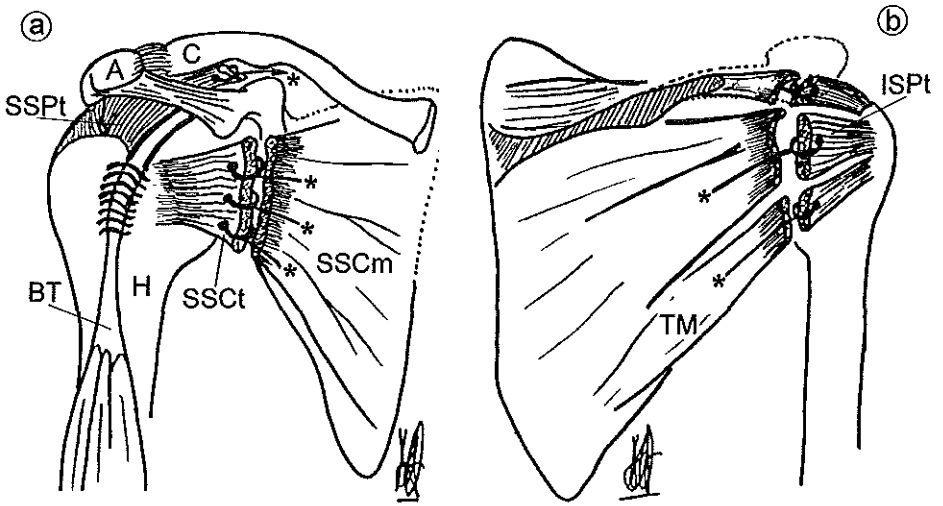


Figure 5.1a Anterior view of the right shoulder showing the reflected subscapularis and supraspinatus muscle-tendon units. A=acromion; BT=biceps tendon; C=clavicula; H=humerus; ISP= infraspinatus tendon; SSCm=subscapularis muscle; SSCt=subscapularis tendon; SSP=supraspinatus tendon.
Figure 5.1b Posterior view of the right shoulder showing the superior and posterior rotator cuff muscle-tendon units and the direction of the suture wires (*) pulling on the tendons. TM=teres minor. For additional legend see figure 5.1a.

After the dissection all specimen displayed a full range of motion and without signs of GHJ instability on manual examination. Finalizing the experiments the shoulder specimens were inspected for abnormalities of the biceps tendon, rotator cuff and joint surfaces. All specimens revealed normal soft tissue structures and congruent joint surfaces without signs of osteoarthritis. The course of the biceps tendon did not show notable anatomical variation. All biceps tendons were broad at the origin, tapering to a smaller diameter at the musculotendinous junction. The biceps tendons were stable in the bicipital groove during the course of the experiments. All biceps tendons were free of macroscopic degenerative changes.

5.2.2 Instrumentation and Kinematic Test

A custom-made three-dimensional positioning device was developed for the kinematic and loading tests (Figure 5.2a). The scapula of each specimen was anchored with clamps fixing the scapular margins to a reference plate. The medial (vertebral) border of the scapula was oriented parallel to the vertical axis of the reference plate, that in turn was perpendicular to the ground. The scapula and humerus were thus placed in the correct anatomical position. In this study, rotation about the longitudinal axis of the humerus is referred to as HR. A specific metal socket contained the humeral shaft in a vertical position. HR was measured with a specially designed goniometer attached to the metal socket. The goniometer had a 360°-scale with a 1°-division of scale. Before the measurements were made, the goniometer was determined to be accurate within 1°. A telescopic device between metal socket and frame, a universal (Hookes) joint and a low-friction ball bearing provided complete freedom of HR and humeral head translation upon the glenoid fossa. A special clamp prevented potential abnormal caudal translation of the humerus in the absence of deltoid muscle force (Figure 5.2b). Experiments were conducted at room temperature. The specimens were kept moist throughout the experiment.

Since it has been suggested [15, 22] that 'true abduction' of the arm should not be in the frontal (coronal) plane but in the plane of the scapula, all movements were defined with respect to the scapular plane. The scapular plane and axes differ from those, normally used in the clinical evaluation of shoulder movements.[9]

Pre-loading of the biceps tendon (2.25 N) and rotator cuff tendons (2.0 N) prevented slack during the experiment and provided a compressive joint force, centering the humeral head with respect to the glenoid cavity.[18] Rotator cuff pre-load was applied by means of nylon sutures (Ethylon 1.O, atraumatic), attached to isotonic spring devices at the border of the reference plate. Loading of the biceps tendon occurred by means of a Ticron-5 suture, woven through the biceps tendon using a modified Bunnell technique. Distally, the Ticron-5 suture was attached to a base plate that slid along the telescopic device. This design allowed for biceps tendon tension in the direction of the humeral shaft. The weight of the base plate was 2.25 N.

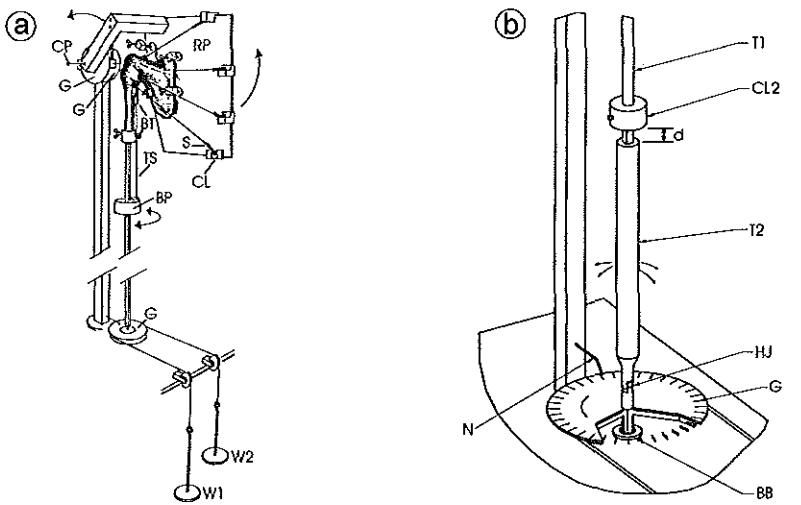


Figure 5.2a Schematic drawing of the custom-made device to measure humeral rotation (HR) through an arc of glenohumeral (GH) elevation. The humerus is free to rotate about its longitudinal axis. Biceps tendon load applied by placing weights on the base plate. Pre-load applied to the rotator cuff by means of isotonic spring devices connected to suture wires (*). BP=base plate; CL=adjustable clamps holding the scapula to the reference plate; CP=one of two movable pins to center the humeral head as the scapula was mounted on the reference plate; G=goniometer; HJ=Hookes joint; RP=reference plate; S=isotonic springs; SC=scapula; TS=ticron suture; W_{1-2}=weights to generate HR.

Figure 5.2b Detail of the telescopic device and Hookes joint that that allowed for translations of the humeral head. An adjustable clamp limited caudal translation of the humerus to 1.0 cm, necessary because of the absence of deltoid muscle force. HJ=Hookes joint; CL_2=special clamp limiting caudal translation; T1/T2=telescopic device. For additional the legend see figure 5.2a.

The neutral position of the GHJ refers to a vertical position of the medial (vertebral) border of the scapula (0° GH elevation) and the rotatory position of the humerus with pre-load on the rotator cuff and biceps tendons (0° HR). Without interfering with neutral position, the goniometer was set to zero. Torsional rotation was defined as the rotation caused by a constant torque of 1.8 Nm applied to the goniometer, causing either internal or external HR. This torque was used since a preliminary study showed this torque caused maximal HR within 10 seconds, without damaging the specimen. Internal rotation was marked (in degrees) with a positive value, external rotation with a negative value. Moving the scapula about an anterior to posterior axis centered on the center of the humeral head simulated GH elevation. Stops were made at -15°, 0°, 10°, 15°, 20°, 30°, 35°, 45°, 60°, 75°, 90°, 105°, 115° and maximal GH elevation. In the interval between 10 and 45° GH elevation, 5°-increments of were used, since preliminary investigations showed here a sudden reversal of HR. At each stop HR was measured.

5.2.3 Loading Tests

The kinematic study was repeated with tension (range 2.25N - 82.25 N) applied to the biceps tendon by placing 10 N weights one by one on the base plate (Figure 5.2a). For each additional weight, a full range of GH elevation was tested. The influence of increasing biceps tendon tension on the rotatory position of the humerus at 0° GH elevation was recorded as well.

5.2.4 Statistical Analysis

In order to quantify the effect of biceps tendon load on HR the Spearman's Rank-correlation coefficient was calculated upon the order of increasing biceps tendon load against the decrease of HR (Table 5.3). For the mean loss of HR caused by maximal biceps tendon load (82.25 N) see Table 5.2.

5.3 Results

5.3.1 Anatomic Observations

Manual testing through full internal and external rotation of the humerus showed that above 10° GH elevation maximal external rotation brings the bicipital groove posterior to the scapular plane in all specimen (Figures 5.4c and 5.6c). Finalizing the experiments, the specimens were taken off the reference plate and the biceps tendon was exposed at its origin. Maximal GH elevation could be induced while holding the humerus in one hand and pulling the biceps tendon with the other. At this position, the humeral tuberosities came in the vicinity of the biceps tendon origin (Figures 5.5b and 5.7).

5.3.2 Kinematic Tests

During GH elevation, with pre-load on the rotator cuff and biceps tendon, the humerus gradually rotated externally towards a position without residual rotational freedom (66 - $\pm$ 6.3° external rotation of the humerus). This position was reached at 111$\pm$ 10.4° GH elevation. At 35° GH elevation, maximal HR (the sum of internal and external rotation) was 155 $\pm$ 11.6° (Figure 5.3a-c).

Table 5.1. Internal Humeral Rotation in 0° GH under increasing Biceps Tendon Load

	Biceps Tendon Load*							
	2.25	12.25	22.25	32.25	42.25	52.25	62.25	82.25
Rotation†	0	5.6	8.4	14.0	18.2	19.0	20.0	23.3
		(4.6)	4.6	(2.3)	(2.2)	(2.2)	(1.8)	(3.7)

* in Newton
† Mean and (Standard Deviation)

5.3.3 Loading Tests

At 0° GH elevation biceps tendon load caused an increase of internal rotation of the humerus, from 0° at 2.25N biceps tendon load (defined as the neutral position) to 23 ± 3.7° at 82.25N biceps tendon load (Table 5.1).

Increased biceps tendon load caused typical patterns of facilitation and restriction of HR. Under 45° GH elevation, the increase of biceps tendon load restricted external rotation of the humerus progressively (Figures 5.4a-c). The largest restriction of external rotation of the humerus occurred at the interval between -15° and 45° GH elevation (Figures 5.4 a-c and Table 5.2). At the interval between -15° and 15° GH elevation, internal rotation of the humerus increased under biceps tendon load. Maximal decrease in total rotation (the sum of internal and external rotation) as a result of biceps tendon load, occurred with 82.25N biceps loading at -15° of GH elevation. In this position, the sum of internal and external rotation of the humerus was 35.5 ± 7.2° at 82.25N biceps load against 108 ± 18.1° at 2.25N. At 10° GH elevation the relative loss of total rotation due to biceps tendon load was maximal: 50 ± 9.3° at 82.25N biceps tendon load against 145 ± 13.3° at 2.25N biceps tendon load. As GH elevation increased to about 15°, biceps tendon load suddenly caused maximal external rotation. With increasing biceps tendon load this sudden external rotation occurred at a higher degree of GH elevation (Figures 5.3ab).

Above 45° GH elevation restriction of both external and internal rotation occurred as a result of biceps tendon load (Figures 5 a-c). Increase of biceps tendon load caused a relatively small, but constant decrease (range 10° to 23°) of internal rotation of the humerus between 45° and 90° GH elevation. The restriction of HR caused by 82.25N biceps tendon load at various levels of GH elevation is listed in Table 5.2. For statistical significance of the results, see Table 5.3.

The kinematic patterns of the unembalmed specimen did not differ from the embalmed specimens, but the magnitude of HR varied (Figure 5.3b).

Table 5.2. Loss of Internal and External Rotation due to 82.25 N Biceps Tendon Load*

	Humeral Rotation					
	Internal Rotation§			External Rotation§		
GH Elev †	Mean	Stdev	Median	Mean	Stdev	Median
-15	-0.8	7.0	0	76.8	20.4	68
0	0.8	7.3	4	90.0	17.8	86
10	3.2	4.1	5	90.2	15.1	94
15	0.2	6.2	1	86.4	17.3	90
20	3.8	5.1	5	82.4	18.1	87
25	5.6	4.2	6	67.2	37.7	81
30	7.4	4.5	7	53.2	46.7	75
35	9.8	4.2	9	50.2	43.3	64
45	17.0	6.7	19	22.0	33.2	10
60	30.8	9.8	31	10.2	4.1	12
75	27.0	8.4	24	10.8	4.2	12
90	16.0	7.8	14	10.4	7.8	8
100	2.0¶			1.0¶		
105	7.3	6.7	4	12.7	5.5	10
115	5.0‡			7.3‡		

* Data from the five embalmed specimens
† Glenohumeral Elevation in the plane of the scapula, in degrees
§ Mean, Standard Deviation and Median loss of humeral rotation, in degrees
¶ One specimen reached this level of GH elevation
‡ Two specimens reached this level of GH elevation

Table 5.3. Spearman's Rank-correlation Coefficients*

	1		2		3		4		5	
	Int	Ext	Int	Ext	Int	Ext	Int	Ext	Int	Ext
-15		0.99	0.88	0.99		0.93		0.88		0.96
0		0.92		0.98		0.88	0.97		0.97	0.94
10		0.93		0.83		0.83	0.99		0.99	0.99
15							0.99	0.83	0.99	0.98
20			0.84				0.99	0.97	0.99	0.99
25		-0.86					0.99	0.95	0.93	0.99
30	0.98						1.00	0.98	0.99	0.98
35	0.90		0.85		0.87		0.99	0.99	0.99	0.99
45	0.93		0.85	0.83	0.98	0.90	1.00	0.98	0.99	0.99
60	0.93	0.88	0.95	0.95	0.86	0.96	1.00	0.99	1.00	1.00
75	0.99		0.99	0.95	0.88	1.00	1.00	1.00	1.00	0.99
90	0.97		0.83	0.86		0.96	1.00	0.99	0.99	0.99
100										
105			-0.83	0.99			0.98	1.00	0.96	0.99
115				0.90			1.00	1.00		

* Calculated upon the order of increasing biceps tendon load against the decrease of Humeral Rotation
Values of $|R_s| > 0.82$ are statistically significant; $p<0.01$

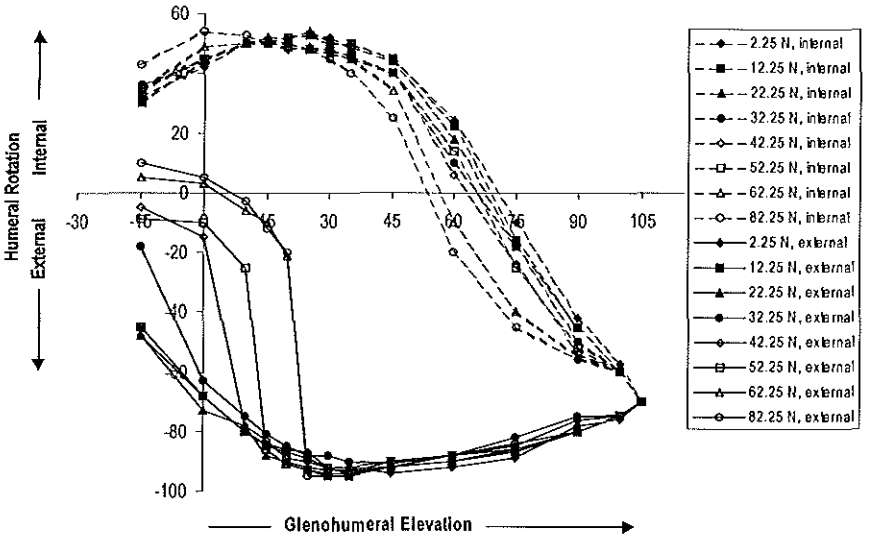


Figure 5.3a Graphical representation of the effect of increasing biceps tendon load on the range of HR at different levels of GH elevation (in the scapular plane). Ranges of motion in degrees. X-axis represents full-range of GH elevation in the scapular plane. Y-axis represents the amount of HR; internal rotation marked with a positive value, external rotation with a negative value. Full lines: external rotational torque applied to the humerus; broken lines: internal rotational torque applied to the humerus. Typical example of an embalmed specimen.

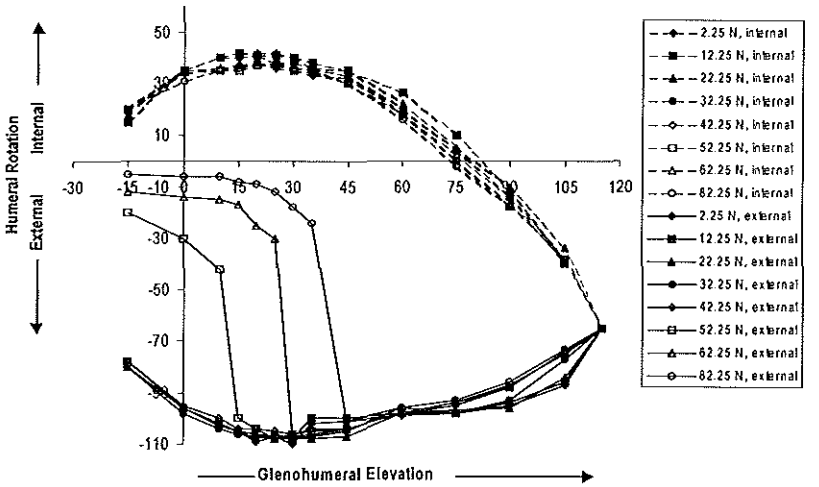


Figure 5.3b The unembalmed specimen showing a similar pattern of changes in HR cause by increasing biceps tendon load. For further explanation, see figure 5.3a.

