



ISAKOS/ESSKA STANDARD TERMINOLOGY, DEFINITIONS, CLASSIFICATION AND SCORING SYSTEMS FOR ARTHROSCOPY

KNEE, SHOULDER AND ANKLE JOINT

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PREFACE

As a chairman of the ESSKA Arthroscopy Committee it was André Frank that launched the idea of a standard terminology within ESSKA. As his successor on the ESSKA Arthroscopy Committee I soon experienced the magnitude of this task. Moreover I felt it would be important to involve ISAKOS. Speaking the same scientific language is a worldwide wish. I therefore made it my primary goal to get this project running. I am very pleased to present you the result of our combined efforts. I am particularly happy with the collaboration of ESSKA and ISAKOS in this project.

It was not our intention to write a text book nor was it our intention to come up with new classifications or scoring systems. We defined the ten most important diagnoses for the knee, shoulder and ankle joint. For each diagnosis we defined the following::

1. A definition
2. Symptoms and signs: clinical classifications system
3. Pathology: X-ray classification and staging systems
4. Findings at arthroscopy: classification and staging systems
5. Outcome measures
6. Documentation sheet

We used existing documentation and the results of a systematic literature searches as well as the AO publication: muscular skeletal outcome measures and instruments 2005 and the Springer publication “Classifications and scores of the shoulder” 2006.

We involved the ACL-study group, the International Cartilage Repair Society and the Patellofemoral study group on sub headings. For the shoulder classification the ISAKOS upper extremity committee was involved. The terminology group consisted of the ESSKA Arthroscopy Committee Members, the ISAKOS Arthroscopy Committee Members and Experts in the field like Alan Anderson, Jon Karlsson, Philippe Hardy and R. Grelsamer.

The Shoulder section was coordinated by Roger Hackney, the Knee section by Bent Wulff Jakobsen and the Ankle section by Niek van Dijk. We had multiple face to face meetings with

the members of the arthroscopy committee and a 2-day consensus meeting in Amsterdam with the majority of the terminology group.

The manuscript has been approved by the ISAKOS scientific committee and the ISAKOS and ESSKA knee, sports and shoulder committees. The end result is not perfect but is hopefully a start of an ongoing process which aims at a universal scientific language for all arthroscopic surgeons.

I would like to thank all those involved for their enthusiastic participation and teamwork during my period as chairman of this terminology project.

C. Niek van Dijk

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SHOULDER

1. Introduction

The aim of this manuscript is to provide standard terminology, definitions and classification systems in the field of shoulder arthroscopy. This chapter will provide guidelines for the assessment of normal and pathologic aspects of the shoulder joint at arthroscopy.

Assessment of shoulder pathology starts by the clinical diagnosis. Clinical diagnosis is based on history and physical examination. The clinical diagnosis might be confirmed by radiological investigations including ultrasound scan, plain radiograph, CAT scan, MRI scan with or without arthrogram. In this chapter 10 different clinical diagnoses in the field of shoulder arthroscopy are described. Each clinical diagnosis is defined followed by a brief description of the symptoms and signs as well as the underlying pathology. Finally the arthroscopic findings are presented that typically belong to the clinical picture. Of these typical arthroscopic findings the definition is given followed by a classification concerning this abnormality.

Clinical Diagnosis and findings at arthroscopy: definitions and classifications

Intra-articular shoulder pathology is assessed in the following manner:

Clinical diagnosis with history and examination, appropriate investigation then →

Examination under anaesthetic, both shoulders and Arthroscopy

Clinical diagnosis	Arthroscopy
2. Asymptomatic shoulder Gleno-humeral joint	Typically one expects to find normal anatomy. Asymptomatic lesions might include: synovitis, positive drive through sign, labral fronding or detachment, calcification of the rotator cuff tendon, partial and complete tearing of the rotator cuff, fraying of the tendon of the long head of biceps, rupture of the long head of biceps, Hill Sachs lesion, articular cartilage degeneration. The centre of the glenoid normally has an area with thin or no articular cartilage

	<p>Anatomical variations include variations in the presence of the three described glenohumeral ligaments, the Buford complex, sublabral hole, superior recess of long head of biceps</p>
3. Subacromial Impingement	<p>Rotator Cuff pathology (also see. 3 cuff tear)</p> <p>Inflammation of the subacromial bursa</p> <p>Thickened subacromial bursa</p> <p>Petechial haemorrhage from contact</p> <p>Calcification of the supraspinatus tendon</p> <p>Anterior hook of the acromion</p> <p>Os acromiale</p> <p>Prominent coraco-acromial ligament</p> <p>Degeneration AC joint</p> <p>Coracoid impingement</p> <p>Bicipital tendonitis</p>
4. Rotator Cuff lesions	<p>Inflammation cuff</p> <p>Partial tear of cuff, bursal side and articular surface</p> <p>Full thickness tear</p> <p>Massive tear with retraction</p> <p>Inflammation of the subacromial bursa</p> <p>Calcification in cuff tissue</p> <p>Os acromiale</p> <p>Acromioclavicular joint osteoarthritis</p> <p>Subluxation of longhead of biceps</p> <p>Fraying, tearing, rupture of long head of biceps</p> <p>Acetabularisation of the acromion</p>
5a. Long head of biceps lesions	<p>Degeneration and fraying</p> <p>Partial and complete rupture</p>

<p>6d. Multidirectional instability</p>	<p>lesion to fraying Partial articular sided rotator cuff injury Capsular laxity, anterior and posterior-no tautness in posterior band of IGHL, drive through sign Rotator interval laxity</p>
<p>7. Glenoid fracture</p>	<p>Bony Bankart Hill Sachs lesion Reverse Hill-Sachs lesion Glenoid neck fracture extending onto articular surface Avascular necrosis</p>
<p>8. Osteoarthrosis/Articular cartilage lesions</p>	<p>Anterior glenoid osteoarthrosis Osteochondral lesion Humeral head and glenoid osteoarthrosis Rotator cuff arthropathy</p>
<p>9. Loose bodies</p>	<p>Loose bodies, bony bankart lesion, Hill-Sachs lesion</p>
<p>10. Synovitis/Capsulitis</p>	<p>Rheumatoid synovitis Pigmented vilonodular synovitis Frozen shoulder/Adhesive capsulitis Reflex sympathetic dystrophy and other syndromes</p>
<p>11. Acromioclavicular joint pathology</p>	<p>Cartilage disc tear Osteoarthrosis Instability; coraco clavicular joint and acromio-clavicular joint disruption, acute and chronic Lateral end of clavicle fracture</p>
<p>12. Septic arthritis</p>	

2. ASYMPTOMATIC SHOULDER

2.1. Introduction: The shoulder girdle is comprised of five joints and other anatomic structures. Intra- or extraarticular pathology of the shoulder does not always produce symptoms. Thorough knowledge of the intra- and extra-articular anatomy is required to be able to separate normal from abnormal complaints and findings.