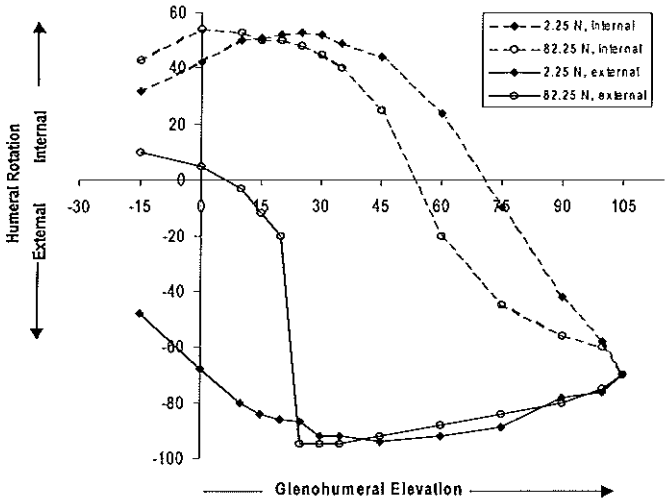


Figure 5.3c Schematic representation showing the effect of maximal biceps tendon load (82.25 N) compared to the pre-loaded (2.25N) situation. Dark area represents the amount of restriction of HR and the shaded area the amount of facilitation of HR. For further explanation, see figure 5.3a.

5.4 Discussion

5.4.1 Biceps Tendon Control of Humeral Rotation

The most important result to emerge from this study is that biceps tendon load can either restrict or facilitate both internal and external rotation of the humerus. The effect of biceps tendon load on HR depends on the degree of GH elevation and the rotatory position of the humerus. Descriptions of biceps tendon function at the level of the GHJ usually refer to the *anatomic position* [8] in which the biceps tendon lies anteriorly of the humeral head and angles medially towards its origin (Figure 5.1a). In this position the long head of the biceps brachii is regarded to generate flexion, abduction, and internal rotation in the GHJ.[2] The results of the present study support that in the anatomic position biceps tendon load facilitates internal rotation and consequently restricts external rotation of the humerus. However, during GH elevation in the interval between 10° and 45° the rotatory influence of the biceps tendon reverses, and the biceps tendon becomes an external rotator of the humerus. The explanation for this reverse of function is twofold. First, during GH elevation the humerus rotates externally under the influence of articular cartilage contours and capsuloligamentous structures. These movements bring the bicipital groove to a more proximal and posterior position in relation to the biceps tendon origin. Second, as the bicipital groove crosses the scapular plane posteriorly, the intertubercular portion of the biceps tendon shifts against the *lateral* wall

of the bicipital groove and, consequently, sudden external rotation occurs. (Figure 5.4a-c). Therefore, if the intertubercular portion of the biceps tendon lies anteriorly to the scapular plane it acts as an internal rotator, posteriorly, it acts as an external rotator of the humerus.

The influence of biceps tendon load on rotation of the humerus dramatically changes at above approximately 45° GH elevation. At this level of GH elevation the bicipital groove levels with the biceps tendon origin in the horizontal plane and the humeral tuberosities approach the biceps tendon origin. In this situation biceps tendon tension straightens the biceps tendon and brings biceps tendon origin, the floor of the bicipital groove and the humeral shaft in line with each other. Since HR in either direction causes angulation of the biceps tendon, biceps tendon load thus opposes this rotation (Figure 5.5a-c).

The findings of the present study are in line with clinical and experimental observations made in two shoulder disorders in which the biceps tendon has a compensatory role: loss of muscle force in case of rotator cuff tendon rupture, and decrease of GHJ stability in case capsuloligamentous failure. First, under 45° GH elevation biceps tendon tension *facilitates* HR. Electromyography (EMG) studies support the present findings: they show that the long head of the biceps brachii muscle is active during external rotation becoming more active with a more externally rotated position.[10] Furthermore, EMG shows larger activity of the long head of the biceps brachii muscle in shoulders with rotator cuff tendon rupture compared to the unimpaired shoulder.[24] These patients are also characterized by a significantly larger biceps tendon diameter,[20] indicating use-induced hypertrophy. Second, earlier research showed that the biceps tendon stabilizes translation of the humeral head in the horizontal [17, 26] and vertical plane.[17, 25, 26] However, it should be emphasized that rotatory stability is important as well. The present study shows that, at the interval of 45° to maximal GH elevation, the long head of the biceps brachii muscle no longer facilitates but *restricts* HR. This restriction of HR increases the rotatory stability GHJ by increasing torsional rigidity (Fig 5.3a-c). From their observations, Rodosky et al.[21] could not explain that the GHJ becomes torsionally stiffer with increasing activity of the long head of the biceps muscle. They hypothesized correctly, as shown by the present study, that the abducted and externally rotated position of the GHJ allows the biceps tendon to act as an internal rotator of the humerus. Particularly in GHJ positions of abduction and external rotation, the inferior GH ligament was shown to be protected against increasing external rotation forces with increasing biceps tendon force.[21] This may explain the increased EMG activity of the biceps brachii muscle in case of anterior shoulder instability in pitchers.[6]

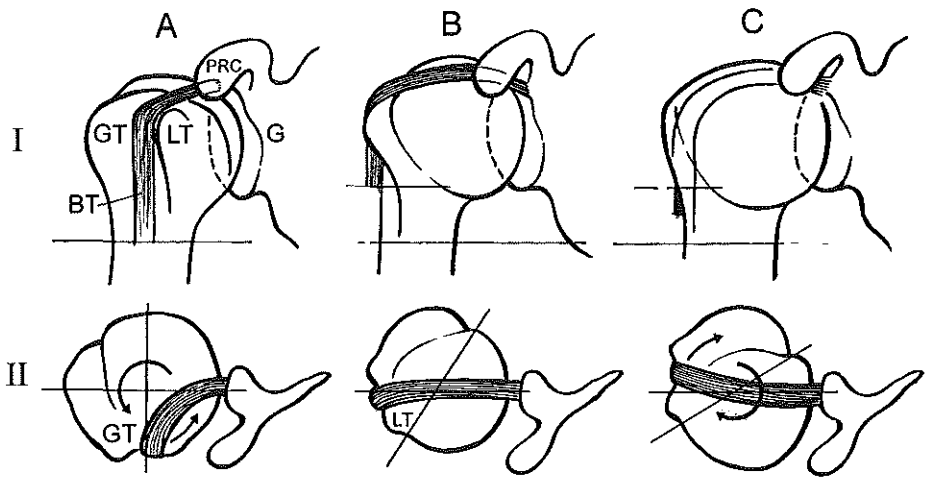


Figure 5.4 Schematic drawing of the relationship between the biceps tendon and the humeral head in various positions of HR. Approximately 10° GH elevation. I. Anterior view; II. Superior view. A. Humerus in internally rotated position. The biceps tendon acts against the lesser tuberosity causing internal rotation. B. Humerus in the mid-range of external rotation. Superior portion of the biceps tendon lies in the scapular plane. No rotational forces occur. C. Humerus in externally rotated position. The biceps tendon acts against the greater tuberosity, causing external rotation. For legends see Abbreviations.

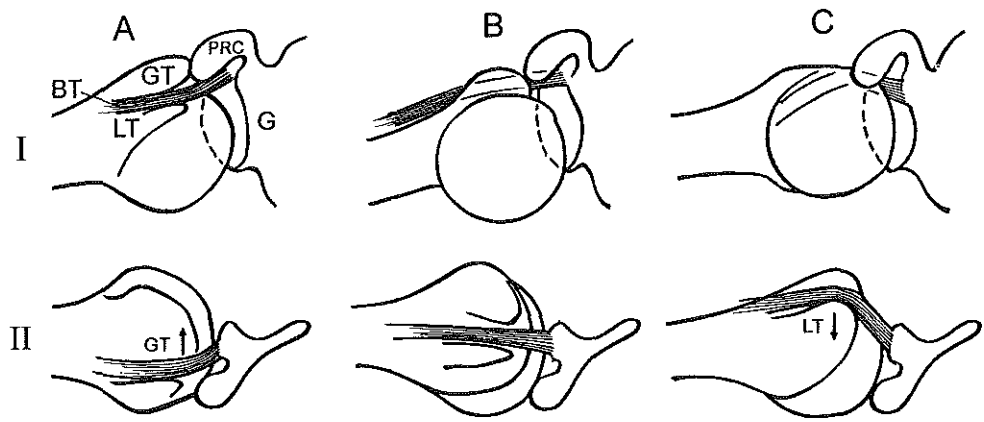


Figure 5.5 Schematic drawing of the relationship between the biceps tendon and the humeral head in various positions of HR. Near-maximal GH elevation. I. Anterior view; II. Superior view. A. Humerus in internally rotated position. The biceps tendon acts against the greater tuberosity causing external rotation bringing the humerus to the position as in figure 5.4 B. B. Biceps tendon, humeral shaft and biceps tendon origin in line with each other. No rotational forces occur. C. Humerus in externally rotated position. The biceps tendon acts against the lesser tuberosity, causing internal rotation, bringing the humerus back to the position as in figure 5.4 B. For legends see Abbreviations.

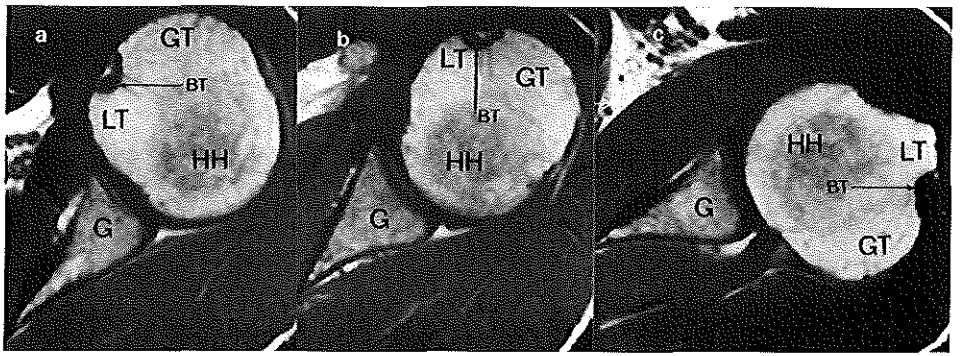


Figure 5.6a-c Magnetic resonance images (T1W) of a healthy male volunteer without complaints of the imaged shoulder. The upper part of the photographs represents the anterior side of the shoulder, the lower part the posterior side. Horizontal sections of the left shoulder in a. maximal internal rotation, b. anatomic position, and c. maximal external rotation. In maximal external rotation the bicipital groove lies posterior to the plane of the scapula. Compare with figure 5.4. BT=bicpes tendon; G=Glenoid; GT=greater tuberosity; HH=Humeral head; LT=lesser tuberosity.

5.4.2 Form and function of the bicipital groove

Although the form of the bicipital groove shows marked variation on horizontal section, in 92% a typical V-shape can be seen, formed by the greater and lesser tuberosities (see also Chapter 2, Figure 2.5).[11] This V-shape supports the findings in this study that the biceps tendon has a bi-directional rotatory function at the GHJ. The medial and lateral wall of the bicipital groove can withstand medially and laterally directed contact pressures caused by biceps tendon tension. In contrast to the trochlear mechanism of the bicipital groove, only one bony wall develops at Lister's tubercle. Here the tendon of the extensor pollicis longus muscle angles 45° radially towards the base of the thumb. Most likely, since tendon forces at Lister's tubercle are directed mainly in a radial direction, a bony wall develops at one side of the tendon only.

5.4.3 Maximal Glenohumeral Elevation

The results of the present study are in line with earlier observations that the final common position of the GHJ for either abduction (GH elevation in the frontal plane) or forward flexion (GH elevation in the sagittal plane) can be reached without simultaneous

HR.[19,22] The results of the present show that during GH elevation the humeral tuberosities move towards the origin of the biceps tendon. In fact the humeral tuberosities ride upon the biceps tendon, comparable with a monorail train. This anatomical mechanism allows for only one joint position at maximal GH elevation. It is shown that in order to reach maximal GH elevation from the anatomic position [8] the humerus rotates externally over an average of 66 $\pm$ 6.3°.

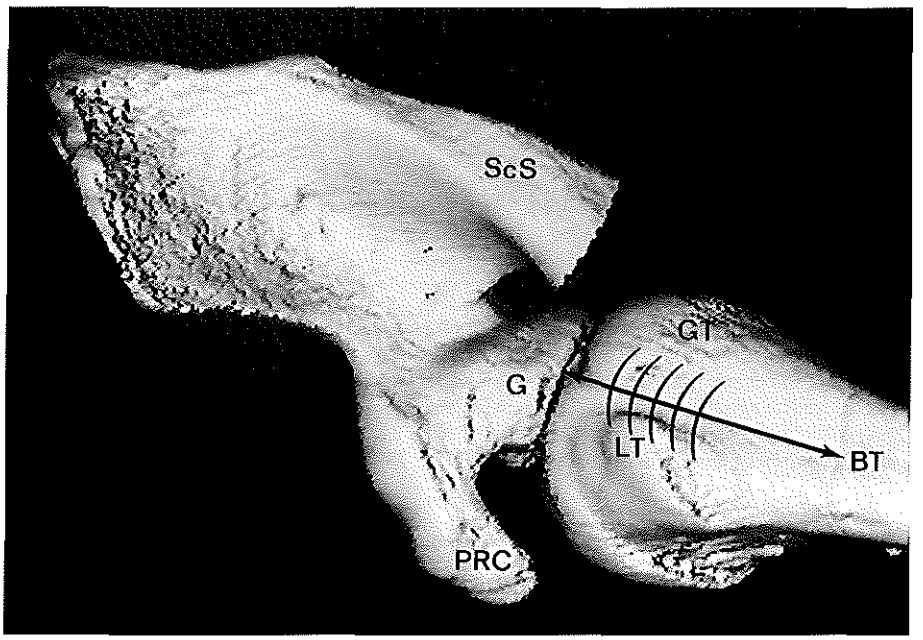


Figure 5.7 Three-dimensional CT-reconstruction of a superior view on the left GHJ when the arm of this healthy volunteer was held in the maximally elevated position. The humerus was free to rotate about is longitudinal axis. Greater and lesser tuberosities are in the vicinity of the biceps tendon origin. Acromion has been (digitally) resected. Compare with figure 5.5. BT= course of the biceps tendon (indicated with long arrow); G=Glenoid; GT=greater tuberosity; LT=lesser tuberosity; ScS=scapular spine.

5.4.4 Long head of the biceps coupling shoulder to the elbow

In sports and daily activities the shoulder and elbow are functionally coupled. However, anatomy texts [7,23] usually describe the function of the long head of the biceps acting on the shoulder and its function on the elbow separately in different chapters. The long head of the biceps brachii is a *poly*-articular muscle and in fact, its contraction effects at least five joints (the proximal and distal radio-ulnar, the humero-radial, the humero-ulnar and the GH joints). Furthermore, any contraction of the long head of the biceps brachii muscle influences both shoulder and elbow joints simultaneously. It is emphasized that

generally contraction of the biceps muscle only occurs if an effect in both joints is needed, or when unwanted side effects of the biceps brachii muscle action on other joints can be counteracted. For example, the biceps brachii muscle has little or no activity in the act of throwing, except when active flexion or eccentric extension in the elbow is made.[13, 14]

5.4.5 Biceps-tendon-tests

The most reliable method to reveal biceps tendon disorders by clinical examination is probably eliciting pain on specific palpation of the bicipital groove. However, based on the results of this study more exact biceps tendon tests can be designed. These tests should include particular GHJ positions, HR and active elbow flexion.

5.5 Conclusions

1. The biceps tendon functions as a monorail, guiding the humeral tuberosities to the biceps tendon origin during GH elevation. This explains that from the anatomic position, maximal GH elevation can only be reached with approximately 65° external rotation of the humerus. In a position of maximal GH elevation, no HR is possible.
2. Biceps tendon tension has a complex effect on shoulder function. Description of biceps tendon function in the anatomic position only does not suffice, since we showed that biceps tendon function strongly depends on the position at the GHJ. Under 45° of GH elevation biceps tendon load facilitates internal and external rotation of the humerus, increasing actively the range of HR. Above 45° of GH elevation biceps tendon load restricts internal and external rotation of the humerus, increasing actively joint stability.
3. In line with the above, strengthening of the biceps brachii will on the one hand facilitate HR and on the other provide additional stability of the GHJ. Based on the results of this study more specific biceps tendon tests can be designed to diagnose disorders of the biceps tendon.

Acknowledgements

The authors thank Cor Goedegebuur for technical assistance, Marieke van Zwienen, Edgar ten Holder and Jan-Bart van Lent for assistance with the dissections and instrumentation tests, Cees Entius and Jan Velkers for their assistance with the preparation of the specimen, Dana-Anne Bakker and Ir. J. Hop for their help with the statistical analysis, and Andry Vleeming for his comments on earlier versions of the manuscript.

REFERENCES

1. **Basmajan JV, Latif MA.** Integrated actions and functions of the chief flexors of the elbow. *J Bone Joint Surg [Am]* 1957;39 A:1106-18.
2. **Braus H.** Bewegungsapparat. Anatomie des Menschen. 2nd ed. Berlin: Julius Springer Verlag, 1929:307. vol 1.
3. **Brown AO, Hoffmeyer P, An KN.** The influence of atmospheric pressure on shoulder stability. *Orthop Trans* 1990;14:259-61.
4. **Freedman L, Munro RR.** Abduction of the arm in the scapular plane: scapular and glenohumeral movements. A roentgenographic study. *J Bone Joint Surg [Am]* 1966;48(8):1503-10.
5. **Gibb TD, Sidles JA, Harryman DTd, McQuade KJ, Matsen FAd.** The effect of capsular venting on glenohumeral laxity. *Clin Orthop* 1991(268):120-7.
6. **Glousman R, Jobe F, Tibone J, Moynes D, Antonelli D, Perry J.** Dynamic electromyographic analysis of the throwing shoulder with glenohumeral instability. *J Bone Joint Surg [Am]* 1988;70(2):220-6.
7. **Gray H.** Arthrology. In: Williams P, Warwick R, eds. Gray's Anatomy. 36th ed. Edinburgh: Churchill Livingstone, 1980:458.
8. **Gray H.** Introduction. In: Williams P, Warwick R, eds. Gray's Anatomy. 36th ed. Edinburgh: Churchill Livingstone, 1980:xv-xvii.
9. **Greene WB, Heckman JD,** eds. The clinical measurement of joint motion. 1st ed. American Academy Of Orthopaedic Surgeons, 1994.
10. **Habermeyer P, Kaiser E, Knappe M, Kreusser T, Wiedemann E.** Zur funktionellen Anatomie und Biomechanik der langen Bizepssehne. *Unfallchirurg* 1987;90(7):319-29.
11. **Hitchcock HH, Bechtol CO.** Observations on the role of the long head of the biceps brachii in its causation. *J Bone Joint Surg [Am]* 1948;30:263-73.
12. **Itoi E, Kuechle DK, Newman SR, Morrey BF, An KN.** Stabilising function of the biceps in stable and unstable shoulders [published erratum appeared in J Bone Joint Surg Br 1994 Jan;76(1):170]. *J Bone Joint Surg [Br]* 1993;75(4):546-50.
13. **Jobe FW, Moynes DR, Tibone JE, Perry J.** An EMG analysis of the shoulder in pitching. A second report. *Am J Sports Med* 1984;12(3):218-20.
14. **Jobe FW, Tibone JE, Perry J, Moynes D.** An EMG analysis of the shoulder in throwing and pitching. A preliminary report. *Am J Sports Med* 1983;11(1):3-5.
15. **Johnston T.** The movements of the shoulder. A plea for the use of the 'plane of the scapula' as the plane of reference for movements occuring at the humero-scapular joint. *British J Surg* 1937;25:252-60.
16. **Kumar VP, Balasubramaniam P.** The role of atmospheric pressure in stabilising the shoulder. An experimental study. *J Bone Joint Surg [Br]* 1985;67(5):719-21.
17. **Kumar VP, Satku K, Balasubramaniam P.** The role of the long head of biceps brachii in the stabilization of the head of the humerus. *Clin Orthop* 1989(244):172-5.
18. **Lippitt S, Matsen F.** Mechanisms of glenohumeral joint stability. *Clin Orthop* 1993(291):20-8.
19. **Martin CP.** A note on the movements of the shoulder joint. *British J Surg* 1932;20:61-6.
20. **Perry J.** Muscle control of the shoulder. In: Rowe CR, ed. The shoulder. New York: Churchill Livingstone, 1988:26.
21. **Rodosky MW, Harner CD, Fu FH.** The role of the long head of the biceps muscle and superior glenoid labrum in anterior stability of the shoulder. *Am J Sports Med* 1994;22(1):121-30.

22. **Saha AK.** The classic. Mechanism of shoulder movements and a plea for the recognition of "zero position" of glenohumeral joint. *Clin Orthop* 1983(173):3-10.
23. **Sobotta.** Atlas of human anatomy. Munich: Urban & Schwarzenberg, 1982:311. vol I.
24. **Ting A, Jobe FW, Barto P.** An EMG analysis of the lateral biceps in shoulders with rotator cuff tears. Third open meeting of the society of American Shoulder and Elbow Surgeons. California, 1987.
25. **Warner JJ, Deng XH, Warren RF, Torzilli PA.** Static capsuloligamentous restraints to superior-inferior translation of the glenohumeral joint. *Am J Sports Med* 1992;20(6):675-85.
26. **Warner JJ, McMahon PJ.** The role of the long head of the biceps brachii in superior stability of the glenohumeral joint. *J Bone Joint Surg [Am]* 1995;77(3):366-72.

CHAPTER

6

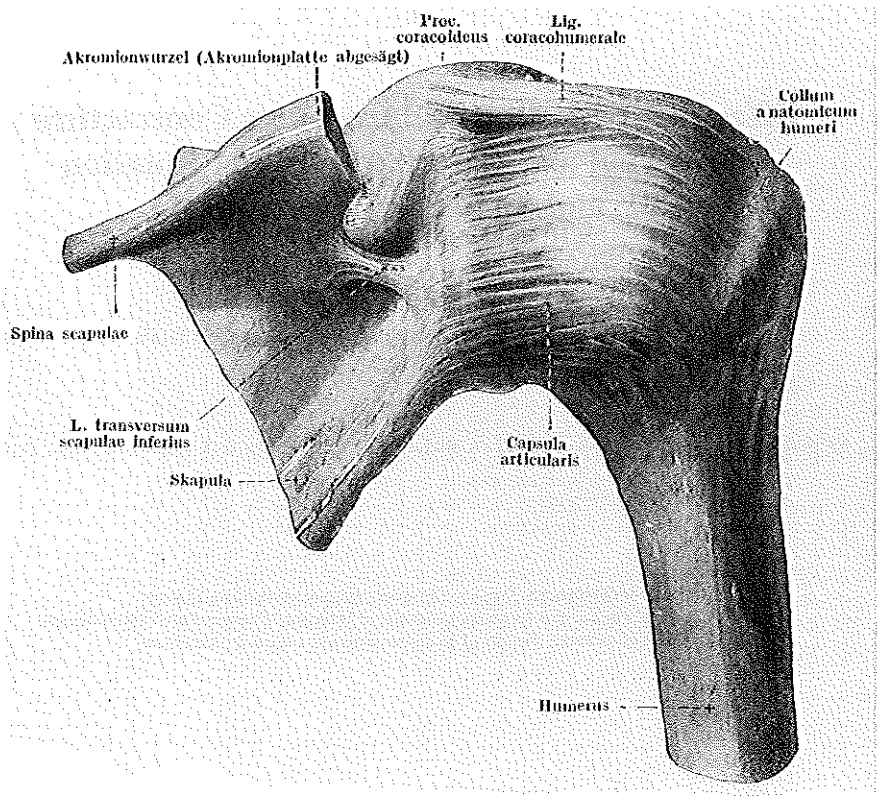
Effects of Glenohumeral Capsular Contracture on Internal and External Humeral Rotation and Shoulder Elevation

AN ANATOMICAL AND BIOMECHANICAL STUDY

ARTHUR DE GAST, MD§, ¶, $, KARIM RAISSADAT, MD¶, CHRIS J. SNIJDERS, PH.D.,¶, $, ROB STOECKART, PH.D.¶

§Dept of Orthopaedic Surgery, University Hospital Rotterdam, Dijkzigt
$Dept of Biomedical Physics and Technology, Faculty of Medicine, Erasmus University Rotterdam
¶Dept of Anatomy, Faculty of Medicine, Erasmus University Rotterdam

Submitted for Publication


Proc. coracoideus
Lig. coracohumerale
Akromionwurzel (Akromionplatte abgesägt)
Collum anatomicum humeri
Spina scapulae
L. transversum scapulae inferius
Skapula
Capsula articularis
Humerus

CHAPTER

6

Effects of Glenohumeral Capsular Contracture on Internal and External Humeral Rotation and Shoulder Elevation

ABSTRACT

Limitation of internal or external rotation of the humerus (humeral rotation) is a common and avoidable complication of shoulder surgery. We assessed the effect of shortening of different regions of the glenohumeral joint capsule on humeral rotation and on glenohumeral elevation in the plane of the scapula. The specimens were obtained from five embalmed human cadavers. They were tested both intact and after methodical shortening of the glenohumeral joint capsule. Shortening occurred at five regions (three at the anterior side and two at the posterior side) with segments of beaded chain and catches. Due to shortening of the beaded chains the range of motion decreased in consistent patterns. A significant decrease of external humeral rotation ($p < 0.0001$) was found after shortening of the three anterior regions of the glenohumeral joint capsule, and of internal rotation ($p < 0.002$) after shortening of the two posterior regions. Shortening of the anterior inferior and posterior inferior regions of the glenohumeral joint capsule caused a 10° to 45° decrease of glenohumeral elevation. A typical, nonlinear relation existed between shortening of the beaded chains and decrease of humeral rotation. Furthermore, the effect of shortening on the range of motion strongly depended on the glenohumeral joint position. Above 45° glenohumeral elevation, shortening of both the anterior inferior and posterior inferior regions of the glenohumeral joint capsule shared in limiting internal and external rotation of the humerus.

The observations are relevant for the shoulder surgeon since they help to explain the relationship between a contracture of specific regions of the glenohumeral joint capsule and limited glenohumeral motion as seen in frozen shoulder and after operations that limit (external) rotation of the humerus.

6.1 Introduction

Shoulder afflictions with limited range of motion (ROM) are thought to originate from pathologic changes in glenohumeral (GH) soft tissue structures.[29, 41, 43] Rotator cuff lesions with secondary bursal reaction [27] and contracture of the glenohumeral joint capsule (GHJC) are often held responsible.[40, 42] In patients with frozen shoulder, both a primary [6] and secondary [30] contracture of the GHJC is correlated with restricted ROM. According to recent reports, in these patients, particularly, a contracture of the coracohumeral ligament, the subscapular bursa and adjacent soft tissue structures play an important role.[9, 31] A study on the potential effect of a contracture of the coracohumeral ligament shows indirectly that this ligament contributes to a limitation of GH ROM.[28] After cutting the ligament, external rotation increased an average of 32° at 0° GH elevation, and of 15° at 90° GH elevation. Harryman et al.[16] assessed effects of shortening of the anterior superior region of the GHJC on displacement and angular motion of the humeral head. This region, usually referred to as rotator interval capsule, was cut and an operative overlap of 1 cm was made. Shortening the rotator interval capsule significantly reduced flexion by 8°, extension by 18°, adduction by 8° and external rotation by 18-38°. Although these experiments yield valuable data, the relationship between GHJC contracture and limited ROM remains not well understood. Recently, important progress has been made in the understanding of relationships between lesions of the GHJC and glenohumeral joint (GHJ) stability.[7] Experimental studies in this field mainly deal with the effect of selective cutting of GH ligaments on the ability to displace the humeral head out of the glenoid fossa.[18, 33, 34, 44] Despite this progress, little is known about the kinematic alteration produced by surgical reconstruction of the GHJC. This is surprising, since these surgical treatments are explicitly designed to restore normal GH mechanics of which little is known.[1] Therefore, to obtain a better understanding of the effect of soft tissue contracture on limitation of GH ROM, we assessed effects of systematic shortening of different regions of the GHJC on internal and external rotation of the humerus. Branch et al.[4] studied effects of capsular lengthening on the range of internal and external rotation of the humerus through a cone of motion. In the present study, a modification was used of their segments of beaded chains to simulate different capsule lengths. Since parts of the GHJC act as position-dependent restraints,[10, 33, 46] internal and external humeral rotation was measured in various GHJ positions. Considering GH elevation in the scapular plane to be more functional than elevation in the frontal plane,[11, 20, 37, 39] scapular plane elevation was chosen for the testing procedures. This elevation is

indicated with 'GH elevation'. Rotation about the longitudinal axis of the humerus is indicated with humeral rotation (either internal or external rotation).

The purpose of this study was to quantify the limitation of humeral rotation (HR) through an arc of GH elevation due to shortening of the anterior and posterior regions of the GHJC.

6.2 Materials and Methods

6.2.1 Dissection and specimen preparation

Dissection was performed on five embalmed specimens (three male, two female; 72 ± 4.2 years of age), three left and two right shoulders, one shoulder of each specimen. Forty-eight to sixty hours postmortem the specimens were embalmed by vascular perfusion with a medium containing: 50g phenol 99%, 20g $MgSO_4$, 20g $NaSO_4$, 10g NaCl, 60ml formaldehyde 37%, 60ml glycerin, H_2O ad 1000ml. The specimens were kept in containers filled with phenol (30 g/l) for six weeks. Subsequently, the specimens were stored in phenoxy-ethanol (10ml/l) at a temperature of 14°C for three months. All muscles were stripped of the shoulder specimens, preserving the rotator cuff muscle-tendon units, the tendon of the long head of the biceps brachii (biceps tendon), and the GHJC and integrated GH ligaments. The joints were not vented, maintaining negative intra-articular pressure, and hence GHJ stability.[5, 13, 21] To assure maximal mobility, the rotator cuff muscles were incised perpendicular to the muscle fiber direction, two centimeters medially of the glenoid rim. To preclude impairment of the joint capsule, the part of the rotator cuff muscles located medially to the incision were freed subperiostally from the scapula. After dissection all specimens displayed a full range of motion without signs of GHJ instability on manual examination.

Table 6.1 Relation between placement of the chain catches at the glenoid side and the capsular regions

	Specimen side	
Capsular region	Right *	Left *
Anterior		
Superior	2-o'clock	10-o'clock
Middle	3-o'clock	9-o'clock
Inferior	5-o'clock	7-o'clock
Posterior		
Superior	10-o'clock	2-o'clock
Inferior	7-o'clock	5-o'clock

*Placement of the catches is related to the hours of a clock with its center projected onto the center of the glenoid fossa.

Segments of beaded chain were sutured into the (humeral-side) insertions of the GH capsular ligaments (Figure 6.1). The beads were exactly 2.5 mm apart when the chain was taut. Five specially designed chain catches were screwed into the bony glenoid. In the GHJC the GH ligaments are recognizable, but they have multiple fibrous connections together making up the GHJC. Furthermore, these ligaments show great variation in size, shape, thickness, and attachment site.[10, 32, 46] Therefore, locations of the origins of the capsular ligaments on the glenoid were standardized. Three catches were placed at the anterior side of the GHJ, each of them corresponding to a standardized origin of one of the anterior GH ligaments. Two catches were placed at the posterior side of the GHJ. For the relationship between the regions of the GHJC and the placement of the catches, see Table 6.1. The above design allowed for controlled shortening of these regions of the GHJC by 2.5-mm increments. After suturing the segments of beaded chain into the insertions of the GH ligaments, attachment to their corresponding catches was done as follows. The specimens were moved through the full passive ROM. To assess the length of each segment of beaded chain, necessary to allow for this full ROM, the GHJ was held in a position that maximally stretched one of the five regions of the GHJC. Then the corresponding segment of beaded chain was attached to its catch. So, the length of the chain equaled the length of that maximally stretched region of the GHJC. The first bead medial to its catch was marked.

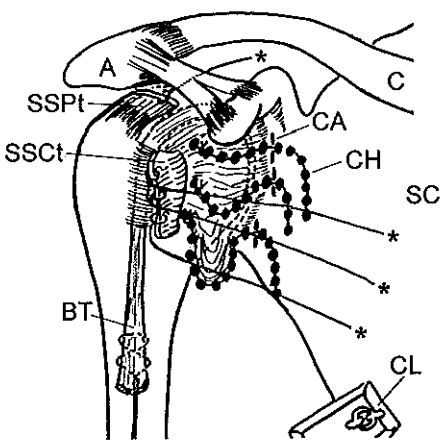


Figure 6.1 Anterior view of the right shoulder. Schematic representation of the segments of beaded chain sutured to the anterior capsule. A similar procedure was performed at the posterior side of the specimen (see materials and methods section for location of the chains at the posterior side). A=acromion; BT=biceps tendon; C=clavicula; CA=catch; CH=segment of beaded chain; CL=adjustable clamps holding the scapula to the reference plate; SW=sutures to apply load to the tendon; SC=scapula; SSCt=subscapularis tendon (reflected); SSP=supraspinatus tendon.

Finalizing the experiments the shoulder specimens were inspected for abnormalities of the rotator cuff, GHJC, biceps tendon and joint surfaces. All specimens revealed normal soft tissue structures and congruent joint surfaces without signs of osteoarthritis.

6.2.2 Instrumentation

For the custom-made three-dimensional positioning device see Figure 6.2a-c. The scapula of each specimen was anchored with clamps fixing the scapular margins to a reference plate. The medial (vertebral) border of the scapula was oriented parallel to the vertical axis of the reference plate, that in turn was perpendicular to the ground. Thus, the scapula and humerus were placed in the correct anatomic position. The reference plate was free to rotate about an anterior to posterior axis centered on the center of the humeral head. Since it has been agreed upon [11, 20, 37, 39] that 'true abduction' of the arm should not be in the frontal (coronal) plane but in the plane of the scapula, all movements were defined with respect to the scapular plane. The scapular plane and GHJ axes differ from those normally used in the clinical evaluation of shoulder movements.[14] After mounting the scapula on the reference plate a counter-weight balanced the weight of the scapula and the reference plate (Figure 6.2c). A metal socket contained the humeral shaft in a vertical position. HR was measured with a specially designed goniometer attached to the metal socket. The goniometer had a 360°-scale with a 1°-division. Before the measurements, the goniometer was determined to be accurate within 1°. A telescopic device between metal socket and frame, a universal (Hookes) joint and a low-friction ball bearing provided complete freedom of humeral head translation and HR. An adjustable clamp limited caudal translation of the humerus to 1.0 cm, necessary because of the absence of deltoid muscle force (Figure 6.2b). Experiments were conducted at room temperature. The specimens were kept moist throughout the experiment.

Loading of the biceps tendon (2.25 N) and rotator cuff tendons (2.0 N) prevented slack during the experiment and provided a compressive joint force, centering the humeral head with respect to the glenoid cavity.(23) Rotator cuff tendon load was applied by means of nylon sutures (Ethylon 1.O, a-traumatic), attached to isotonic springs at the border of the reference plate. Loading of the biceps tendon occurred by means of a similar nylon suture, woven through the tendon using a modified Bunnell technique. Distally, this suture was attached to a base plate that slid along the telescopic device. This design allowed for biceps tendon tension in the direction of the humeral shaft. The weight of the base plate was 2.25 N.

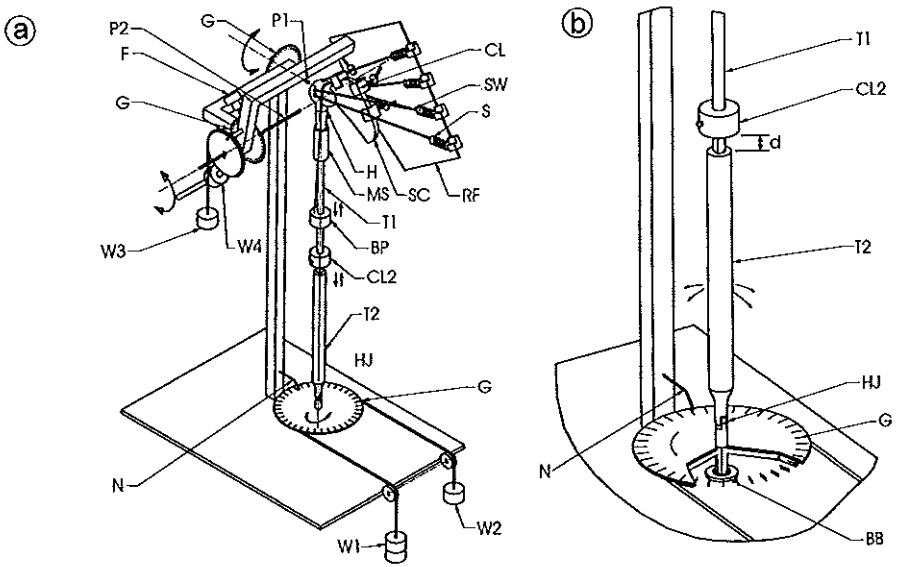


Figure 6.2a Schematic drawing of the device and of the shoulder specimen. BP=base plate; Cl=adjustable clamps holding the scapula to the reference plate; F=fastening bold for locking the device at positions of GH elevation; G=goniometer; H=humerus; SW=suture wires; HJ=Hookes joint; MS=metal socket; P_{1-2}= movable pins to center the humeral head as the scapula was mounted on the reference plate; RF=reference plate; S=isotonic springs; SC=scapula; T=telecopic device; W_{1-3}=weights to generate GH elevation and axial humeral rotation; W_4=movable weight to counter balance the weight of the scapula and the reference plate.
Figure 6.2b Detail of the telescopic device and Hookes joint that allowed for translations of the humeral head. An adjustable clamp limited caudal translation of the humerus to 1.0 cm, necessary because of the absence of deltoid muscle force. This construction prevented potential damage to the capsuloligamentous complex of the specimens. BB=ball bearing; CL_2=special clamp limiting caudal translation; HJ=Hookes-joint; N=Needle; T1/T2=telescopic device. For additional legend see figure 6.2a.