2.2 Definition: A patient has an asymptomatic shoulder when there are no complaints and when test at physical examination are negative, regardless the fact whether there is any pathologic finding present on arthroscopy.

2.3 Symptoms and signs: There may be a history of intermitted or transient symptoms but there are no current complaints. There is a wide variation of examination findings within the normal range. It is important to compare the symptomatic and normal sides. Instability is defined as symptomatic laxity. Radiographic changes include early osteoarthritis, calcification of the rotator cuff, tearing of the rotator cuff, acromio-clavicular joint osteoarthritis.

2.4 Pathology: A variety of pathologies can remain asymptomatic. Symptoms can develop spontaneously, with prolonged activity, particularly overhead activity, throwing, heavy lifting and trauma either from a direct blow to the shoulder, a fall onto an outstretched arm or the arm being pulled.

2.5 Normal Findings at arthroscopy: The surgeon must develop a systematic approach to the intra-articular inspection of the joint. The superior labrum arises from the superior glenoid tubercle and labrum with a variable recess. The tendon of the long head of biceps arises from the superior glenoid tubercle and superior labrum, and passes over the humeral head into the bicipital groove between the tendons of subscapularis and supraspinatus. The tendon is supported by a loop or sling of fibrous tissue, the roof of the groove is strengthened by a superior transverse ligament. There is normally a degree of vascularity of the tendon. The wedge shaped glenoid labrum sits over the anterior surface of the glenoid with capsular attachments arising from the labrum. The variants of normal, the sub-labral hole and the Buford complex should be recognised. The articular surface of the humeral head is consistent, but there is frequently a central bare area in the middle of the glenoid. In the

asymptomatic shoulder the capsule is not usually inflamed. The three described glenohumeral ligaments have a variable presence and thickness, but are not stretched or torn. The rotator interval and associated recess between the superior and middle gleno-humeral ligaments is normal. The coracoid process lies in the recess but is not usually seen. The superior gleno-humeral ligament arises from the superior glenoid tubercle and passes across to the medial ridge of the bicipital groove. The middle gleno-humeral ligament is more variable and is absent in 8% to 30% of shoulders. (O'Brien 1990, Warner 1992) It arises from just beneath the superior glenohumeral ligament, passes across the tendon of subscapularis and attaches to the medial aspect of the lesser tuberosity. The attachments of the inferior gleno-humeral ligament arise at 3 and 9 o'clock on the glenoid, with a hammock like sling under the humeral head. The pouch of the shoulder should be examined for loose bodies and detachment of the gleno-humeral ligament from the humeral head, the so-called HAGL lesion. The tendon of subscapularis is easily visible. The capsule can be seen attaching to the humeral head in with a 'bare area' devoid of articular cartilage between the edge of the articular cartilage and the capsular and rotator cuff attachments. The Hill-Sachs lesion of anterior shoulder dislocation unless massive, generally has an area of articular cartilage between it and the bare area. The rotator cuff tendons arise smoothly from behind the bare area, though partial articular surface tears are common and age related.

Arthroscopic examination of the shoulder should include the sub-acromial bursal space. The bursa can be difficult to enter. Debridement of part of the wall is often required to enter the true bursal cavity. The bursal cavity extends from beyond the greater tuberosity to the acromioclavicular joint. The bursa has a smooth white surface covering the undersurface of the acromion and rotator cuff. Further debridement may be necessary to fully assess structures such as the coraco-acromial ligament, the acromio-clavicular joint the surface of the rotator cuff around the insertion and the acromion.

1.5.1 Definition asymptomatic shoulder: A patient is asymptomatic when there are no complaints of symptoms and when tests at physical examination are negative regardless the fact whether there is any intra-articular pathology present in the shoulder joint.

2.5.2 Variants of normal anatomy A sub labral hole may be found in up to 11% of shoulders (Snyder 1994) This is a variant of normal and should be differentiated between a traumatic or degenerate labral tear.

The Buford complex occurs less frequently, in approximately 1.5% of shoulders. The middle glenohumeral ligament is present as a thick cord-like structure running over the subscapularis tendon. The glenoid labrum is absent until the site of the beginning of the inferior glenohumeral ligament at 3 o'clock.

The glenohumeral ligaments are highly variable in their anatomy. The ligaments vary in all aspects: shape, size, position, attachments and even presence. The middle glenohumeral ligament is the most variable (Morgan 1992) In about 20% of cases the ligament is a thick, cord like structure passing over the tendon of subscapularis. In 5% the ligament is a thin sheet only.

The os acromiale is present in 3% of shoulders and represents a failure of fusion of the acromial epiphysis by the age of 25.

The long head of biceps may have a synovial mesentery which varies from a thin transparent sheet to an adhesion-like band. The tendon may have a cleavage line running down it as though it were composed of two bundles (Bunker 1991)

3. SUBACROMIAL IMPINGEMENT

3.1 Introduction: Subacromial impingement syndrome was described by Neer in 1972 and remains a common cause of shoulder pain. (Neer 1972)

3.2 Definition: Subacromial impingement involves repetitive trauma to the subacromial bursa and the rotator cuff as they pass through the subacromial outlet under the coraco-acromial arch. The impingement occurs as the tuberosities move into the outlet.

3.3 Symptoms and signs: The patient complains of pain when elevating the arm in abduction, typically with a painful arc of motion between 90-120 degrees, though this is highly variable. There may be relief of pain in a fully elevated position. There may be pain when reaching out, more often reaching behind, and consequently pain when dressing or undressing. The arm may feel weak. Typically there is pain at night which may be severe enough to disturb sleep. Examination demonstrates the painful arc of motion. There may be accompanying abnormalities of rhythm of motion including hitching of the shoulder girdle. Formal testing of the power of the individual rotator cuff muscles will reveal pain, but there may be associated

weakness in the absence of a rotator cuff tear. The special tests for impingement, the Hawkins and Neer signs are positive. Neer's test is positive with pain at the limit of forward flexion. Hawkin's test involves passively abducting the arm to the limit of range in the plane of the scapula in slight external rotation, followed by passive internal rotation to reproduce the pain.

Neer described three stages of impingement syndrome. Stage 1 oedema and haemorrhage 2 fibrosis and tendinitis 3 bone spurs and tendon rupture. This is of historical interest, but the stages do not necessarily follow one another, and the evidence is that the majority of tears of the rotator cuff occur on the articular surface and are not caused by impingement.

Jobe combined impingement and instability symptoms in athletes with 4 stages.

1 Pure impingement with no instability

2 Primary instability with capsular and labral injury with secondary impingment which can be internal impingement or subacromial

3. Primary instability because of generalised ligamentous laxity with secondary impingment

4. Pure instability and no impingement

(Jobe 1989)

3.4 Pathology: As a result of chronic repetitive impingement, the subacromial bursa becomes thickened and synovitic. Associated arthroscopic changes are numerous and may be directly or indirectly contributory or incidental findings. The long head of biceps is frequently involved in impingement and may be inflamed or degenerate and flattened.

The pathologies which physically reduce the subacromial space include a hooked acromion, os acromiale, osteoarthritis of the acromio-clavicular joint, thickening of the coraco acromial ligament, calcification within the rotator cuff, fractures of acromion glenoid or humeral head and thickening of the bursa.

Investigation of choice is with dynamic ultrasound scanning from an experienced radiologist.

Milgrom graded impingement changes from ultrasound images.

Stage 1 Bursal thickness 1.5 to 2.0mm

Stage 2 Bursal thickness over 2.0mm

Stage 3 Partial or full thickness tear of the rotator cuff

Indirect causes may have their effect by impaired function of the rotator cuff and subsequent up-riding of the humeral head under the influence of the deltoid muscle. These include full or partial thickness tearing of the rotator cuff, nerve injury or entrapment and altered posture.

Loss of control from instability may occur in hyperlaxity syndromes, instability lesions such as capsulo-labral tears and SLAP lesions. There is glenohumeral internal rotation deficit and labral pathology.

Impingement is frequently associated with adhesive capsulitis or frozen shoulder. In this situation passive external rotation of the glenohumeral joint is restricted and therefore not missed.

3.5 Findings at arthroscopy: Examination under anaesthetic and arthroscopy with full inspection of the glenohumeral joint allows associated pathology to be identified. The incidence of co-incidental pathology within the glenohumeral joint may be as high as 30%.

The clinical evaluation of the patient together with the results of pre-operative investigations will assist the surgeon in determining whether the intra-articular pathologies are incidental findings or are the cause of shoulder pain and secondary impingement.

The bursa may be thick walled and difficult to enter without initial debridement. The bursa may contain excess fluid and synovitis. There may be a discrete impingement area with petechial haemorrhages at the site of the impingement. The shape of the acromion with regard to the presence of a hook is assessed using more than one portal if necessary. The anterolateral edge of the acromion should not be removed beyond the attachment of the deltoid muscle.

The acromio-clavicular joint is examined, balloting the lateral end of the clavicle will help identify the position intra-operatively. Any inferior osteophytosis can be smoothed, and the lateral end excised if indicated pre-operatively.

Tearing of the rotator cuff may be obvious in the presence of a full thickness tear, partial thickness tears extending onto the bursal surface may require debridement of the bursa and careful examination of the insertion whilst moving the shoulder into a number of positions.

Coracoid Impingement: Coracoid impingement is characterised by anterior shoulder pain aggravated by flexion adduction and internal rotation. The tendon of subscapularis is caught between the lesser tuberosity and the coracoid process (Gerber 1985) Investigation with CT scan may demonstrate narrowing of the space between the humerus and coracoid process, and occasionally a full or partial thickness tear of the tendon of subscapularis. Arthroscopic findings include a prominent coracoid process in the rotator interval, with or without an associated synovitis. The tendon of subscapularis may be tented as the shoulder is moved into adduction and internal rotation. The structure of the tendon may be damaged with evidence of tearing. Successful rthroscopic coracoplasty and repair of the subscapularis has been reported. (Lo 2003)

Internal Impingement: Walch and Jobe described an internal impingement lesion that occurs in overhead athletes. The articular surface of the rotator cuff impinges against the posterior superior portion of the glenoid as the arm is placed in full abduction and external rotation. Chronic fraying and partial thickness tearing of the insertion of supraspinatus is seen along with a lifting of the posterior superior glenoid labrum, creating a variant of the SLAP lesion.

EUA reveals anterior shoulder laxity/instability and posterior capsular tightness. Arthroscopic findings associated with anterior instability including a drive through sign, glenoid labral damage, but specifically under surface tearing of the supraspinatus which can be observed impinging upon and elevating the posterior superior glenoid labrum. (Davidson 1995, Walch 1992)

4. ROTATOR CUFF LESIONS

4.1 Definition: The rotator cuff is formed by the coalescence of four muscles arising from the scapula and inserting into the tuberosities of the head of the humerus. The understanding and treatment of rotator cuff pathology has been greatly enhanced by shoulder arthroscopy.

Rotator cuff disease is a spectrum of conditions ranging from symptomatic tendinopathy through partial and full thickness tearing to destructive rotator cuff arthropathy.

4.2 Symptoms and signs: Symptoms attributable to rotator cuff disease are very similar to those of subacromial impingement. Pain may be gradual in onset or follow trauma such as a

fall. Symptoms may begin after a fall in a previously asymptomatic joint, though radiographs or ultrasound scan reveal the presence of a pre-existing chronic tear. Patients may complain of night pain, pain with overhead activity and pain on reaching out or behind and dressing or undressing. In addition, there may be a greater element of weakness and loss of control of movement. In extreme cases the shoulder may become flail with complete loss of active abduction, or require the assistance of the other arm to reach an overhead position. Passive movements generally remain full. Loss of motion may be associated with osteoarthritis or adhesive capsulitis. Despite this there are many individuals with massive tears of the rotator cuff which have deteriorated into a rotator cuff arthropathy who demonstrate a full range of motion without loss of control.

4.3 Pathology: The primary cause of tendon degeneration and tearing is aging. (Uthoff 1991) The changes include loss of fibrocartilage at the insertion, reduction in vascularity, fragmentation of the substance of the tendon and disruption of the attachment to bone via Sharpey's fibres. With the exception of some stages in the progression of calcific tendonitis and with some rotator cuff tears, these changes are asymptomatic.

The incidence of rotator cuff tears increases with age. Tears are rare under the age of 40, but may be associated with trauma such as dislocation, contact sports and throwing injuries. The most vulnerable area is the articular surface attachment, and 95% of rotator cuff tears begin as 'rim rents', partial thickness avulsions.

Milgrom found that the incidence of partial or full thickness tears increased markedly after 50 years of age, tears were present in over 50% in the seventh decade and 80% in subjects over 80 years of age. This corresponds with other investigators. (Milgrom 1977?, Chard 1991)

Matsen describes eight clinical entities associated with rotator cuff pathology (Matsen 1998)

- 1 Asymptomatic cuff failure
- 2 Posterior capsular tightness
3. Subacromial abrasion without significant defect in the rotator cuff
4. Partial thickness cuff lesion
5. Full thickness cuff tear
6. Cuff tear arthropathy
7. Failed acromioplasty

8. Failed cuff surgery

The size, position severity and shape of tear are important considerations when assessing the tear for repair.