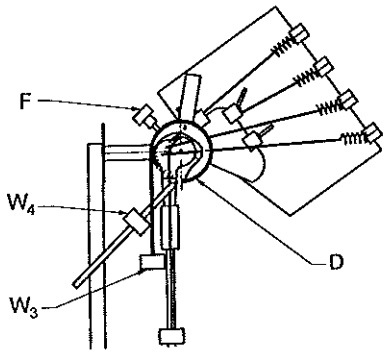


Figure 6.2c Frontal 'exploded' view of the device. D=disk for the application of an elevatory momentum to the shoulder specimen; F=fastening bold for locking the device at positions of GH elevation; W_3= weight generating an elevatory momentum to the shoulder specimen; W_4= counter balance weight.

In this study we used the following definitions: 1) neutral GH elevation (0° GH elevation) refers to a vertical position of the medial border of the scapula; 2) neutral HR (0° rotation of the humerus) refers to initial rotatory position of the humerus with load on the rotator cuff and biceps tendons; 3) GH scapular plane adduction (referred to as GH adduction) was marked in degrees with a negative value, GH elevation with a positive value; 4) internal rotation was marked in degrees with a positive value, external rotation with a negative value. GH elevation was simulated by constant torque of 1.2 Nm about an anterior to posterior axis applied to the reference plate. A constant torque of 1.2 Nm applied to the goniometer produced HR. A preliminary study showed these torque's of 1.2 Nm caused (within 10 seconds) maximal GH elevation and maximal HR, without damaging the specimens or compromising the lateral attachments of the beaded chains. The scapula was free to move through a complete arc of GH elevation.

6.2.3 Tests

Both internal and external rotation of the humerus were measured after positioning the GHJ in -15° elevation (15° GH adduction). A test series consisted of one set of measurements through a complete arc of GH elevation. By fastening a bold, stops were made at -15°, 0°, 15°, 30°, 45°, 60°, 75°, 90° and maximal GH elevation. At each stop both internal and external rotation were recorded. Since a preliminary study showed that repeated measurements differed maximally 3° with two to six series, each series was performed twice. After assessing maximal GH elevation and HR, one segment of a beaded chain was shortened 5 mm (2 beads) and again two test series were performed. If, by shortening of the segments GH elevation decreased, no attempts were made to force GH elevation further. In this situation maximal GH elevation could be less than 90°. For each beaded chain separate test series were made. Maximal shortening of the beaded chains was defined as the minimal chain-length that just did not force the GHJ out of the neutral position.

6.2.4 Data Analysis

For each shoulder, ten sets of data were collected, one for each of the five regions of the GHJC in internal rotation and in external rotation (Figure 6.3a-e). To establish the relationship between HR and GH elevation, the data collected from the five specimens were subjected to repeated measurements Analysis of Variance tests (rmANOVA, computed with SPSS and SAS). The same test was used to establish the effect of shortening a region of the GHJC on HR and on GH elevation.

6.3 Results

In all specimens, the relationship between GH elevation and HR showed a similar pattern. The range of HR reached its maximum between 30° and 45° GH elevation. Above 45° GH elevation, the range of HR strongly diminished (Figure 6.3a-e). The patterns of decrease of HR due to shortening of the chains were consistent in all specimens, but the magnitude varied. For a typical example, see Figures 6.3a-e. Detailed values of the loss of axial humeral rotation due to shortening of the five GHJC regions are listed in Table 6.2. Both external and internal rotation were negatively affected by shortening of the anterior inferior and posterior inferior regions of the GHJC respectively (Figure 6.3c and 6.3e). Shortening of the anterior superior, anterior middle, and anterior inferior regions of the GHJC significantly decreased external rotation ($p<0.0001$), while shortening of the both posterior regions significantly decreased internal rotation ($p<0.002$). Typically, the shortening of the regions of the GHJC and the decrease of HR had a nonlinear relation (Figure 6.3a-e). GH elevation was negatively affected ($p<0.002$) by shortening of the anterior inferior and posterior inferior regions of the GHJC.

Shortening of the *anterior superior region* of the GHJC resulted in a decrease of external rotation ($p<0.0001$), with no significant effect on GH elevation (Figure 6.4ab). Above 60° GH elevation, the shortening had no significant effect on external rotation. Maximal shortening of the beaded chain was 20 mm in three specimens (15 mm in two specimens).

Shortening of the *anterior middle region* of the GHJC significantly decreased external rotation ($p<0.0001$) (Figure 6.4cd). In all but one specimen (maximal shortening 15 mm), maximal shortening of the beaded chain was 25 mm.

Shortening of the *anterior inferior region* of the GHJC significantly decreased external rotation ($p<0.0001$) (Figures 6.4ef). GH elevation decreased also ($p<0.002$): with maximal shortening of the beaded chain, the decrease of GH elevation averaged 34° (range 20-45°). Shortening of the anterior inferior region rendered the largest decrease of GH elevation. At higher angles of GH elevation, shortening of this region of the GHJC decreased internal rotation in addition. In all specimens, maximal shortening of the beaded chain was 30 mm.

Shortening of the *posterior superior region* of the GHJC significantly decreased internal rotation ($p<0.002$) (Figure 6.4gh). Shortening of this region of the GHJC did not affect GH elevation. In two specimens, maximal shortening of the beaded chain was 20 mm, in two specimens 15 mm, and in one specimen 10 mm.

Shortening of the *posterior inferior region* of the GHJC decreased both internal rotation ($p < 0.0001$) and GH elevation (Figure 6.4i-j). The decrease of GH elevation averaged 24° (range 10-45°). In two specimens, maximal shortening of the beaded chain was 25 mm, in two specimens 20 mm and in one specimen 15 mm.

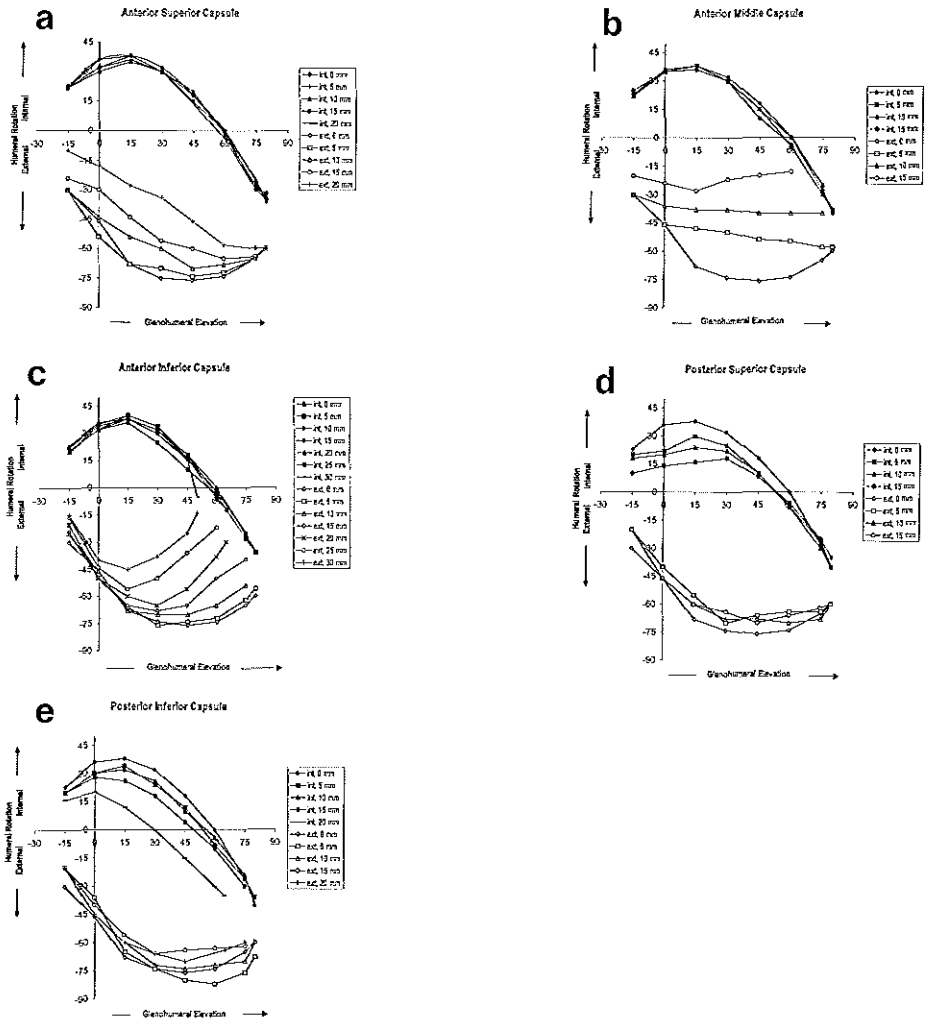


Figure 6.3a-e Graphical example showing the effects of shortening of the regions of the GHJC, in one specimen. Figures 6.3a-c represent the effects of shortening at the anterior side, 6.3d and 6.3e at the posterior side. The amount of shortening is shown in mm. (int=internal rotation, ext=external rotation) The set of heavy lines on the upper side of the chart represents the influence of progressive shortening of the GHJC on internal rotation, the set of lines on the lower side of the chart represents the influence on external rotation (in degrees). 0 mm shortening represents the range of HR without modification of the length of the GHJC.

Table 6.2 Mean Loss of Humeral Rotation due to Shortening of the Anterior GHJ Capsule

	Anterior Superior Capsule											
	Internal Rotation *						External Rotation *					
GHE†	Shortening §						Shortening §					
	5	10	15	20	25	30	5	10	15	20	25	30
-15	**1.8**	**1.8**	**1.8**	**1.3**			**-4.4**	**-4.0**	**-2.4**	**13**		
	(2.2)	(2.2)	(2.2)	(1.5)			(11.7)	(12.1)	(14.5)	(12.1)		
0	**1.6**	**3.2**	**2.4**	**2.7**			**1.6**	**6.4**	**12.8**	**19.3**		
	(1.7)	(2.3)	(2.2)	(2.3)			(6.5)	(5.0)	(8.1)	(16.8)		
15	**1.2**	**2.6**	**3.6**	**2.7**			**6.6**	**15.4**	**24.2**	**34.3**		
	(1.9)	(3.4)	(3.0)	(1.2)			(10.9)	(11.9)	(9.4)	(8.1)		
30	**2.4**	**2.0**	**3.2**	**3.7**			**11.6**	**15.8**	**26.8**	**43.0**		
	(2.5)	(1.2)	(3.4)	(1.5)			(11.8)	(11.9)	(11.3)	(6.2)		
45	**3.2**	**2.4**	**3.4**	**4.7**			**7.0**	**9.2**	**20.0**	**34.3**		
	(4.9)	(5.2)	(3.8)	(4.0)			(10.0)	(9.4)	(13.5)	(9.3)		
60	**5.2**	**3.6**	**3.6**	**5.0**			**5.2**	**6.0**	**12.8**	**21.3**		
	(7.2)	(5.2)	(6.2)	(4.6)			(4.1)	(4.7)	(9.7)	(6.1)		
75	**4.0**	**3.4**	**3.2**	**2.7**			**2.4**	**4.6**	**5.6**	**5.0**		
	(12.5)	(9.8)	(9.0)	(2.3)			(3.5)	(6.1)	(6.5)	(5.0)		
90	**3.0**	**9.5**	**7.5**	**0.0‡**			**8.5**	**6.0**	**1.0**	**2.0‡**		
	(4.2)	(13.4)	(13.4)				(2.1)	(8.5)	(1.4)			
	Anterior Middle Capsule											
-15	**0.8**	**0.4**	**0.4**	**5.4**	**5.4**		**0.4**	**0.4**	**3.4**	**2.8**	**4.8**	
	(1.3)	(2.2)	(2.2)	(10.0)	(10.0)		(1.5)	(1.5)	(4.4)	(3.9)	(7.3)	
0	**1.6**	**1.0**	**0.4**	**8.6**	**7.4**		**2.0**	**4.6**	**9.8**	**10.0**	**16.3**	
	(1.5)	(2.2)	(2.5)	(15.4)	(16.1)		(3.9)	(3.8)	(7.9)	(7.1)	(13.6)	
15	**1.6**	**1.8**	**2.4**	**10.0**	**10.0**		**7.8**	**11.2**	**18.2**	**22.0**	**31.3**	
	(3.8)	(3.6)	(3.3)	(15.9)	(16.0)		(7.0)	(10.8)	(13.6)	(4.8)	(13.0)	
30	**1.6**	**1.6**	**2.8**	**9.6**	**9.2**		**9.6**	**16.2**	**30.6**	**35.3**	**47.8**	
	(2.7)	(2.7)	(2.6)	(13.2)	(13.2)		(8.1)	(11.6)	(12.8)	(3.9)	(2.6)	
45	**3.4**	**3.0**	**5.8**	**8.0**	**10.4**		**8.8**	**18.8**	**34.6**	**41.8**	**55.5**	
	(3.4)	(3.5)	(5.9)	(7.6)	(9.7)		(7.5)	(10.1)	(15.1)	(11.5)	(12.1)	
60	**5.8**	**4.8**	**7.6**	**9.3**	**5.3**		**8.6**	**18.0**	**34.8**	**42.3**	**58.0**	
	(8.0)	(5.8)	(7.1)	(8.5)	(8.1)		(6.8)	(9.1)	(5.5)	(13.0)	(5.7)	
75	**1.8**	**-0.4**	**5.2**	**5.3**	**3.0¶**		**4.6**	**12.2**	**28.5**	**41.3**	**56.0¶**	
	(2.0)	(3.2)	(8.4)	(6.5)			(5.3)	(8.3)	(4.3)	(19.4)		
90	**5.3**	**5.0**	**7.0**	**5.0¶**	**6.0‡**		**8.5**	**14.0**	**32.0**	**53.5¶**	**58.0‡**	
	(4.2)	(4.6)	(6.1)				(4.9)	(5.7)	(17.0)			
	Anterior Inferior Capsule											
-15	**1.8**	**1.4**	**1.4**	**1.8**	**1.8**	**1.4**	**1.1**	**3.0**	**4.0**	**4.8**	**6.0**	**5.8**
	(3.5)	(1.9)	(1.9)	(2.7)	(2.7)	(1.9)	(9.9)	(3.7)	(4.5)	(5.3)	(6.8)	(6.8)
0	**1.6**	**1.4**	**3.0**	**3.4**	**3.8**	**4.2**	**0.4**	**2.8**	**1.0**	**2.8**	**5.2**	**8.0**
	(0.5)	(1.3)	(1.0)	(0.9)	(2.0)	(1.5)	(3.2)	(3.0)	(5.4)	(6.4)	(5.0)	(5.0)
15	**1.0**	**1.2**	**1.0**	**1.0**	**4.2**	**4.8**	**0.2**	**2.2**	**2.2**	**4.4**	**8.6**	**15.6**
	(3.0)	(2.7)	(2.2)	(3.0)	(4.5)	(3.9)	(1.8)	(2.9)	(2.6)	(3.4)	(5.5)	(8.8)
30	**0.0**	**1.0**	**1.6**	**0.2**	**3.2**	**2.8**	**0.4**	**2.6**	**4.0**	**7.0**	**18.2**	**28.8**
	(2.1)	(1.0)	(1.1)	(2.3)	(2.6)	(4.0)	(2.3)	(2.6)	(5.8)	(6.8)	(8.8)	(10.8)
45	**1.4**	**1.0**	**1.2**	**3.0**	**5.6**	**5.2**	**2.4**	**3.8**	**8.2**	**18.2**	**32.0**	**45.0**
	(1.7)	(1.0)	(1.3)	(2.4)	(5.2)	(6.9)	(3.8)	(2.8)	(5.4)	(9.7)	(10.9)	(7.1)
60	**5.2**	**2.2**	**4.0**	**6.6**	**5.0**	**7.5**	**3.0**	**4.6**	**20.0**	**33.2**	**32.8**	**52.5**
	(7.5)	(2.9)	(6.0)	(4.9)	(4.8)	(3.5)	(2.6)	(5.7)	(14.1)	(11.5)	(20.5)	(9.2)
75	**8.2**	**5.0**	**5.2**	**8.8**	**13.0‡**		**2.6**	**7.2**	**22.1**	**32.5**	**50.0‡**	
	(6.0)	(6.2)	(6.1)	(4.8)			(4.4)	(7.3)	(5.3)	(12.0)		
90	**4.0**	**3.5**	**7.0‡**				**4.5**	**1.0**	**25.0‡**			
	(1.4)	(2.1)					(3.7)	(12.7)				

* Mean (and Standard Deviation) Loss of Humeral Rotation in degrees,
† Glenohumeral Elevation in degrees
‡ One specimen reached this level of GH Elevation
¶ Two specimens reached this level of GH Elevation
§ Shortening of the beaded chains in millimeters