Partial thickness tears are classified according to the position

Bursal side partial tear of rotator cuff BPRCT

Articular side partial tear of rotator cuff APRCT

A PASTA lesion is a Partial Articular Supraspinatus Tendon Avulsion. This acronym is frequently employed in the literature to describe this pathology.

Cofield (Cofield 1982) based his classification according to size,

Full thickness tears, based on maximum length of tear in any direction

Small > 1cm

Medium 1-3 cm

Large 3-5 cm

Massive >5cm

Snyder published the Southern California Orthopaedic Institute rotator cuff classification system. This is a comprehensive classification including the size position and quality of tendon.

Location

A Articular surface

B Bursal surface

C Complete tear

Partial thickness tears

0 Normal

1 Minimal superficial bursal or synovial irritation or slight capsular fraying over a small area

2 Fraying and failure of some rotator cuff fibres in addition to synovial bursal or capsular injury

3 More severe rotator cuff injury fraying and fragmentation of tendon fibres often involving the whole of a cuff tendon, usually <3cm

4 Very severe partial rotator cuff tear that contains a sizeable flap tear and more than one tendon

Full thickness rotator cuff tears

C1 Small complete tear, pinhole sized

C2 Moderate tear <2cm of only one tendon without retraction

C3 Large complete tear with an entire tendon with minimal retraction usually 3-4 cm

C4 Massive rotator cuff tear involving 2 or more rotator cuff tendons with associated retraction and scarring of the remaining tendon. The tear is often L shaped . (Snyder 2003)

Recognition of the shape of the tear is vital in decision making when repair is undertaken.

Ellman and Gartsman (Ellman 1993) described 5 patterns

1 Crescent

2 Reverse L

3 L shaped

4 Trapezoidal

5 Massive tear

Massive tears with retraction may be not repairable. Goutallier has a classification of muscle degeneration and fatty degeneration using CT scan which can aid decision making and prognosis. Stages 3 and 4 have less chance of return to function

Stage 0 Normal muscle

Stage 1 Some fatty streaks

Stage 2 Less than 50% fatty muscle atrophy

Stage 3 50% fatty muscle atrophy

Stage 4 Greater than 50% fatty muscle atrophy

4.4 Findings at arthroscopy: The spectrum of rotator cuff disease necessitates a full inspection of all structures within the glenohumeral joint and subacromial bursa. In particular the state of the articular surface, glenoid labrum and long head of biceps should be observed. The rotator cuff should be inspected methodically noting the integrity of the superior glenohumeral ligament and transverse ligament over the long head of biceps. The shoulder

should be internally rotated to observe the insertion of the subscapularis tendon. The tendons of supraspinatus and infraspinatus are inspected for articular surface damage, any tendinopathic changes and of course the presence of any full thickness tears. The tendon of biceps should be probed to assess stability and mobility.

Bursoscopic findings vary from normal, as until articular surface tears reach the bursal surface, then histology is frequently normal, through to the acetabularisation of the under surface of the acromion found in rotator cuff arthropathy. (Suenaga 2002)

Debridement and probing of the bursa may be necessary to detect small full or partial thickness defects. Calcification of the rotator cuff tendon may be an incidental finding or associated with a synovitis within the bursa.

The shape size mobility and complexity of the tendon rupture should be recorded as these factors determine the feasibility and prognosis of repair. Mobilisation of the tendon by freeing of the bursal adhesions is required to fully assess the potential for repair. Arthroscopic release as far medial as the spine of the scapula and scapular notch, and the coracoid process is achievable.

Calcific Tendinitis ????

Definition: Calcific deposits which arise within the tendon of the rotator cuff itself. Calcific tendonitis is a common disorder of unknown aetiology. The process involves deposition of calcium which passes through several phases but eventually leads to spontaneous healing. The incidence is approximately 20% in asymptomatic shoulders in the susceptible age group, 31-40 years of age. The incidence is higher in the painful shoulder.

Calcification in the subacromial space must be distinguished from ectopic calcification around the edges of a massive rotator cuff tear.

Symptoms and signs: Calcific tendonitis is a cyclical condition passing through several phases. Symptoms vary in each phase, but include shoulder pain and impingement pain. The severe pain of acute calcific tendonitis occurs in the phase of resorption. It should be emphasised that the majority of individuals with calcific deposits remain largely asymptomatic.

Pathology: The aetiology of the condition is unknown. Fibrocartilaginous tissue with chondrocyte like cells. The deposits are thought to arise in areas of relative avascularity Uthoff proposes a

1 Pre-calcific stage

Metaplasia of tenocytes into chondrocytes

2 Calcific stage

Formative phase

Resorptive stages

3 Post-calcific phase

Investigation includes plain radiograph and ultrasound scan. Dynamic ultrasound scanning allows assessment as to whether impingement is occurring with abduction. Attempts at classification have used the size and/or density of the calcification.

Bosworth divides the deposits into tiny; medium, less than 1.5cm; large greater than 1.5cm

Mole's (Mole 1993) classification has 4 types

A calcification dense homogenous with clear contours

B calcification dense split/separated with clear contours

C calcification non-homogenous serrated contours

D calcification dystrophic calcification of the insertion in continuity with the tuberosity

Findings at Arthroscopy: The rotator cuff may appear vascularised on the articular surface. Partial resection of the bursa is frequently required to observe the bursal surface of the rotator cuff. Swelling within the rotator cuff may indicate the site of the deposit. The rotator cuff is examined by needling with a spinal needle. The calcific material is released by dividing rotator cuff in the line of the fibres. The appearance of the calcium is usually of a toothpaste like consistency, occasionally with solid flakes. Most authors add a subacromial decompression procedure in addition to the release of calcium.

5a. LONG HEAD OF BICEPTS LESIONS

5a.1 Introduction: The function and anatomy of the biceps tendon has been the subject of much investigation on recent years. The attachment, function, pathology and treatment are all under debate.

5a.2 Definition: The long head of biceps has an intra-articular portion. The tendon arises from the superior labrum and supra-glenoid tubercle passing over the head of the humerus into the bicipital groove. It is bounded by the subscapularis and superior gleno-humeral ligament anteriorly and supraspinatus posteriorly.

5a.3 Symptoms and signs: Patients may complain of a great variety of symptoms. Lesions of the biceps tendon and its attachment may cause all of the following: shoulder pain, referred pain anteriorly, posteriorly and down the arm, instability, catching, clicking, may mimic acromio-clavicular joint pain.