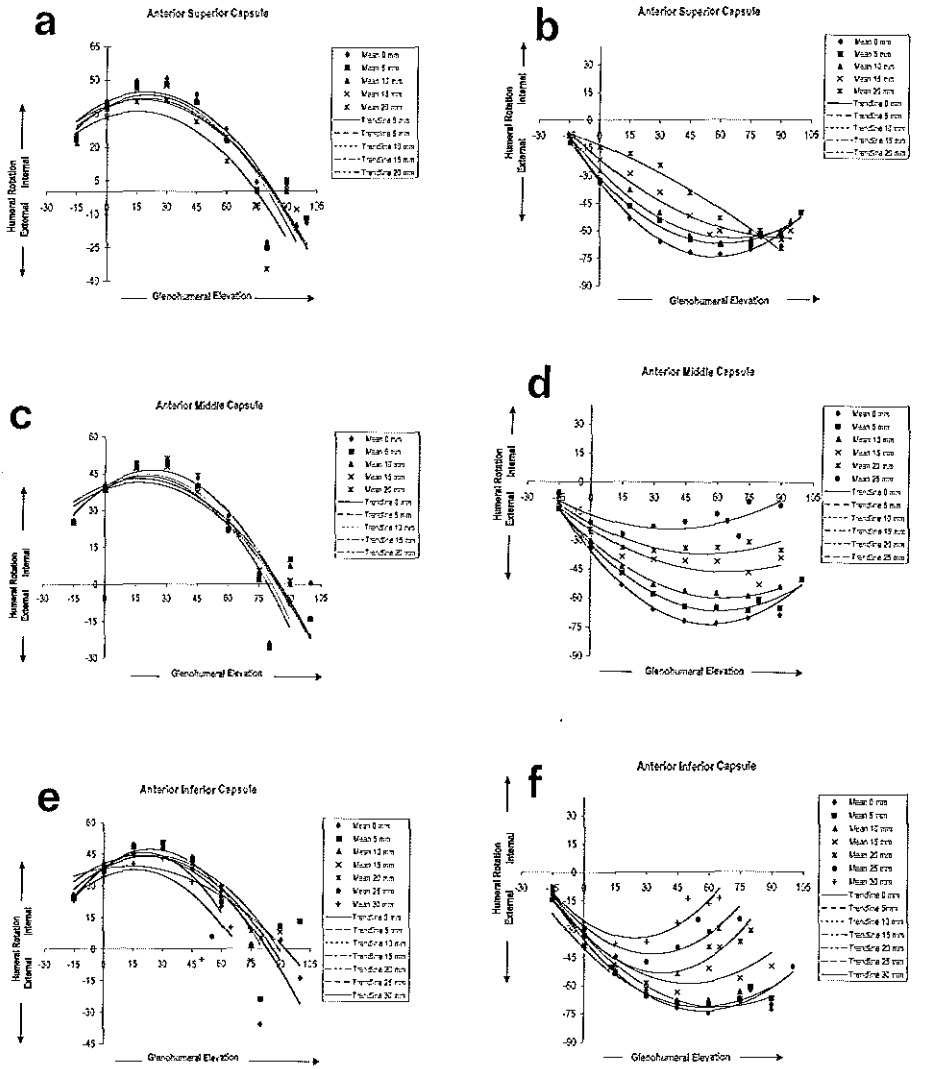


Figure 6.4a-e Graphical representation of the mean loss (and Trendlines) of axial humeral rotation due to shortening of the anterior regions of the GHJC. Figures 6.4a,c and e represent the effect of shortening on the range of internal rotation, figures 6.4b,d and f the effect on external rotation. Ranges of motion in degrees.

Table 6.3 Mean Loss of Humeral Rotation due to Shortening of the Posterior GHJ Capsule

	Posterior Superior Capsule											
	Internal Rotation *						External Rotation *					
GHE†	Shortening §						Shortening §					
	5	10	15	20	25	30	5	10	15	20	25	30
-15	**5.8**	**9.0**	**3.3**	**0.0‡**			**3.4**	**3.8**	**4.3**	**-1.0‡**		
	(11.4)	(17.5)	(6.5)				(4.4)	(4.1)	(4.6)			
0	**12.4**	**16.4**	**18.5**	**6.0‡**			**5.0**	**4.6**	**5.3**	**0.0‡**		
	(11.3)	(18.9)	(10.1)				(4.0)	(4.3)	(6.2)			
15	**12.6**	**17.6**	**24.8**	**22.0‡**			**7.4**	**2.6**	**6.0**	**10.0‡**		
	(12.2)	(19.7)	(12.6)				(4.4)	(3.6)	(4.3)			
30	**9.4**	**17.2**	**22.8**	**42.0‡**			**3.0**	**2.8**	**3.0**	**5.0‡**		
	(8.3)	(15.3)	(13.3)				(3.3)	(5.4)	(5.7)			
45	**6.4**	**12.0**	**18.0**	**47.0‡**			**1.6**	**0.8**	**0.3**	**5.0‡**		
	(4.0)	(8.7)	(12.8)				(6.1)	(6.1)	(6.6)			
60	**4.4**	**8.4**	**12.5**	**39.0‡**			**1.8**	**0.2**	**2.0**	**3.0‡**		
	(3.4)	(6.5)	(13.4)				(5.8)	(4.1)	(5.9)			
75	**4.4**	**4.2**	**7.3**	**34.0‡**			**0.8**	**-1.8**	**0.8**	**6.0‡**		
	(4.6)	(4.1)	(10.0)				(7.0)	(5.4)	(7.6)			
90	**1.0**	**12.5¶**	**12.5¶**	**29.0‡**			**4.7**	**7.5¶**	**6.0¶**			
	(2.2)						(6.4)					
	Posterior Inferior Capsule											
-15	**1.8**	**1.8**	**2.2**	**3.0**	**1.6**		**4.4**	**4.4**	**4.8**	**4.6**	**2.6**	
	(2.7)	(2.7)	(3.5)	(4.1)	(3.6)		(5.3)	(5.3)	(5.9)	(5.1)	(4.3)	
0	**6.0**	**3.6**	**7.2**	**10.6**	**7.2**		**7.4**	**3.6**	**5.2**	**5.2**	**1.2**	
	(3.5)	(3.9)	(5.4)	(7.7)	(10.0)		(6.8)	(5.1)	(5.0)	(5.1)	(1.8)	
15	**5.2**	**6.4**	**15.2**	**20.8**	**13.6**		**3.4**	**10.8**	**4.8**	**3.2**	**-0.4**	
	(5.4)	(5.7)	(8.5)	(14.5)	(18.8)		(4.4)	(16.7)	(5.9)	(3.7)	(0.9)	
30	**6.8**	**10.2**	**20.4**	**27.2**	**18.0**		**2.0**	**6.6**	**3.8**	**0.6**	**1.2**	
	(4.3)	(7.5)	(10.8)	(17.8)	(24.7)		(2.1)	(5.5)	(6.8)	(4.3)	(3.9)	
45	**5.0**	**11.8**	**23.8**	**31.6**	**20.6**		**0.0**	**3.2**	**5.8**	**2.6**	**2.5**	
	(3.2)	(7.8)	(11.4)	(20.8)	(28.3)		(2.9)	(3.8)	(4.4)	(2.8)	(6.4)	
60	**4.8**	**9.4**	**17.6**	**35.0**	40‡		**-0.6**	**2.8**	**5.8**	**4.8**	**2.0‡**	
	(6.1)	(9.0)	(9.1)	(14.3)			(4.3)	(2.3)	(4.8)	(5.0)		
75	**3.6**	**4.4**	**7.0**				**0.2**	**3.8**	**8.3**			
	(7.5)	(7.3)	(4.4)				(7.0)	(7.5)	(4.7)			
90	**5.0¶**	**18.0‡**	**0.0‡**				**6.0¶**	**14.0‡**	**7.0‡**			
							(8.5)					

* Mean (and Standard Deviation) Loss of Humeral Rotation in degrees
† Glenohumeral Elevation in degrees
‡ One specimen reached this level of GH Elevation
¶ Two specimens reached this level of GH Elevation
§ Shortening of the beaded chains in millimeters

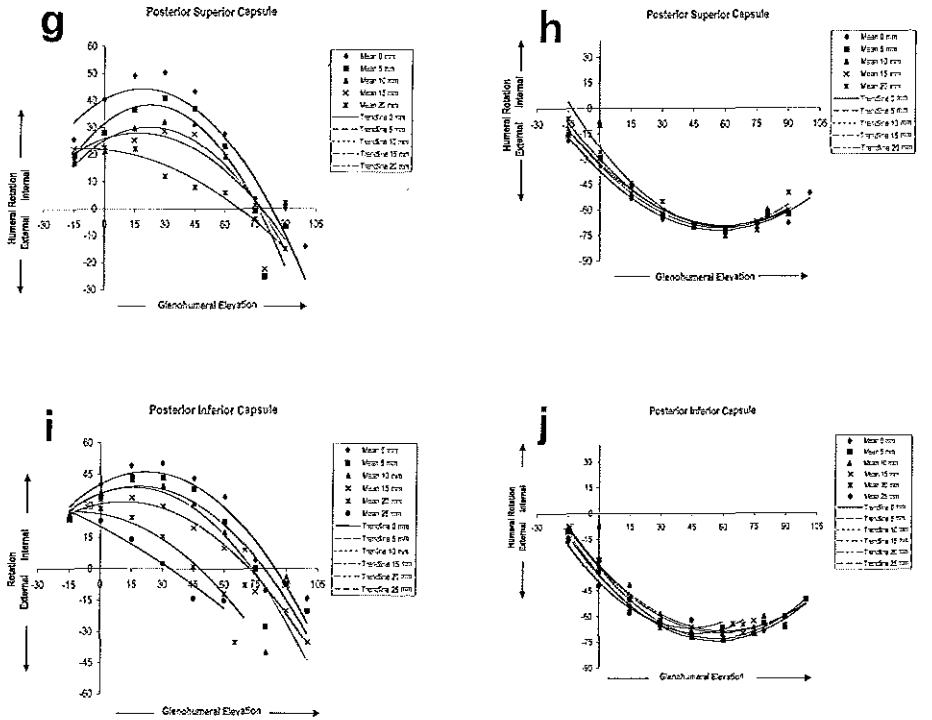


Figure 6.4g-j Graphical representation of the mean loss (and Trendlines) of axial humeral rotation due to shortening of the posterior regions of the GHJC. Figures 6.4g and i represent the effect of shortening on the range of internal rotation, figures 6.4h and j the effect on external rotation. Ranges of motion in degrees.

6.4 Discussion

Studies based on selective cutting of the GHJC and incorporated GH ligaments have played an important role in the understanding of the kinematics of the GHJ. However, selective cutting is not possible without interfering with the normal synergy of these structures. Furthermore, selective cutting of (GH) ligaments in order to investigate the potential effects of a contracture, as done by Neer,[28] has the disadvantage of creating hypermobility instead of a contracture. To avoid such hypermobility, in the present study the coracohumeral and GH ligaments were not cut. This approach has also several limitations. The scapula was not able to move as in normal motion of the arm. The magnitude of the movements differs from that *in vivo*, due to release of the rotator cuff muscles and resection of the skin, subcutaneous tissues and shoulder muscles.[35] Furthermore, the segments of beaded chain are stiff compared to the visco-elastic capsuloligamentous structures *in vivo*. So, shortening of these structures *in vivo* may result in less limitation of ROM than predicted by the present study. The main justification of an *in vitro* study is that it yields data that cannot be obtained *in vivo*. Of course, it is not possible to stepwise shorten certain regions of the GHJC to study kinematics of the GHJ *in vivo*.

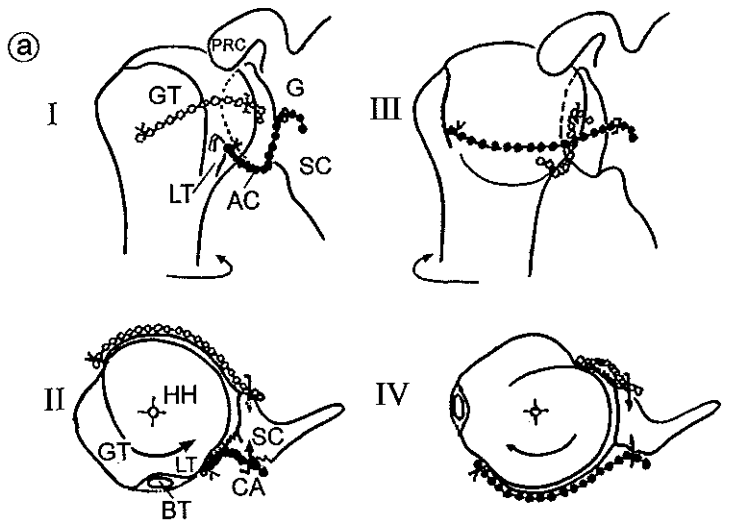


Figure 6.5a Anterior (I and III) and superior view (II and IV) of the right shoulder at 0° GH elevation showing the functional anatomy of the anterior and posterior regions of the GHJC during internal and external rotation. I and II. In internal rotation the anterior chain (AC; dark beads) relaxes and the posterior chain (PC; open beads) tensions. II and IV. In external rotation the anterior chain (AC; dark beads) tensions and the posterior chain (PC; open beads) relaxes. AC=anterior chain; BT=tendon of the long head of the biceps brachii; CA=chain catch; G=glenoid; GT=greater tuberosity; H=humerus; HH=humeral head; LT=lesser tuberosity; PC=posterior chain; PRC=coracoid process; SC=scapula.

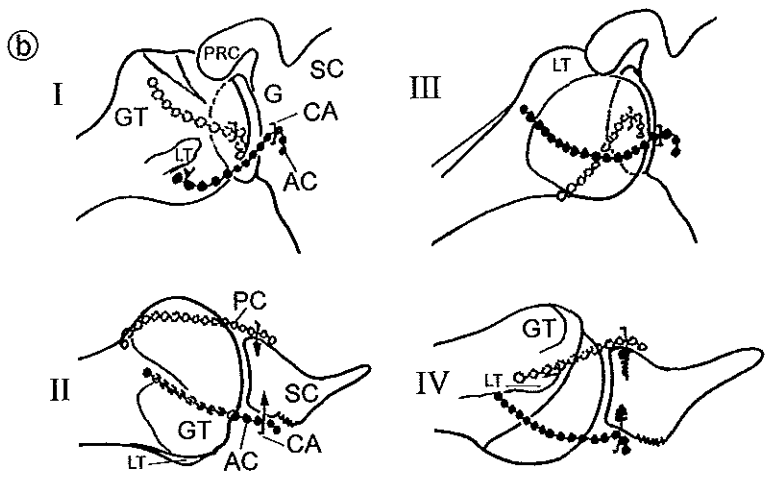


Figure 6.5b Anterior (I and III) and superior view (II and IV) of the right shoulder at 75° GH elevation showing the functional anatomy of the anterior and posterior regions of the GHJC during internal and external rotation. I and II. In internal rotation both the anterior chain (AC; dark beads) and the posterior chain (PC; open beads) tension. II and IV. In external rotation both the anterior chain (AC; dark beads) and the posterior chain (PC; open beads) tension. For additional legend see figure 6.5a.

In the present study, the initial length of each segment of beaded chain corresponding to the five regions of the GHJC was determined by moving the GHJ through the full ROM. The length of a particular segment of beaded chain necessary to allow for a full ROM appeared to be greater than the anatomic length, measured in the anatomic position from chain origin to chain insertion. Nevertheless, Branch et al.[4] used the anatomic length, to study the relationship between HR and the lengths of the anterior and posterior regions GHJC. In their approach, the anterior and posterior regions of the GHJC shared in resisting internal and external rotation ('load sharing') in the range of 50° to 80° GH elevation. In contrast, Warner et al.[46] found that at 90° GH elevation, the inferior GH ligament complex 'cradles the humeral head like a hammock,' with internal and external rotation causing alternate tightening of the anterior and posterior portions of the ligament complex, respectively.

The results of the present study confirm that load sharing of the anterior inferior and posterior inferior regions of the GHJC occurs at higher angles of GH elevation. Our explanation is as follows. Since at 0° GH elevation the humeral axis stands vertically, internal and external rotation about this axis loads the posterior and anterior regions of the GHJC, respectively (Figure 6.5.a-d). At 75° GH elevation the humeral axis gets close to the horizontal plane; 75° is chosen because at higher angles of GH elevation

rotation strongly diminishes (Figure 6.3a-e, see also chapter 4). In this position of GH elevation, rotation about the humeral axis loads both the anterior and posterior regions of the GHJC (Figure 6.5.e-h). The present data show that at 60° GH elevation and above, shortening of the anterior inferior region of the GHJC decreases both internal and external rotation (Figure 6.4ef).

6.4.1 Effects per region of the GHJC

The data of the present study concerning the effect of shortening of the *anterior superior region* of the GHJC, matched the (semi-quantitative) anatomic observations made by Ferrari [10] and others.[8, 12, 16, 22, 28] Shortening of this region mainly restricted external rotation between 0° and 60° of GH elevation. Above 80° GH elevation there was no effect of shortening on external rotation (Figure 6.4b). Several authors have commented on the role of a contracture of the coracohumeral ligament and adjacent anterior superior region of the GHJC [45, 47] in frozen shoulder and recommended their release at the time of open shoulder manipulation.[28, 30, 36] In the present study, the anterior superior region of the GHJC (including the coracohumeral ligament) did not cause a restriction of GH elevation. Therefore, it can be questioned whether a contracture of the anterior superior region alone can be held mechanically responsible for the typical patterns of restricted ranges of HR *and* GH elevation as seen in frozen shoulder.

Shortening of the *anterior middle region* of the GHJC strongly restricted external rotation between 30° and maximal GH elevation. These results differ from those of Ferrari [10] and Turkel et al.;[44] they show that the middle GH ligament restrains external rotation exclusively in the mid-range of GH elevation. Our findings emphasize that there are important differences between the factual effects of capsular shortening on GH kinematics and the potential effects of capsular shortening determined by extrapolating data from unmodified joints.[10] The same statement holds for extrapolating data from selective cutting [44] of these regions of the GHJC.

Shortening of the *anterior inferior region* of the GHJC significantly decreased both external rotation and GH elevation. There are no quantitative data available in the literature that can be compared with these data of our study. Our data support the clinical finding that in frozen shoulder the anterior inferior region of the GHJC is involved. Like in frozen shoulder,[26] a contracture of the anterior inferior region of the GHJC restricts external rotation stronger than GH elevation (Figure 6.4f).

Many studies have addressed the role of the anterior regions of the GHJC in GHJ kinematics [8, 10, 12, 15, 16, 28] and in maintaining GHJ stability.[3, 7, 18, 35, 44] Only few deal with

the posterior joint capsule.[33,34] As shown by Branch et al.,[4] the posterior superior region of the GHJC mainly restricted internal rotation. Our data confirm these findings. The present study furthermore shows that shortening of the posterior inferior region of the GHJC by 20 mm restricted internal rotation, especially between 30° and 90° GH elevation. Shortening of this region also strongly restricted GH elevation. Our findings support the clinical impression of Matsen [25] that forced forward elevation in the case of a contracture of the posterior inferior region of the GHJC forces the humeral head in an anterior superior direction, promoting subacromial impingement.

6.4.2 Clinical relevance

In case of instability of the shoulder due to hyperlaxity of a region of the GHJC, principally, the amount of surgical shortening should be known to restore normal stability without limiting GHJ motion. Therefore, our data have relevance for the shoulder surgeon. The present study shows that the relationship between shortening of certain regions of the GHJC and GHJ motions is rather complex. First, a certain amount of capsular shortening had a GH elevation-dependent, nonlinear influence on HR. As an example, 30 mm shortening of the anterior inferior region of the GHJC caused at 0° GH elevation an 8.0 ± 5.0° (about 20%) decrease of external rotation of the humerus, at 30° GH elevation a 28.8 ± 10.8° (about 45%) decrease and at 60° 52.5 ± 9.2° (about 70%). For the data of the other capsular regions, see Tables 6.2 and 6.3. Second, this non-linear decrease of HR also depended on the amount of shortening. For example, at 45° GH elevation, 10 mm shortening of the anterior inferior region of the GHJ caused a 3.8 ± 2.8° decrease of external rotation. With 20 mm shortening, this decrease is about five times greater (18.2 ± 9.7°), and with 30 mm about twelve times (45.0 ± 7.1°).

Only recently, it has been shown that the anterior region of the GHJC is quite sensitive to shortening. Black et al.[2] studied the effect of a classic Banckart repair on the tension distribution in the GHJC during external rotation of the humerus. They also showed a nonlinear increase in capsular tension after operative shortening of the anterior region of the GHJC. However, from the data of their study, absolute changes in ROM could not be determined.

The present study shows that the effects of shortening of the GHJC on the ranges of HR and GH elevation strongly depend on the region that is shortened and the amount of shortening. Additionally, the effect of shortening on the range of HR was also GH-position dependent. As an example, surgical shortening the anterior inferior region of the GHJC by 20 mm, with the involved arm at the side, will leave ample external

rotation. However, at 60° GH elevation (as in throwing [19]) the same repair will limit external rotation to about 50% of the normal. Knowledge of these GH-position dependent effects of capsular shortening is important, since, it has been shown that abnormal translation of the humeral head during movements of the arm is associated with tightness of the GHJC.[15] Clinically, these abnormal translations are held responsible for early degenerative disease of the shoulder.[17] Such is the case with capsulorraphy arthritis, an uncommon but well-known complication of (tight) anterior capsular reconstruction.[17, 24] In a 15-year follow-up of patients with a Bankart-reconstruction, Rosenberg et al.[38] reported a relationship between radiographic degeneration of the GHJ and loss of external rotation at 90° of abduction and length of follow-up.

The data of our study are too extensive to memorize the outcome precisely while performing shoulder reconstruction. Nevertheless, they can be practically applied. The involved arm should be held close to the position that would just stretch the region of the GHJC that is reconstructed. To avoid subluxation, compression of the humeral head towards the glenoid fossa will center the humeral head. To assess the preliminary result of the reconstruction the range of HR through an arc of GH elevation can be tested and compared with that of the opposite (uninjured) shoulder. Obviously, this should be done after reconstruction, but before completion of the surgery.

6.5 Conclusions

1. The effects of capsular shortening on the ranges of HR and GH elevation show significant regional differences.
2. Shortening of the capsule has a nonlinear relation with the decrease of the ranges of GH elevation and HR.
3. This study sheds light on the relationship between limited glenohumeral elevation and limited HR as seen in frozen shoulder on one hand and on the other after reconstructive surgery of the GHJC.
4. The results of this study emphasize that to avoid overtightening of the joint capsule particular care must be taken in (anterior) capsular reconstruction.
5. The data of our study can be practically applied while performing shoulder reconstruction by assessing the physiological length (of a region) of the GHJC at the time of capsule closure.

Acknowledgements

The authors thank Paul G.H. Mulder, Ph.D. for his help with the statistical analysis, Cor Goedegebuur for technical assistance, Rosalie Kievit for assistance with the preliminary studies, and Cees Entius and Jan Velkers for their assistance with the preparation of the specimens.

References

1. **Bigliani LU, Weinstein DM, Glasgow MT, Pollock RG, Flatow EL.** Glenohumeral arthroplasty for arthritis after instability surgery. *J Shoulder Elbow Surg* 1995;4(2):87-94.
2. **Black KP, Lim TH, McGrady LM, Raasch W.** In vitro evaluation of shoulder external rotation after a Bankart reconstruction. *Am J Sports Med* 1997;25(4):449-53.
3. **Boardman ND, Debski RE, Warner JJ, et al.** Tensile properties of the superior glenohumeral and coracohumeral ligaments. *J Shoulder Elbow Surg* 1996;5(4):249-54.
4. **Branch TP, Lawton RL, Iobst CA, Hutton WC.** The role of glenohumeral capsular ligaments in internal and external rotation of the humerus. *Am J Sports Med* 1995;23(5):632-7.
5. **Brown AO, Hoffmeyer P, An KN.** The influence of atmospheric pressure on shoulder stability. *Orthop Trans* 1990;14:259-61.
6. **Bunker TD, Esler CN.** Frozen shoulder and lipids. *J Bone Joint Surg [Br]* 1995;77(5):684-6.
7. **Curl LA, Warren RF.** Glenohumeral joint stability. Selective cutting studies on the static capsular restraints. *Clin Orthop* 1996(330):54-65.
8. **Edelson JG, Taitz C, Grishkan A.** The coracohumeral ligament. Anatomy of a substantial but neglected structure. *J Bone Joint Surg [Br]* 1991;73(1):150-3.
9. **Ekelund AL, Rydell N.** Combination treatment for adhesive capsulitis of the shoulder. *Clin Orthop* 1992(282):105-9.
10. **Ferrari DA.** Capsular ligaments of the shoulder. Anatomical and functional study of the anterior superior capsule. *Am J Sports Med* 1990;18(1):20-4.
11. **Freedman L, Munro RR.** Abduction of the arm in the scapular plane: scapular and glenohumeral movements. A roentgenographic study. *J Bone Joint Surg [Am]* 1966;48(8):1503-10.
12. **Gagey O, Bonfait H, Gillot C, Hureau J, Mazas F.** Anatomic basis of ligamentous control of elevation of the shoulder (reference position of the shoulder joint). *Surg Radiol Anat* 1987;9(1):19-26.
13. **Gibb TD, Sidles JA, Harryman DTd, McQuade KJ, Matsen FAd.** The effect of capsular venting on glenohumeral laxity. *Clin Orthop* 1991(268):120-7.
14. **Greene WB, Heckman JD,** eds. The clinical measurement of joint motion. 1st ed. American Academy Of Orthopaedic Surgeons, 1994.
15. **Harryman DTd, Sidles JA, Clark JM, McQuade KJ, Gibb TD, Matsen FAd.** Translation of the humeral head on the glenoid with passive glenohumeral motion. *J Bone Joint Surg [Am]* 1990;72(9):1334-43.
16. **Harryman DTd, Sidles JA, Harris SL, Matsen FAd.** The role of the rotator interval capsule in passive motion and stability of the shoulder. *J Bone Joint Surg [Am]* 1992;74(1):53-66.
17. **Hawkins RJ, Angelo RL.** Glenohumeral osteoarthrosis. A late complication of the Putti-Platt repair. *J Bone Joint Surg [Am]* 1990;72(8):1193-7.
18. **Helmig P, Sojbjerg JO, Kjaersgaard-Andersen P, Nielsen S, Ovesen J.** Distal humeral migration as a component of multidirectional shoulder instability. An anatomical study in autopsy specimens. *Clin Orthop* 1990(252):139-43.
19. **Jobe CM.** Superior glenoid impingement. *Orthop Clin North Am* 1997;28(2):137-43.

20. **Johnston** T. The movements of the shoulder. A plea for the use of the 'plane of the scapula' as the plane of reference for movements occuring at the humero-scapular joint. *British J Surg* 1937;25:252-60.
21. **Kumar VP, Balasubramaniam** P. The role of atmospheric pressure in stabilising the shoulder. An experimental study. *J Bone Joint Surg [Br]* 1985;67(5):719-21.
22. **Leffert RD.** The frozen shoulder. *Instr Course Lect* 1985;34:199-203.
23. **Lippitt S, Matsen** F. Mechanisms of glenohumeral joint stability. *Clin Orthop* 1993(291):20-8.
24. **Lusardi DA, Wirth MA, Wurtz D, Rockwood CA, Jr.** Loss of external rotation following anterior capsulorrhaphy of the shoulder. *J Bone Joint Surg [Am]* 1993;75(8):1185-92.
25. **Matsen FA, Arntz** CT. Subacromial impingement. In: Rockwood CA, Matsen FA, eds. The shoulder. Philadelphia: W.B. Saunders, 1990:623-46. vol 2.
26. **Murnaghan JP.** Frozen shoulder. In: Rockwood CA, Matsen FA, eds. The shoulder. Philadelphia: W.B. Saunders, 1990:837-62. vol 2.
27. **Neer CSd.** Impingement lesions. *Clin Orthop* 1983(173):70-7.
28. **Neer CSd, Satterlee CC, Dalsey RM, Flatow EL.** The anatomy and potential effects of contracture of the coracohumeral ligament. *Clin Orthop* 1992(280):182-5.
29. **Neviaser RJ, Neviaser TJ.** Observations on impingement. *Clin Orthop* 1990(254):60-3.
30. **Nobuhara K, Sugiyama D, Ikeda H, Makiura M.** Contracture of the shoulder. *Clin Orthop* 1990(254):105-10.
31. **Nobuhara K, Supapo AR, Hino T.** Effects of joint distention in shoulder diseases. *Clin Orthop* 1994(304):25-9.
32. **O'Brien S, Neves MC, Arnoczky SP, et al.** The anatomy and histology of the inferior glenohumeral ligament complex of the shoulder. *Am J Sports Med* 1990;18(5):449-56.
33. **Ovesen J, Nielsen S.** Anterior and posterior shoulder instability. A cadaver study. *Acta Orthop Scand* 1986;57(4):324-7.
34. **Ovesen J, Nielsen S.** Posterior instability of the shoulder. A cadaver study. *Acta Orthop Scand* 1986;57(5):436-9.
35. **Ovesen J, Nielsen S.** Stability of the shoulder joint. Cadaver study of stabilizing structures. *Acta Orthop Scand* 1985;56(2):149-51.
36. **Ozaki J, Nakagawa Y, Sakurai G, Tamai S.** Recalcitrant chronic adhesive capsulitis of the shoulder. Role of contracture of the coracohumeral ligament and rotator interval in pathogenesis and treatment. *J Bone Joint Surg [Am]* 1989;71(10):1511-5.
37. **Poppen NK, Walker PS.** Normal and abnormal motion of the shoulder. *J Bone Joint Surg [Am]* 1976;58(2):195-201.
38. **Rosenberg BN, Richmond JC, Levine WN.** Long-term followup of Bankart reconstruction. Incidence of late degenerative glenohumeral arthrosis. *Am J Sports Med* 1995;23(5):538-44.
39. **Saha AK.** The classic. Mechanism of shoulder movements and a plea for the recognition of "zero position" of glenohumeral joint. *Clin Orthop* 1983(173):3-10.
40. **Schollmeier G, Uhthoff HK, Sarkar K, Fukuhara K.** Effects of immobilization on the capsule of the canine glenohumeral joint. A structural functional study. *Clin Orthop* 1994(304):37-42.
41. **Seeger LL, Gold RH, Bassett LW, Ellman H.** Shoulder impingement syndrome: MR findings in 53 shoulders. *AJR Am J Roentgenol* 1988;150(2):343-7.

42. **Shaffer B, Tibone JE, Kerlan RK.** Frozen shoulder. A long-term follow-up. *J Bone Joint Surg [Am]* 1992;74(5):738-46.
43. **Soifer TB, Levy HJ, Soifer FM, Kleinbart F, Vigorita V, Bryk E.** Neurohistology of the subacromial space. *Arthroscopy* 1996;12(2):182-6.
44. **Turkel SJ, Panio MW, Marshall JL, Girgis FG.** Stabilizing mechanisms preventing anterior dislocation of the glenohumeral joint. *J Bone Joint Surg [Am]* 1981;63(8):1208-17.
45. **Uitvlugt G, Detrisac DA, Johnson LL, Austin MD, Johnson C.** Arthroscopic observations before and after manipulation of frozen shoulder. *Arthroscopy* 1993;9(2):181-5.
46. **Warner JJ, Deng XH, Warren RF, Torzilli PA.** Static capsuloligamentous restraints to superior-inferior translation of the glenohumeral joint. *Am J Sports Med* 1992;20(6):675-85.
47. **Wiley AM.** Arthroscopic appearance of frozen shoulder. *Arthroscopy* 1991;7(2):138-43.

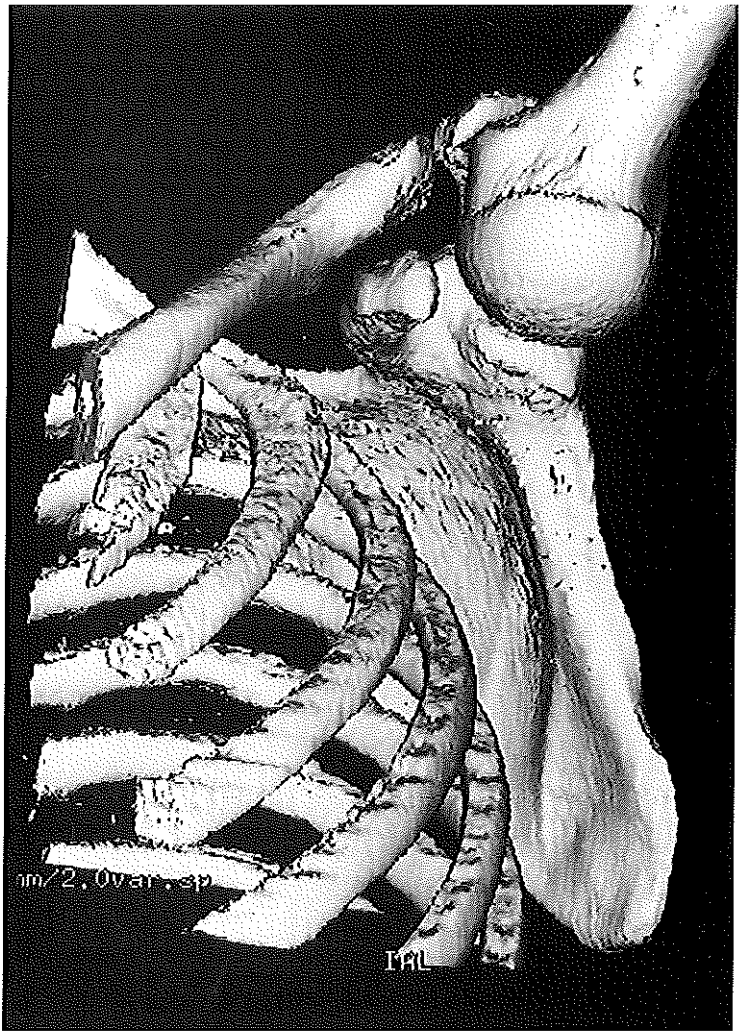
CHAPTER

7

General Discussion

ARTHUR DE GAST, MD¶

¶Dept. of Biomedical Physics and Technology, Faculty of Medicine, Erasmus University Rotterdam
Dept. of Anatomy, Faculty of Medicine, Erasmus University Rotterdam


nm/2.0var.sp
IAL

CHAPTER

7

General Discussion

The experimental anatomical and biomechanical approach in this study covers major issues of shoulder anatomy and of the etiology, diagnosis, and treatment of shoulder disorders. The functional anatomical aspects of the glenohumeral joint (GHJ) presented in this thesis form a basis for the clinical evaluation of patients with complaints of the shoulder. Functional properties of periarticular soft tissue structures, such as the role of the supraspinatus muscle-tendon unit in elevation of the arm, are most easily derived from descriptive anatomy. However, descriptive anatomy relates to the anatomic *position*, which can only be used with utmost care to describe *movements* of the GHJ. The assessment of position-dependent properties of periarticular soft tissue structures of the GHJ resulted in new insights in functional anatomy and in pathomechanics of the GHJ. This can be illustrated by the role of the tendon of the long head of the biceps brachii muscle (biceps tendon) in controling humeral rotation (HR; either internal or external rotation of the humerus). Traditional teaching refers to the long head of the biceps brachii as an internal rotator of the humerus.[1] Our experiments showed that this role is more complex, since biceps tendon load either restricted or facilitated both internal and external rotation of the humerus. The effect of biceps tendon load on HR depended on the degree of GH elevation and on the rotatory position of the humerus. Under 45° glenohumeral (GH) elevation biceps tendon tension *facilitated* internal and external rotation of the humerus, actively increasing the rotatory range of motion. Electromyography (EMG) studies support this finding clinically; they show that the long head of the biceps brachii muscle is active during external rotation with the arm at the side and becomes more active with a more externally rotated position.[9] Furthermore, EMG shows larger activity of the long head of the biceps brachii muscle in shoulders with rotator cuff tendon rupture compared to the unimpaired shoulder.[15] These patients are characterized also by a significantly larger biceps tendon diameter,[13] indicating use-

induced hypertrophy. Above 45° GH elevation, the role of the biceps tendon reversed and it no longer facilitated but *restricted* internal and external rotation of the humerus. This finding explains the mechanism of certain damage patterns of the origin of the biceps tendon and adjacent glenoid labrum (*S*uperior *L*abrum *A*nterior *P*osterior lesions) in throwing athletes with acquired hyperlaxity of the shoulder. Since in the late cocking position,[10] with active elbow flexion, abduction of the arm and maximal external rotation, further external rotation induces a combination of shear and tension stresses at the biceps tendon origin. We were able to assess the complex kinematic role of the biceps tendon, because the study design allowed for kinematic testing with biceps tendon load through a complete arc of GH elevation.