There are a number of tests which have been described, none of which are sufficiently sensitive or specific to be used alone with any reliability and clinical suspicion of a lesion of biceps is paramount. Examination should include test for instability, impingement and acromio-clavicular joint pathology. Investigations may include MRI, but even the addition of arthrography does not give sufficient sensitivity or specificity to be relied upon. EUA and arthroscopy in experienced hands gives direct information and allows definitive treatment to occur at the same time.

The 'Pop-eye sign' of a bunched biceps muscle indicates a complete rupture of the long head.

5a.4 Pathology: Sublabral recess. This is a variant of normal anatomy differentiated from a tear by the presence of articular cartilage up to the attachment of the labrum.

Habermeyer and Walch Classification of Biceps lesions (Habermeyer 1996)

1 Origin

2 Interval lesions

A Biceps tendonitis

B Isolated ruptures

C Subluxation

Type 1 Superior subluxation with partial or complete tear of the rotator interval sling (the circular sling of the SGHL and coracohumeral ligaments). There may be an associated tear of supraspinatus.

Type II Subluxation at the groove. The tendon slips over the medial rim of the groove and sits upon the lesser tuberosity, with detachment of the superior portion of subscapularis.

Type III Malunion or non union of the lesser tuberosity

3 Dislocation of long head of biceps

Type 1 A extra-articular with a partial subscapularis tear

The biceps tendon is completely dislocated and lies over the lesser tuberosity. The deep part of subscapularis remains intact. The SGHL and coraco-humeral ligaments are detached. There is frequently an associated tear of supraspinatus.

Type 1 B extra-articular with an intact subscapularis tendon

Type 2 Intra-articular dislocation of the long head of biceps combined with a complete tear of the subscapularis tendon.

Biceps is widened and flattened. There may be erosions or impending rupture of the tendon. Subscapularis is completely torn from the lesser tuberosity adjacent to the lesser tuberosity, biceps dislocates into the joint space infero-medially

Rupture of biceps associated with a cuff tear

Pulley lesions (Habermeyer 2004)

Group 1 isolated lesions of the SGHL

Group 2 lesion of the SGHL and partial articular side lesion of the supraspinatus tendon

Group 3 combination of a lesion of the SGHL and deep surface tear of the subscapularis tendon

Group 4 combination of a lesion of the SGHL and a deep surface of the tear of the supraspinatus and subscapularis tendon

5b. SLAP LESIONS

Snyder (Snyder 1990), superior labrum anterior and posterior

Type 1 fraying without frank tear or detachment

21% a frequent incidental finding in middle aged or elderly patients

Type 2 traumatic detachment of the biceps anchor without a midsubstance tear

55% the middle glenohumeral ligament may be included in the lesion

Type 3 longitudinal mid-substance tear of the superior labrum with a bucket handle tear fragment as often seen in the meniscus of the knee

9% usually very mobile

Type 4 as type 3 but the tear extends in to the biceps tendon so that a portion of the tendon is attached to the displaced fragment

10% variable split in biceps tendon

Maffet's (Maffet 1995) Subclassification of SLAP lesions.

Types 1 – 4 equivalent to the Snyder classification

Type 5 Antero-inferior bankart lesion which continues superiorly to include separation of the biceps tendon

Type 6 Unstable flap tear of the labrum with biceps tendon separation

Type 7 SLAP lesion that extends antero-inferiorly along the labrum to below the MGHL

Morgan (Morgan 1998) subdivided type 2 SLAP lesions to anterior/posterior/combined lesions

Internal impingement, Walch and Jobe. Posterior superior avulsion of the labrum which has been pushed off the normal attachment by pressure from the greater tuberosity and undersurface of supraspinatus which frequently suffers a partial thickness tear.

5a.5 Findings at arthroscopy: The stability of the biceps and its anchor is assessed using a probe. The extend of any sublabral recess is probed and the tendon lifted to ascertain any tearing of the deep under-surface of the attachment. Anything other than smooth articular cartilage is pathological.

The presence and extent of any SLAP lesion is noted, and any association with a sublabral hole or Buford complex, which may give arise to confusion. It is helpful when assessing this to hold the tissue under suspicion against the glenoid rim using an arthroscopic grasper and then external rotate the shoulder with the elbow held against the chest wall. Early lifting away of the tissue indicates an association with the variants described, and repair to the glenoid of that portion of tissue is contra-indicated.

The tendon appearance may be degenerative with fraying and fronding, partial thickness tearing and changes in shape and size, becoming wider and flatter.

Hour glass tendon

A swelling of the substance of the tendon may catch in the bicipital groove. This may cause an effect very similar to the ‘triggering’ of a trigger thumb. There is loss of the last 30 degrees of flexion/abduction. Arthroscopically a probe is used to pull the tendon out into the glenohumeral joint space reproducing the triggering

6. SHOULDER INSTABILITY

6.1 Introduction: There is not a precise or universally agreed upon definition of instability, or of the clinical or arthroscopic criteria to make the diagnosis. Multiple systems of classification have been advocated, based upon anatomical injury, direction of humeral head translation, frequency (acute versus recurrent onset), degree (complete versus partial dislocation), type of symptoms, or physical findings^{1,2} (Farber 2006, Bole 2007). It appears that several anatomic and biomechanical factors are required to produce the symptoms that are usually associated with shoulder instability³⁻⁵ (Labriola 2005, Lee2000, Curl 1996). This review will utilize a functional definition for instability- excessive humeral head translation in relation to the glenoid that disrupts normal coupled biomechanical motions and function of the shoulder joint.

6a. TRAUMATIC ANTERIOR SHOULDER INSTABILITY

6a.1 Definition: Complete dislocation (luxation) of the humeral head in relation to the glenoid, following significant trauma, such as a fall on the outstretched arm, a hyperabduction/external rotation force, or a direct blow from posterior/superior. In the initial and subsequent incidents, the path of dislocation is anterior, and the humeral head is seen or felt to be in an anterior or anterior/inferior position before reduction⁶ (Matsen 1991)

6a.2 Symptoms and signs: The presenting symptom is dislocation, either present or reduced. The mechanism of the initial injury should be noted, and in recurrent cases, what positions reproduce the symptoms or cause the re-dislocation. It is helpful to know the mechanism of reduction if the joint has already been reduced. In the acute setting, anterior pain and muscle weakness in abduction or rotation may be seen. Neurologic injury is rare, but must be checked, especially the axillary nerve. In chronic cases, pain and actual dislocation may be present at night. The physical exam should note if there is a position of apprehension, usually

abduction more than 80 degrees and external rotation more than 45 degrees, with patient complaint of impending dislocation. Other exam tests include the anterior load and shift, external rotation relocation, sulcus sign, and anterior drawer¹ (Farber 2006). Plain x-rays are needed to show the adequacy of reduction and any bony damage. Advanced imaging, either CT arthrography or MRI, may be helpful in determining the extent of the anatomic injury and in planning surgical interventions.

6a.3 Pathology: The major pathologic lesion is the Bankart lesion, a separation of the capsulolabral tissues from the glenoid socket. The acronym TUBS was devised to classify individuals who have traumatic unidirectional instability associated with a Bankart lesion which responds well to surgery⁷ (Thomas 1989). Most commonly, the injury is soft tissue only, with detachment of the capsulolabral complex off the bone, or including a tear through the substance of the capsule. The tissue may lie a variable distance from the anatomic position. “Bony Bankarts” include a variable amount of glenoid bone in the detached tissue. The bone may be a slight avulsion of a strip of bone, but may include a sizable portion of the anterior/inferior glenoid rim⁸ (Burkhart 2000). The capsular tissues may be detached from their humeral origin the Humeral Avulsion of the Glenohumeral Ligament injury⁹ (Richard 2004). The posterior superior portion of the humeral head may be injured as a result of humeral head impaction on the glenoid during dislocation. Depending on the position of the arm at the dislocation, the orientation of the groove may make the bone more likely to dislocate in abduction/external rotation (engaging Hill- Sachs lesion) or to have minimal effect on recurrent dislocation (non engaging Hill- Sachs lesion)⁸ (Burkhart 2000)

6a.4 Findings at arthroscopy: There are multiple possible individual/combined findings at arthroscopy. This requires a thorough evaluation of all anatomic areas of the joint. It starts with an evaluation of translation under anesthesia. Single plane translation motions in anterior, posterior, and inferior directions should be done, and the amount of motion categorized as 1+ (slight movement to the glenoid rim), 2+ (over the glenoid rim) and 3+ (lock out)¹⁰⁻¹² (Hawkins 1987, Krishnan 2004, McFarland 1996) When viewing inside the joint, the use of probes and graspers to probe, pull, and check the attachments of the anatomic structures can determine the security of the attachments. The exam should be done in a systematic manner, from superior to inferior and anterior to posterior. The superior exam may show a superior labral injury, demonstrated as detachment with a peel back of the labrum from the glenoid. About 20% of patients with anterior instability will show superior labral

injuries. The anterior exam should look for the Bankart lesion and/or capsular laxity¹³ (Burkhart 2003). Separation of the damaged tissue from the glenoid may be linear, or tear into the capsule, and may be widely displaced or may be covered by a thin covering of normal appearing tissue. Probing may be needed, and the tissue should be mobilized to bring it back to the normal attachment area. The tissue needs to be brought back to the normal position on the glenoid surface in order to restore normal anatomy and the normal “bumper” effect of the labrum on the humeral head. Medial displacement of the tissue on the glenoid neck- the ALPSA lesion- is common¹⁴ (Neviaser 1993). The capsule should also be evaluated for laxity. A large patulous capsule will indicate lack of tension and support of the humeral head. The “drive through” sign, in which the scope can be moved under the humeral head from superior to inferior in the joint, can help determine capsular laxity¹⁵ (McFarland 2001). Evaluation of the posterior band of the inferior glenohumeral ligament is a sensitive indicator of capsular laxity. Excessive laxity is shown as lack of tension in the band. The scope should be directed to the humeral attachment of the capsule to evaluate possible humeral avulsion lesions (HAGL). The posterior exam is similar to the anterior exam, with particular attention paid to the capsular attachment site. The Kim lesion, in which a deficiency of the attachment is covered by a thin piece of apparently normal tissue, is relatively common in patients with posterior instability symptoms, but may also be present in patients with primarily anterior instability¹⁶ (Kim 2004). Probing and palpation of the rim attachment is necessary to confirm absence of injury to the posterior labrum.

6b. TRAUMATIC POSTERIOR INSTABILITY

6b.1 Definition: Complete dislocation (luxation) of the humeral head on the glenoid following significant trauma, either due to a direct blow to the anterior shoulder or an extreme horizontal adduction maneuver. The dislocated humeral head is posterior to the glenoid. The injury may be acute or recurrent.

6b.2 Symptoms and signs: The dislocation may be present but is usually reduced. The mechanism of reduction should be identified if the luxation is reduced. The pain is usually posterior, but may be anterior as well if there has been direct blow trauma. If there has been recurrence, the position and mechanism of dislocation should be identified. The position of apprehension, usually horizontal adduction or forward flexion, should be identified. There is usually difficulty, pain, and weakness when load bearing in forward flexion, such as a bench press. The physical exam shows pain posteriorly along the joint line, and will show

apprehension or translation with a horizontal adduction position of the arm. There may be a palpable subluxation of the humeral head over the posterior glenoid rim, with reduction as the arm is placed in neutral rotation. This is known as a “jerk sign”, indicating the presence of a jerking sensation as the humeral head reduces into the glenoid articulation. Sulcus testing is performed with the arm at the side, both in 0 and 30 degrees of external rotation to determine the laxity of the rotator interval.

6b.3 Pathology: The major pathologic lesion is a posterior Bankart lesion, extending from 6:00 superiorly along the posterior glenoid rim. The may be easily seen as a tear, but may be subtle, and may appear to be more of a softening of the rim without an overt lesion seen on the surface. Probing of the area will reveal the underlying detachment off the glenoid and the lack of support to the labral attachment. This has been identified as the Kim lesion¹⁶ (Kim 2004). Posterior capsular deficiency, demonstrated as lack of tension in the posterior band of the inferior glenohumeral ligament, may also be present. The rotator interval is frequently lax in posterior instability, but it may be normal in traumatic posterior instability.

6b.4 Findings at arthroscopy: The same thorough evaluation should be done of all aspects of the joint prior to starting treatment. The exam under anesthesia should reveal excessive posterior translation, with the same grading scale as used for anterior instability. In the joint, the rotator interval should be inspected and probed to check for laxity, and should be grasped and tightened to see the effect on the posterior structures. The anterior ligamentous structures should be evaluated. The posterior ligaments should be palpated to check the extent of the posterior Bankart lesion, and the mobility of the tissues. The tissues should be completely mobilized back to the glenoid rim. The Kim lesion should be evaluated by probing of the posterior labrum. If a Kim lesion is identified, the intact superficial attachment should be incised and the entire lesion should be mobilized back to the normal attachment.