With the custom-made positioning and loading device kinematic data of the GHJ were obtained that corresponded well with GH ranges of motion that were measured *in vivo*.[2, 6, 8] This study showed that the relationship between GH elevation and the range of HR had two distinct characteristics. First, between 30° and 45° GH elevation, the range of HR reached its maximum. This result parallels the general opinion that at mid-elevation the GHJ capsule and incorporated GH ligaments are slackest, and permit the greatest range of HR.[4, 11] Second, above 45° GH elevation, HR strongly diminished and at maximal GH elevation all HR was lost (see Chapter 4, Figure 4.3). GH position-dependent tensioning of the passive restraints (the GHJ capsule and GH ligaments) caused this decrease of HR. These results are in agreement with earlier observations, showing that at maximal GH elevation, the GHJ reaches its maximally close packed position [7] or 'zero position';[14] this is considered the most stable joint position. New in our study was the quantitative description of the 'zero position' of the GHJ and of the relation between capsular length and GH elevation and HR. Clinically, knowledge of the relation between the range of HR and GH elevation provides better parameters for the assessment of GHJ mobility. The range of HR at several levels of GH elevation of the affected shoulder compared to the unaffected shoulder determine which part of the GHJ capsule is involved in either hypo- or hypermobility of the GHJ. For instance, patients with a contracture of the anterior superior region of the GHJ capsule, such as in longstanding subacromial impingement,[12] are expected to show a limitation of external rotation that is most pronounced between 15° and 45° GH elevation. Above 75° GH elevation, this limitation disappeared completely (see Chapter 6, Figure 6.5b). Such identification of the involved capsular region was previously done on the basis of humeral head translation (drawer tests) in patients with regional capsular insufficiency.[3] These drawer tests rely on joint laxity and muscle relaxation of the patient. Since laxity of the passive constraints of the GHJ is an essential feature of shoulder motion, it is difficult to draw a line between normal and pathologic laxity. Furthermore, the degree of

laxity varies considerably between individuals;[5] this necessitates a distinction between instability and hyperlaxity. In our opinion, assessment of HR provides a better diagnostic tool for the identification of regional capsular insufficiency. Furthermore, HR can be also used as a parameter for hypomobility of the GHJ.

Our experimental approach contrasts with that of studies using the anatomic position to describe GHJ function. With respect to the subacromial-subdeltoid bursa (SASDB), confusion already starts with the nomenclature. The subacromial (SA) and subdeltoid portions (SD) of the subacromial-subdeltoid bursa are frequently referred to as separate entities. We have shown that the SA and SD portions form one structure and that its nomenclature only relates to the anatomic position.

In most anatomical and medical illustrations, the SASDB slides completely under the coracoacromial arc during full elevation of the GHJ. However, we showed that only the deep wall (i.e., the bursal wall that lies directly over the rotator cuff) of the SD portion slided under the coracoacromial arc during GH elevation. Consequently, impingement related lesions of the SASDB only occur at predictable sites and do not involve the whole SASBD. The fact that these lesions involve limited areas of the SASDB advocates against complete bursectomy during subacromial decompression and open repairs of rotator cuff tears. Another argument against complete bursectomy is the ability of normal bursal tissue to promote repair of rotator cuff tissue damage.[16]

We demonstrated wrinkling of the walls of the SASDB during movements of the GHJ through arcs of motion that are usually applied during physical examination of the shoulder. Wrinkling of the deep wall of the SASDB perpendicular to the coracoacromial arc, indicating tensioning of this wall, was most apparent during external rotation of the humerus and during forward GH flexion with slight internal rotation. This wrinkling was most pronounced in areas of the SASDB where impingement lesions are frequently observed. These findings help to explain why, additionally to the impingement maneuvers, patients with subacromial impingement feel pain at the extremes of GH flexion and of external rotation. The complexity of SASDB transformations during movements of the GHJ and its close relation to the rotator cuff renders the specificity of tests designed to elicit 'bursal pain' questionable.

A new finding was also that the SASDB was attached to structures that control SASDB transformations during movements in the GHJ. This was well demonstrated with the supraspinatus muscle fibers that directly attach to the medial margin of the SASDB. These fibers create a control system pulling the medial margin of the SASDB medially over a distance of 7 mm between 0° and 45° GH elevation and reeling it out laterally 4 mm between 45° and maximal GH elevation.

In the first part of this thesis an extensive overview on functional and clinical shoulder anatomy is given. It represents the 'state of the art' knowledge that served as a reference for the results of our research. The aim of this study on the GHJ was to assess the functional anatomy of the SASDB to contribute to the knowledge of functional and of clinical anatomy of periarticular soft tissue structures, to determine how soft tissue structures contribute to the normal relationships between the range of HR and GH elevation in the scapular plane, and to assess the potential effects of surgical modification of the GHJ capsule on GH motion. The results of the experiments described in this thesis can serve as a basis for further clinical research on the assessment of normal and impaired mobility of the GHJ.

References

1. **Basmajan JV, Latif MA.** Integrated actions and functions of the chief flexors of the elbow. *J Bone Joint Surg [Am]* 1957;39 A:1106-18.
2. **Boone DC, Azen SP.** Normal range of motion of joints in male subjects. *J Bone Joint Surg [Am]* 1979;61(5):756-9.
3. **Cofield RH, Nessler JP, Weinstabl R.** Diagnosis of shoulder instability by examination under anesthesia. *Clin Orthop* 1993(291):45-53.
4. **Curl LA, Warren RF.** Glenohumeral joint stability. Selective cutting studies on the static capsular restraints. *Clin Orthop* 1996(330):54-65.
5. **Emery RJ, Mullaji AB.** Glenohumeral joint instability in normal adolescents. Incidence and significance. *J Bone Joint Surg Br* 1991;73(3):406-8.
6. **Freedman L, Munro RR.** Abduction of the arm in the scapular plane: scapular and glenohumeral movements. A roentgenographic study. *J Bone Joint Surg [Am]* 1966;48(8):1503-10.
7. **Gray H.** Arthrology. In: Williams P, Warwick R, eds. Gray's Anatomy. 36th ed. Edinburgh: Churchill Livingstone, 1980:458.
8. **Greene WB, Heckman JD,** eds. The clinical measurement of joint motion. 1st ed. American Academy Of Orthopaedic Surgeons, 1994.
9. **Habermeyer P, Kaiser E, Knappe M, Kreusser T, Wiedemann E.** Zur funktionellen Anatomie und Biomechanik der langen Bizepssehne. *Unfallchirurg* 1987;90(7):319-29.
10. **Jobe FW, Tibone JE, Perry J, Moynes D.** An EMG analysis of the shoulder in throwing and pitching. A preliminary report. *Am J Sports Med* 1983;11(1):3-5.
11. **Lippitt S, Matsen F.** Mechanisms of glenohumeral joint stability. *Clin Orthop* 1993(291):20-8.
12. **Neer CS.** Shoulder reconstruction. 1st ed. Philadelphia: W.B. Saunders Company, 1990.
13. **Perry J.** Muscle control of the shoulder. In: Rowe CR, ed. The shoulder. New York: Churchill Livingstone, 1988:26.
14. **Saha AK.** The classic. Mechanism of shoulder movements and a plea for the recognition of "zero position" of glenohumeral joint. *Clin Orthop* 1983(173):3-10.
15. Ting A, Jobe FW, Barto P. An EMG analysis of the lateral biceps in shoulders with rotator cuff tears. Third open meeting of the society of American Shoulder and Elbow Surgeons. California, 1987.
16. **Uhthoff HK, Sarkar K.** Surgical repair of rotator cuff ruptures. The importance of the subacromial bursa. *J Bone Joint Surg [Br]* 1991;73(3):399-401.

SUMMARY

In *Chapter 1* the central theme and aim of this thesis are introduced. The central theme adresses the glenohumeral joint position-dependent function of the peri-articular soft tissues. The aim of this study is threefold. First, to assess the functional anatomy of the subacromial-subdeltoid bursa in relation to the following questions. How are bursal transformations controlled during movements of the glenohumeral joint and what are the consequences of these transformations for the evaluation and treatment of glenohumeral joint disorders involving the subacromial-subdeltoid bursa? Second, to determine how articular soft tissue structures contribute to the normal pattern of scapular plane elevation and humeral rotation. Third, to assess the effects of (surgical) modification of glenohumeral joint capsule length on the range of glenohumeral elevation and humeral rotation.

In *Chapter 2* we describe the topographical, applied and functional anatomy of the shoulder. The limitations of the use of tropographical anatomy in interpreting clinical signs and symptoms are discussed. The importance of glenohumeral joint position-dependent descriptions of soft tissue anatomy is emphasized.

In *Chapter 3* we describe the largest bursa of the glenohumeral joint: the subacromial-subdeltoid bursa. Clinically, the subacromial-subdeltoid bursa is the most important bursa of the glenohumeral joint. In subacromial impingement the subacromial-subdeltoid bursa is frequently involved. Nevertheless, descriptions of the functional anatomy and of the pathomechanics of the subacromial-subdeltoid bursa are very confusing. Therefore, four unembalmed and fifty-nine embalmed human shoulder specimens were studied in detail to describe the anatomy of the subacromial-subdeltoid bursa. Subsets of specimens were used to perform kinematic studies and to produce cross sections. Special attention was paid to the structures that dynamically control the structural adaptation of the subacromial-subdeltoid bursa during movements of the shoulder.
The study showed that, during elevation of the glenohumeral joint in various planes, the rotator cuff tendons, parts of the coracobrachialis muscle and the short head of the biceps brachii control subacromial-subdeltoid bursal transformations. Furthermore, during elevation of the glenohumeral joint wrinkling of the superficial layer of the subacromial-subdeltoid bursa prevents accumulation of the surplus of bursal tissue. The results of this study showed that impingement related lesions of the walls of the subacromial-subdeltoid bursa predominantly occur at the superficial wall of the

subacromial portion of the subacromial-subdeltoid bursa and the deep wall of the subdeltoid portion. The results of this study are relevant for the medical practitioner because a better understanding of the functional anatomy of the subacromial-subdeltoid bursa will add to the diagnosis and treatment of patients with complaints of the shoulder. It is concluded that: 1) the subacromial-subdeltoid bursa has subacromial and subdeltoid portions that were present and continuous in all specimens; 2) division of the subacromial-subdeltoid bursa in a subacromial and subdeltoid prtion is based on a topographical taxonomy that relates to the *anatomic position*; 3) structural adaptations of the subacromial-subdeltoid bursa are dynamically controlled by parts of the rotator cuff, the coracobrachialis, deltoid and short head of the biceps brachii muscles; 4) there is no anatomical basis for specific 'bursa tests' during physical examination.

In *Chapter 4* we describe the relationship between glenohumeral elevation and humeral rotation (internal and external rotation of the humerus). Evaluation of the glenohumeral range of motion is an essential part of the physical examination of patients with shoulder complaints. To assess the range of humeral rotation through an arc of glenohumeral elevation in the plane of the scapula, two unembalmed and five embalmed shoulder specimens were used. Between 30° and 45° glenohumeral elevation all specimens reached the maximal range of humeral rotation: 144 $\pm$ 4° for the unembalmed and 155 $\pm$ 23.7° for the embalmed specimens. All humeral rotation was lost at maximal glenohumeral elevation: at 125° for the unembalmed and at 109 $\pm$ 13.4° for the embalmed specimens. The results were in accordance with the physiological glenohumeral range of motion.
The data of the present study are relevant for the medical practitioner because accurate evaluation of humeral rotation at different levels of glenohumeral elevation will add to the diagnosis and treatment of shoulder disorders, such as frozen shoulder, subacromial impingement and glenohumeral instability.
It is concluded that: 1) the mobility of adequately prepared embalmed human shoulder specimens corresponds well with ranges of glenohumeral joint motion that are measured *in vivo*; 2) the range of internal and external rotation of the humerus strongly depends on glenohumeral elevation; 3) the amount and direction of humeral rotation necessary to reach maximal glenohumeral elevation depends on the rotatory position of the humerus; 4) clinical assessment of the range of humeral rotation through an arc of glenohumeral elevation will add to the diagnosis of shoulder disorders, such as frozen shoulder, subacromial impingement and glenohumeral instability.

In *Chapter 5* we describe the glenohumeral position-dependent role of the tendon of the long head of the biceps brachii muscle (biceps tendon).

The biceps tendon is shown to either facilitate or restrict humeral rotation. Its effect on glenohumeral motion is strongly related to 1) the amount of biceps tendon load, 2) glenohumeral scapular plane elevation and 3) the rotatory position of the humerus. Under 45° of glenohumeral elevation, biceps tendon load facilitated internal and external humeral rotation, actively increasing the rotatory range of motion. Above 45° biceps tendon load restricted internal and external rotation of the humerus, actively increasing joint stability. In addition, biceps tendon load mimicked a restriction of humeral rotation commonly found in patients with frozen shoulder.

These findings on the function of the biceps tendon have consequences for the clinical interpretation of shoulder pain, shoulder instability, restriction patterns of glenohumeral range of motion and the use and interpretation of tests specifically related to the biceps brachii muscle.

It is concluded that 1) the biceps tendon functions as a monorail, guiding the humeral tuberosities to the biceps tendon origin, during glenohumeral elevation. By this mechanism it can be explained that from the anatomic position, maximal glenohumeral elevation can only be reached with approximately 65° external rotation of the humerus; 2) in a position of maximal glenohumeral elevation, humeral rotation is not possible; 3) biceps tendon tension has a complex effect on shoulder function. Obviously, description of biceps tendon function in the anatomic position does not suffice; 4) training of the biceps brachii muscle will on one hand facilitate humeral rotation and on the other provide additional stability of the glenohumeral joint.

In *Chapter 6* we describe the potential effect of contracture of the glenohumeral joint capsule on glenohumeral elevation and humeral rotation.

Loss of internal or external rotation of the humerus is a common complication of shoulder surgery. Five cadaver shoulder specimens were used to assess the effect of shortening of different regions of the glenohumeral joint capsule on internal and external rotation of the humerus and on glenohumeral elevation in the plane of the scapula. The specimens were obtained from embalmed human cadavers and tested intact and after methodical shortening the glenohumeral joint capsule. Shortening occurred in five regions (three at the anterior side, two at the posterior side) with segments of beaded chain and catches. Due to shortening of the beaded chains the range of motion decreased in consistent patterns, but the magnitude varied. A significant decrease ($p<0.0001$) of

external rotation of the humerus was found in case of shortening of the three anterior regions of the glenohumeral joint capsule, and of internal rotation ($p < 0.002$) in case of shortening of the two posterior regions. Shortening of the anterior inferior and posterior inferior regions of the glenohumeral joint capsule caused a 10° to 45°decrease of glenohumeral elevation. Typically, a nonlinear relation existed between shortening of the beaded chains and decrease of internal or external rotation of the humerus. Furthermore, the effect of shortening on the range of motion strongly depended on glenohumeral joint position. Above 45° glenohumeral elevation, both the anterior inferior and posterior inferior regions of the glenohumeral joint capsule shared in limiting internal and external rotation of the humerus.

The observations are relevant for the shoulder surgeon since they help to explain the relationship between a contracture of regions of the glenohumeral joint capsule and limited glenohumeral motion as seen in frozen shoulder and after operations that limit (external) rotation of the humerus.

It is concluded that: 1) the effects of capsular shortening on the glenohumeral range of motion show significant regional differences; 2) shortening of the capsule has a nonlinear relation with the decrease of the glenohumeral range of motion; 3) this study sheds light on the relationship between limited glnohumeral elevation and limited humeral rotation as seen in frozen shoulder on one hand and on the other after reconstructive surgery of the glenohumeral joint capsule; 4) the results of this study emphasize that to avoid overtightening of the joint capsule particular care must be taken in (anterior) capsular reconstruction. The data of our study can be practically applied while performing shoulder reconstruction by assessing the physiological length (of a region) of the glenohumeral joint capsule at the time of capsule closure.

In *Chapter 7* we discuss the results of the experiments that were conducted in chapters 3 to 6 with emphasis on the clinical implications.

SAMENVATTING

In *hoofdstuk 1* worden het centrale thema en het doel van dit onderzoek geïntroduceerd. Het centrale thema betreft de rol van de peri-articulaire weke delen bij normale en beperkte beweeglijkheid van het glenohumerale gewricht (schoudergewricht). Het doel van de studie is drieledig. Ten eerste, het vastleggen van de topografische en functionele anatomie van de klinisch belangrijkste slijmbeurs van het schoudergewricht, de bursa subacromialis-subdeltoidea in relatie tot diagnostiek en behandeling van schouderaandoeningen. Ten tweede, vastlegging van de relatie tussen enerzijds de mate van rotatie van de humerus en anderzijds de mate van elevatie van de humerus in het schoudergewricht in het vlak van de scapula (hier verder aangeduid als glenohumerale elevatie). Hierbij wordt tevens nagegaan wat de bijdrage is van de peri-articulaire weke delen. Tot slot, het vastleggen van het effect van (experimentele) inkorting van het kapsel van het schoudergewricht op de mate van rotatie van de humerus en de mate van glenohumerale elevatie.

In *hoofdstuk 2* wordt de 'state of the art' anatomie van het schoudergewricht beschreven aan de hand van de literatuur. Dit overzicht dient als referentiekader voor de hier beschreven experimenten.