6c. ATRAUMATIC SHOULDER INSTABILITY

6c.1 Introduction: Although it may be debatable whether or not atraumatic instability is a true instability in the absence of a prior episode of complete joint luxation, the shoulder joint may indeed be functionally unstable. There is the absence of one time or acute trauma; there is infrequent luxation, but frequent subluxation. Pain, rather than dislocation is the more common symptom, and this occurs after repetitive rotational motions rather than a direct blow. It is commonly seen in athletes who throw or hit in an overhead position, and in

workers who do repetitive overhead arm motions under load. The term “disabled throwing shoulder” can be used to describe all the alterations that contribute to the dysfunction of the shoulder¹⁷ (Burkhart 2003).

6c.2 Definition: There is excessive humeral head translation - luxation or subluxation - that decreases function and creates symptoms, in the absence of an overt acute traumatic episode. Pain is the major presenting symptom. The pain or other symptoms are reproducible with specific motions and arm positions, and there is no generalized ligamentous laxity by clinical testing.

6c.3 Symptoms and signs: The most common symptom is pain in the joint. This usually develops over time, but also may be seen as an acute “pop”. Although pain may be localized to either the anterior or posterior joint line, complaints of pain associated with atraumatic shoulder instability are frequently identified as being diffuse within the joint. The pain may be reproduced by the assumption of specific arm positions. Typically pain is experienced at the extremes of external rotation/abduction or internal rotation/adduction and is usually worst at the moment of maximal load such as ball release in throwers. In throwers, there is decreased ability to throw with maximum velocity or accuracy, and inability to throw as many pitches as in the past. Posterior rotator cuff weakness, especially in external rotation, is common. Joint internal derangement symptoms - clicking, popping, and sliding are common. Scapular dyskinesis, with abnormal scapular position at rest (S.I.C.K. scapula)¹⁸ (Burkhart 2003) and abnormal motion upon arm elevation¹³ (Burkhart 2003), is seen frequently as an adaptive response to the excessive translation and pain. The physical exam may demonstrate excessive translation in a load and shift test, may demonstrate findings of labral pathology¹³ (Burkhart 2003), and shows minimal apprehension or other tests relating to traumatic dislocation. The anterior pain and feeling of “dead arm” with the arm in external rotation may be related to a pseudo-laxity of the anterior capsule due to the posterior superior humeral head translation due to GIRD and the superior labral lesion¹⁷ (Burkhart 2003b). GIRD - glenohumeral internal rotation deficit - is seen frequently¹⁷ (Burkhart 2003b). The scapular exam will reveal abnormalities of position and motion¹⁹ (Kibler 2003)

6c.4 Pathology: The symptoms are usually the result of a combination of alterations in one or more of the constraint systems around the joint. There may be a capsular deficiency/laxity on the anterior or posterior inferior glenohumeral ligament, a capsulolabral injury, either fraying

or small Bankart lesion, and a superior labral lesion, with loss of the biceps anchor¹⁷ (Burkhart 2003b). Accompanying any of these lesions may be an undersurface partial thickness rotator cuff injury²⁰ (Morgan 1008). The posterior capsule and posterior muscles are tight and inflexible, not allowing normal glenohumeral internal rotation.

6c.5 Findings at arthroscopy: The exam under anesthesia is usually fairly normal, with minimal anterior or posterior translation. The status of the superior labrum should be evaluated closely. There are several variations in the attachment of the biceps and labrum^{21, 22} (Snyder 1990, Vangsness 1994). Probing the biceps anchor will often show the instability of the attachment and may demonstrate undersurface injury and fraying. “Peel back” of the biceps attachment may be demonstrated by taking the arm into external rotation and abduction. This maneuver creates traction that will lift the biceps off the glenoid, and is characteristic of the superior labral lesion²⁰ (Morgan 1998). Elimination of the peel back finding and stabilization of the biceps attachment is a key surgical goal. This has several effects to stabilize the capsule and decrease translation^{23, 24} (Pagnini 1995, Pagnani 1996). The anterior and posterior inferior glenohumeral capsule areas should be evaluated in the same manner described in the sections on traumatic instability. The ligaments are commonly not detached from the glenoid. Capsular deficiency with associated laxity, however, is seen, and should be evaluated. One of the best clues of capsular deficiency is the status of the posterior band of the inferior glenohumeral ligament. If there is little tension seen in this band, then the capsule should be plicated anteriorly and/or posteriorly to restore the correct tension. The rotator cuff may have under surface tears of varying size and thickness. They should be palpated to determine the depth of the lesion and the amount of footprint that is detached. The rotator cuff may be debrided if the tear involves less than 50% of the full thickness, but should be repaired if the tear involves more than 50% of the full thickness of the tendon or of the footprint²⁵ (Weber 1999).

6d. MULTIDIRECTIONAL INSTABILITY

6d.1 Introduction: This type of instability is different from the other types. It is like traumatic instability in that dislocation is a common sign, but there is usually not a history of significant trauma to initiate the instability, and the capsule and ligaments display abnormal laxity before the symptoms are present. The instability, however, is not commonly at the end ranges of motion, where ligamentous tension is the major constraint to translation, but is usually in the mid ranges of motion, where muscular control of translation is most important. Studies have

shown that imbalance of the scapular stabilizers and the rotator cuff muscles are key components in creating the instability²⁶ (Eisenhart-Rothe von, 2005). This is why this type of instability responds better to rehabilitation than the other types. In addition, there is a voluntary component to this type that is not present in the other types.

6d.2 Definition: The acronym AMBRII was originally conceived by Thomas and Matsen⁷. AMBRII is defined as atraumatic multidirectional instability that is bilateral and often responds to rehabilitation. Should surgery be elected, an inferior capsular shift should be performed and the rotator interval be closed²⁷ (Matsen 1008). There is luxation/subluxation that decreases shoulder function. The symptoms may be present without a history of discrete trauma. There may be a voluntary component to the luxation/subluxation. The humeral head position at the time of instability will vary, being both anterior and posterior. Reduction is usually quite easy, and may not be associated with much post instability pain. The patient will exhibit signs of generalized ligamentous laxity in other joints of the body.

6d.3 Signs and symptoms: The instability is frequent, as many as 4- 5 times per day, and the reduction is usually easy, with motion of the arm and scapula. There is frequently a chronic subluxation, either in an inferior, posterior, or anterior direction, and may be associated with maintenance of shoulder use. The instability occurs in the mid ranges of motion, usually as the arm is being elevated or flexed forwardly, and is associated with alteration of scapular motion so that the scapula wings. This winging, called scapular dyskinesis, may also be seen with the arms at rest. The instability can be frequently minimized or eliminated by stabilization of the scapula in a retracted position. Muscle weakness and imbalance in the rotator cuff and the serratus anterior is common. The physical exam will reveal the signs of generalized capsular laxity, with increased range of motion of all joints, and excessive translation of multiple joints upon testing.