In *hoofdstuk 3* wordt de bursa subacromialis-subdeltoidea besproken. Deze bursa is vrijwel aangedaan bij patiënten met subacromiale impingement (dat wil zeggen, abnormale compressie tussen humeruskop en schouderdak van delen van de bursa en de insertie-aponeurose van de rotatorenmanchet). De beschrijving in de literatuur van de vorm en functie van deze bursa is echter verwarrend. Realistische beschrijvingen van de vormverandering van de bursa subacromialis-subdeltoidea tijdens beweging in het schoudergewricht ontbreken. Om de leemte op te vullen worden in dit hoofdstuk de vorm, ligging en functie van de bursa subacromialis-subdeltoidea beschreven bij ongebalsemde en gebalsemde schouderpreparaten (n=63). Een aantal schouderpreparaten werd gebruikt voor kinematische proeven en voor het vervaardigen van doorsneden.
Aangetoond werd dat tijdens beweging in het schoudergewricht, vormveranderingen van bursa subacromialis-subdeltoidea mede bepaald worden door delen van de insertie-aponeurose van de rotatorenmanchet, de musculus coracobrachialis en de korte kop van de musculus biceps brachii. Ook werd aangetoond dat tijdens beweging in het

schoudergewricht bepaalde gebieden van de bursa subdeltoidea op spanning komen. Deze waarneming is relevant voor de interpretatie van klinische tests die speciaal voor het diagnostiseren van aandoeningen van de bursa ontwikkeld zijn. Aan de hand van de onderzoeksresultaten wordt aannemelijk gemaakt dat beschadigingen van de bursa subacromialis-subdeltoidea die gerelateerd zijn aan subacromiale impingement slechts in specifieke gebieden van de bursa voorkomen.
Geconcludeerd werd dat: 1) de bursa subacromialis en bursa subdeltoidea een geheel vormen; 2) de onderverdeling van de bursa subacromialis-subdeltoidea in een subacromiaal en een subdeltoidaal gedeelte berust op een topografische beschrijving welke betrekking heeft op de anatomische uitgangspositie; 3) vormveranderingen van de bursa subacromialis-subdeltoidea mede bepaald worden door de rotator cuff, de musculus deltoideus, delen van de musculus coracobrachialis en de korte kop van de musculus biceps brachii; 4) een anatomische basis voor (bestaande) 'specifieke' bursa-testen ontbreekt.

Hoofdstuk 4 betreft onderzoek naar de relatie tussen rotatie van de humerus en elevatie van de humerus in het schoudergewricht in het vlak van de scapula (glenohumerale elevatie). Kennis van deze relatie is van belang omdat het vaststellen van (de mate van de) gewrichtsmobiliteit een essentieel onderdeel is van het lichamelijk onderzoek.
Om in verschillende posities van glenohumerale elevatie de grootte van de endo- en exorotatie van de humerus te bepalen, werd gebruikt gemaakt van twee ongebalsemde en vijf gebalsemde gewrichtspreparaten van de schouder. Aangetoond werd dat de maximale bewegingsuitslag van endo- en exorotatie samen ($144 \pm 4^{\circ}$ voor de ongebalsemde preparaten en $155 \pm 23.7^{\circ}$ voor de gebalsemde preparaten) bereikt werd tussen 30° en 45° glenohumerale elevatie;. Rotatie van de humerus was onmogelijk bij maximale glenohumerale elevatie; van de ongebalsemde preparaten ligt dat maximum bij 125° glenohumerale elevatie, voor de gebalsemde bij $109 \pm 13.4^{\circ}$. De bewegings-uitslagen zijn in overeenstemming met *in vivo* gemeten waarden. De uitkomsten van deze studie zijn van belang voor de medicus practicus omdat nauwkeurige evaluatie van de rotatie van de humerus in verschillende posities van glenohumerale elevatie noodzakelijk is voor adequate diagnostiek en behandeling van verscheidene schouderaandoeningen (frozen shoulder, subacromiale impingement en instabiliteit van het schoudergewricht).
Er werd geconcludeerd dat: 1) de mobiliteit van adequaat geprepareerde schouders sterk overeenstemt met *in vivo* gemeten waarden; 2) de mate van endo- en exorotatie van de humerus sterk afhankelijk is van de mate van glenohumerale elevatie; 3) de mate en

richting van rotatie van de humerus, nodig voor het bereiken van maximale glenohumerale elevatie, afhankelijk is van de rotatie-positie van de humerus in het schoudergewricht; 4) nauwkeurige evaluatie van de endo- en exorotatie van de humerus van belang is voor het differentiëren tussen subacromiale impingement, *frozen shoulder* en instabiliteit van het schoudergewricht.

In *hoofdstuk 5* wordt de gewrichtspositie-afhankelijke functie van de oorsprongspees van de lange kop van de musculus biceps brachii (verder aangeduid als bicepspees) besproken.
Spanning in de bicepspees veroorzaakte ofwel facilitatie ofwel remming van de rotatie van de humerus. De grootte en richting van het effect was sterk afhankelijk van de grootte van de peesspanning, de mate van glenohumerale elevatie en de rotatiestand van de humerus. Bij het begin van glenohumerale elevatie (onder de 45°), veroorzaakte spanning in de bicepspees een toename van endo- en exorotatie. Boven de 45° werden endo- en exorotatie geremd; hierdoor nam de rotatoire stabiliteit van het schoudergewricht toe.
De uitkomst van dit experiment is van belang voor het begrijpen van schouderpijn die gerelateerd is aan beschadiging van de bicepspees en instabiliteit van het schoudergewricht. De uitkomst is ook belangrijk voor het op een juiste manier toepassen van provocatietesten van (de pezen van) de musculus biceps brachii.
Er werd geconcludeerd dat: 1) de bicepspees onder meer dienst doet als een monorail waarlangs het tuberculum minus en majus glijden tijdens glenohumerale elevatie. Aan de hand van dit monorail-mechanisme kunnen niet alleen de noodzaak van, maar ook richting en grootte van aan glenohumerale elevatie gekoppelde rotatie van de humerus worden verklaard; 2) het effect van spanning in de bicepspees op schouderbewegingen complex is; het effect is sterk afhankelijk van de grootte van de spanning en van de positie van het schoudergewricht. Hierdoor is een beschrijving van de functie van de bicepspees misleidend wanneer die alleen gerelateerd is aan de anatomische uitgangspositie; 3) gerichte training van de musculus biceps brachii gunstig is voor zowel de mobiliteit als de stabiliteit van het schoudergewricht.

In *hoofstuk 6* wordt het effect beschreven van het systematisch inkorten van (delen van) het gewrichtskapsel van het schoudergewricht.
Beperking van rotatie van de humerus is een veel voorkomende complicatie van schouderchirurgie. Om het effect van inkorting van het gewrichtskapsel op de beweeglijkheid van het schoudergewricht te bepalen werd gebruik gemaakt van vijf

gebalsemde schouderpreparaten. De bewegingsuitslagen (rotatie van de humerus en glenohumerale elevatie) werden gemeten voor en na het systematisch inkorten van (delen van) het gewrichtskapsel. Inkorting vond plaats met kettinkjes in verschillende kapselregio's (drie aan de voorzijde en twee aan de achterzijde van het gewricht). Inkorting van het gewrichtskapsel veroorzaakte consistente patronen van bewegingsbeperking. Inkorting aan de voorzijde veroorzaakte een significante ($p<0.0001$) vermindering van de exorotatie, inkorting aan de achterzijde van de endorotatie ($p<0.002$). Inkorting van de onderste kapselregio's aan de voor- of achterzijde van het gewricht veroorzaakte een beperking van 10-45° van de glenohumerale elevatie. Tussen de mate van inkorting en het verlies van beweeglijkheid bestond een non-lineair verband. Het effect van kapselinkorting op de rotatie van de humerus was sterk afhankelijk van de mate van glenohumerale elevatie.
De bevindingen van dit onderzoek zijn van belang voor de praktijk. Hiermee kan een verklaring worden gegeven voor de specifieke relatie tussen contracturen van het gewrichtskapsel en het te verwachten verlies aan beweeglijkheid van het schoudergewricht.
Er werd geconcludeerd dat: 1) het effect van het inkorten van het gewrichtskapsel op de beweeglijkheid van het schoudergewricht sterk afhankelijk is van exacte plaats van de ingekorte kapselregio; 2) de mate van inkorting van het kapsel een non-lineair verband heeft met de mate van bewegingsverlies van het schoudergewricht; 3) de resultaten van dit onderzoek bijdragen aan doorgronden van de relatie tussen een (al dan niet chirurgische aangebrachte) contractuur van het gewrichtskapsel en het te verwachten verlies aan beweeglijkheid van het schoudergewricht; 4) voorzichtigheid geboden is bij kapselreconstructie; strak hechten van het kapsel aan de voorzijde van het gewricht dient met name te worden voorkomen.

In *hoofdstuk 7* worden de experimenten bediscussieerd en in een klinisch kader geplaatst.

DANKWOORD

Het onderzoek dat ten grondslag ligt aan dit proefschrift is tot stand gekomen op de Afdeling Anatomie van de Faculteit der Geneeskunde en Gezondheidswetenschappen van de Erasmus Universiteit Rotterdam in nauwe samenwerking met de afdeling Biomedische Natuurkunde en Technologie van dezelfde universiteit. Aan de totstandkoming van dit proefschrift hebben velen meegewerkt.

In eerste instantie gaan mijn dank en waardering uit naar mijn promotor prof.dr ir C.J. Snijders. Beste Chris, je hebt me nog net op tijd bij de lurven gegrepen en een enorme impuls gegeven voor de afronding van dit proefschrift. Je gastvrijheid, enthousiasme en deskundigheid zijn van onschatbare waarde geweest. Hoe je er ooit zo gemakkelijk op kwam om aan de hand van mijn ondoorzichtige omschrijvingen in *no-time* de 'schouderopstelling' te construeren is voor mij nog steeds een raadsel.

Prof. dr J. Voogd, bedank ik voor de faciliteiten die mij al die jaren geboden zijn op de afdeling Anatomie. De beloofde artikelen zijn er gelukkig toch gekomen.

Prof.dr J.A.N. Verhaar dank ik voor zijn bereidheid als tweede promotor op te treden. Zijn enthousiaste opstelling heeft de afronding in een vlot tempo mogelijk gemaakt.

Rob Stoeckart, zonder jou was dit proefschrift er nooit in deze vorm gekomen. Je vermogen om de zoveelste versie van het manuscript met evenveel en vaak met nog meer aandacht te lezen is niet te evenaren. Nogmaals mijn excuses voor de onrijpheid van sommige geschreven stukken die ik op je bureau heb neergelegd. Ik hoop alleen dat je niet alsnog een verbeterde versie van het dankwoord inlevert.

Gert Jan Kleinrensink, bedankt voor de ruim 12 jaar plezier die ik in onze samenwerking met je beleefd heb. Je opgewekte karakter, eindeloze 'lolligheidjes' en inspiratie maakten het gemakkelijk om 'moeilijke' momenten te overwinnen.

Andry Vleeming, bedankt voor de mogelijkheden die je me hebt geboden me verder te verdiepen in de anatomie van het bewegingsapparaat. Een deel van mijn kwaliteiten als anatomie-docent heb ik zeker aan je te danken.

Karim Raissadat, jouw vlijt en precisie bij het meten van 'onze' schouders tijdens de kapselexperimenten hebben er toe bijgedragen dat deze promotie 'op tijd' kon plaatsvinden. Ik wens je veel succes in het vinden van een opleidingsplaats, je bent het meer dan waard om orthopedisch chirurg te worden. Aangezien dit boekje heel Nederland doorgaat kan de vorige zin misschien een bijdrage hieraan leveren.

Cor Goedegebuur, zonder echte werktekeningen maakte je een prachtige 'schouder-opstelling'. Onvermoeibaar kwam je steeds weer tegemoet aan de wisselende eisen die door de verschillende onderzoeken aan de constructie werden gesteld. Bedankt voor je vakkundige inzet en het feit dat je me vaak 'even tussendoor' hielp.

Paul Mulder, bedankt voor je hulp bij de statistische bewerking van hoofdstuk 6. Het meest significante moment voor beiden was ongetwijfeld dat waarop één ding wel direct duidelijk werd: we hadden het allebei even niet begrepen.

Prof.dr. B van Linge, wil ik bedanken voor het vertrouwen dat hij in mij heeft gesteld door mij aan te nemen voor de opleiding orthopedie. De positieve interesse die hij toonde voor de eerste schouder-onderzoekjes heeft mij mede gestimuleerd 'de schouderlijn' verder uit te bouwen.

Prof.dr J.S. Laméris, beste Han, hoewel het proefschrift uiteindelijk niet meer over schouder-echografie gaat, heeft juist de mogelijkheid die je mij bood om mij te bekwamen in de echografische anatomie van het schoudergewricht de basis gevormd voor dit proefschrift. Het was ook erg aardig dat je een toekomstig orthopedisch chirurg, toen nog student geneeskunde, zoveel vrijheid gaf in de radiologische keuken.

Arnoud de Leeuw, ik heb genoten van de tijd dat we samen schouder-echo's maakten op de donderdagmiddagen. Terecht heb je me gewezen op de gevaren van het 'hinein interpretieren' van de echografische bevindingen. Maar toch had ik het vaak bij het rechte eind, of niet soms? Marcel de Witte, je was een waardige opvolger van Arnoud de Leeuw. Jammer dat onze plannen van de *S*houlder *I*mpingement *S*tudy *R*otterdam vooralsnog met een SISR zijn afgelopen. Het was wel erg leerzaam.

Uiteraard bedank ik ook de overige medewerkers van de afdeling radiodiagnostiek van het Dijkzigt ziekenhuis en het Sophia Kinderziekenhuis (SKZ). Met name de laboranten van het SKZ hebben er alles aan gedaan om mooie 'dynamische' CT's en MRI's van de schouder te maken. De foto achter op het proefschrift getuigt hiervan.

Student-assistenten Edgar ten Holder, Jan Bart van Lent, Rosalie Kievit en M^2 (Marieke van Zwienen en Marieke Joosten), jullie hebben met mij alle mislukkingen doorstaan die bij het opstarten van onderzoek nu eenmaal gebeuren. Maar juist jullie doorzettingsvermogen en het feit dat jullie steeds net op tijd opzij sprongen als er weer eens een gewicht naar beneden viel heeft bijgedragen aan het slagen van het geheel.

Ir. G.Y. Roelofs, beste Gwen, zonder jouw *Exel*lente hulp had ik nooit zulke nette grafieken kunnen maken. Ik werd wel een beetje duizelig als ik naar de muiscursor keek tijdens je uitleg.

Ing. W.H. Groeneveld, Wim, veel dank voor je hulp bij het scannen van de lijntekeningen. Met het devies 'wat moet, dat moet goed' heb je vele uren geïnvesteerd.

In de afrondingsfase van dit proefschrift was de gastvrijheid van de afdeling Anatomie en met name de afdeling Biomedische Natuurkunde en Technologie voor mij de 'gouden gelegenheid' om het manuscript af te ronden. Nu dat het ei gelegd is bedank ik nogmaals alle medewerkers voor hun grotere en kleinere bijdragen.

Zo, lieve Akje, het zit erop, tot zo.

CURRICULUM VITAE

De schrijver van dit proefschrift werd in 1959 geboren te 's Gravenhage. Na het eindexamen VWO-B aan het Erasmus college in 1977 werd in 1982 de opleiding tot fysiotherapeut aan de Rotterdamse Academie voor Fysiotherapie volbracht.
Na een aantal malen uitgeloot te zijn, werd in 1984 gestart met de studie geneeskunde aan de Erasmus Universiteit te Rotterdam. Vanaf die tijd werkte de schrijver als student-assistent op de afdeling Anatomie I. Het vervaardigen van anatomische preparaten, het assisteren bij practica en het geven van hoorcolleges maakten deel uit van de bijna dagelijkse activiteiten. Bovendien werd anatomisch onderwijs georganiseerd voor studenten van de Rotterdamse Academie voor Fysiotherapie.
In de periode 1986-1990 breidde de klinisch-anatomische onderwijsactiviteiten van de PAOG te Rotterdam zich uit naar het Nederlands Paramedisch Instituut (destijds Stichting Wetenschap en Scholing Fysiotherapie) en universiteiten in het buitenland (Freie Universität Berlin en Goethe Universität te Frankfurt).
In de periode 1989-1991 werd in een samenwerkingsverband tussen de afdelingen Radiodiagnostiek (hoofd prof.em.dr H.E. Schütte), Anatomie I (hoofd prof.dr J. Voogd) en Orthopaedie (hoofd. prof.em.dr B. van Linge) onderzoek verricht naar de toepassingsmogelijkheden van echografie bij weke delen aandoeningen rond het schoudergewricht.
Het artsexamen werd in november 1990 afgelegd, vergezeld door een toezegging voor de opleiding orthopedie in het Academisch Ziekenhuis Rotterdam, Dijkzigt. In 1991 werd begonnen met onderzoek dat ten grondslag ligt aan dit proefschrift. Het onderzoek werd uitgevoerd op de afdelingen Anatomie I en Biomedische Natuurkunde en Technologie (hoofd prof.dr ir C.J. Snijders) van de Faculteit der Geneeskunde en Gezondheidswetenschappen te Rotterdam.
Van oktober 1991 tot oktober 1993 volgde de schrijver de chirurgische vooropleiding in het Medisch Centrum Alkmaar (hoofd dr P. de Ruiter / dr A.B. Bijnen). Van november 1993 tot november 1997 was de schrijver werkzaam als assistent geneeskundige in opleiding bij de afdeling Orthopedie van het Academisch Ziekenhuis Rotterdam, Dijkzigt (hoofd prof.dr J.A.N. Verhaar). Per 1 november 1997 volgde inschrijving in het specialistenregister als orthopedisch chirurg. In de periode van 1 november 1997 tot 1 maart 1997 was de schrijver werkzaam op de afdeling Biomedische Natuurkunde en Technologie.

ACKNOWLEDGMENT OF SOURCES

The illustrations at the beginning of each chapter are reproduced from the following sources:

Chapter 1, page 14

Vesalius A. De humani corperis fabrica. From reproduced copies of the 2nd edition. Basel: Johannes Oporinus, 1555. Presented by F.Hoffman-La Roche & Co Ltd. Basel Switzerland.

Chapter 2, page 22

Vesalius A. De humani corperis fabrica. From reproduced copies of the 2nd edition. Basel: Johannes Oporinus, 1555. Presented by F.Hoffman-La Roche & Co Ltd. Basel Switzerland.

Chapter 3, page 48

Kopsch F. Rauber's Lehrbuch der Anatomie des Menschen. Abteilung 3:Muskeln, Gefaesse. 11th ed. Leipzig: Georg Thieme, 1919.

Chapter 4, page 68

Vesalius A. De humani corperis fabrica. From reproduced copies of the 2nd edition. Basel: Johannes Oporinus, 1555. Presented by F.Hoffman-La Roche & Co Ltd. Basel Switzerland.

Chapter 5, page 84

Lucas DB. Biomechanics of the shoulder joint. Arch Surg 1973;107:425-32

Chapter 6, page 104

Bier A, Braun H, Kümmel H. Chirurgische Operationslehre. 2nd ed. Leipzig: Verlag von Johan Ambrosius Barth, 1917. Vol. 5.

Chapter 7, page 128

Courtesy of **Simon Robben**, MD. Dept. of Radiology, Sophia Children's Hospital.