6d.4 Pathology: The predominant finding is generalized capsular laxity and deficiency. This may be anterior and posterior, and involves the rotator interval. With long standing instability, or following significant trauma, there may be anterior or posterior Bankart lesions.

6d.5 Findings at arthroscopy: The exam under anesthesia will show translation in all directions, with at least 2+ laxity. The sulcus sign will be positive at both 0 and 90 degrees of abduction. There may be a predominant direction of excessive translation, but the humerus

will move in all directions. The joint exam may show other pathology, including Bankart lesions, capsular fraying, or superior labral lesions. The rotator interval will be lax, and there will be minimal tension in the inferior glenohumeral ligament. The drive through sign will be positive. The capsule will be lax, and will need to be probed and tensioned to determine the exact amount of plication, the location of the plication stitches, and whether or not the capsule may need to be repaired back to the glenoid. The goal of the capsular repair is to balance the tension in the anterior and posterior bands of the inferior glenohumeral ligament to recreate the sling of the ligament under the humeral head, without limiting rotation. Therefore, the repair is performed with sutures that plicate the capsule in an inferior to superior fashion, rather than a lateral to medial plication of the capsule. Plication sutures are placed to imbricate approximately 1 cm of tissue with each suture placed. The repair is completed with 1 or 2 sutures to reduce the volume of the rotator interval.

7. GLENOID FRACTURE Hill Sachs lesion

Burkhart and De Beer classify Hill-Sachs lesions into engaging or non-engaging. An engaging fracture is one that sits with the long axis of the fracture parallel to the anterior rim of the glenoid with the shoulder in a functional position of abduction and external rotation. A non-engaging fracture is one that is not parallel or low, and will not therefore engage in normal function.

The presence of a glenoid deficit, bony Bankart lesion should be noted. This concept is important in clinical decision making with regard to the potential success of arthroscopic stabilisation.

Goss proposed a classification of glenoid fractures. There is the potential for advances in the arthroscopically assisted reduction and fixation of glenoid fractures. A classification that can be used to aid decision making in this process is required. (Goss 1995)

Type 1 Glenoid fracture, a) anterior rim, b) posterior rim

Type 2 fracture line through glenoid fossa exiting through lateral border of scapula

Type 3 through the superior border of scapula

Type 4 through the medial border of the scapula

Type 5 a) combination of 2 and 4

b) 3 and 4

c) 2, 3, and 4

Type 6 Comminuted fracture

Alternatives include classifications according to Iderberg and Euler and Ruedi, though these are more radiological than arthroscopically achievable

Classifications of Intra and extra capsular fractures

A. fractures of the body

B. fractures of the processes

C. fractures of the scapular neck

D. Articular fractures

D.1 Glenoid rim fractures

D.2 Glenoid fossa, a) inferior fragment, b) horizontal split, c) coracoglenoid bloc formation, d) comminuted fractures

E. fractures combined with humeral head

8. OSTEOARTHRITIS/ARTICULAR CARTILAGE LESIONS

8.1 Introduction: Damage to the articular cartilage may arise from inflammatory arthritis, osteoarthritis or from direct trauma.

8.2 Definition: Osteoarthritis of the glenohumeral joint may involve the two opposing surfaces to a variable extent.

8.3 Symptoms and Signs: The patient complains of variable amounts of pain of increasing pain and stiffness. Night pain is common and prevent the patient sleeping on the affected side. Worsening symptoms may cause loss of sleep. There is pain with activities of daily living such as dressing and un-dressing and overhead activity. Reaching behind is restricted. Both active and passive movement is restricted and painful. There may be associated rotator cuff signs. Diagnosis is confirmed radiologically in at least two planes.

8.4 Pathology: The standard Outerbridge classification applies to the shoulder.

Grade 1 softening and swelling of the cartilage

Grade 2 fragmentation and fissuring in an area half an inch in diameter

Grade 3 extended area beyond grade 2

Grade 4 erosion of cartilage to bone

Specific to the shoulder there may be osteophytes of the humeral head and glenoid.

8.5 Arthroscopic findings: The findings are of damage to the articular cartilage and a probe may be required to assess the extent and depth of the lesion. It should be remembered that the middle of glenoid is frequently devoid of articular cartilage as a normal finding. There is also a bare area between rotator cuff and edge of the articular cartilage of the humeral head.

The site of the changes to the articular cartilage follows the aetiology. Anterior osteoarthritis may follow repeated episodes of dislocation or excessive tightening of the shoulder following surgical stabilisation. Examine of range of motion under anaesthetic is important in surgical the decision making process. The long head of biceps may rupture leaving an intra-articular portion that catches between the humeral head and glenoid abrading the adjacent articular cartilage. Similarly the prolapsed long head of biceps can get caught between the two articular surfaces. There may be florid synovitis.

Rotator cuff arthropathy (Milwaukee shoulder) includes acetabularisation of the undersurface of the acromion where curved bare bone is seen arthroscopically.

9. LOOSE BODIES

9.1-2 Introduction /Definition: A loose body is an unattached piece of tissue arising from structures within or surrounding the joint. This definition can be extended to include an abnormal piece of tissue on a pedicle.

9.3 Symptoms and signs: The loose body may cause clicking, catching, pain or a sensation of instability. Occasionally it may be palpable over the anterior joint line or bicipital groove.

9.4 Pathology: The loose body may arise from the anterior glenoid following fracture dislocation or from the impaction Hill-Sachs or reverse Hill-Sachs fracture. Synovial chondromatosis will give rise to multiple small loose bodies. There is no classification of loose bodies in the shoulder joint.

9.5 Arthroscopic findings: Loose bodies may be found in the inferior recess, the rotator interval, or the bicipital groove where it may not be visible arthroscopically. A loose body may have associated synovitis and damage to the articular cartilage.

10. SYNOVITIS/CAPSULITIS/FROZEN SHOULDER

10.1 Introduction: Frozen shoulder presents with a restriction of the shoulder with extreme pain. There are a number associated of regional pain syndromes which may co-incide with or mimic frozen shoulder.

10.2 Definition: Zuckerman and Cuomo defined the condition as one of uncertain aetiology characterised by substantial restriction of both active and passive movement in the shoulder occurring in the absence of a known intrinsic disorder of the shoulder. (Zuckerman ??)
Frozen shoulder may be primary or as a result of trauma, especially surgery.

10.3 Symptoms and signs: Frozen shoulder has a spectrum of severity and variety of presentations. Pain is the predominant initial complaint, with loss of sleep at night, and a painful restriction of range of motion. Activities such as dressing, reaching overhead or behind are all painful. Pain tends to last around 6 months followed by a phase where pain subsides and stiffness becomes more prominent. The natural history is of frozen shoulder is of spontaneous relief. There may be associated Dupuytren's contractures. Insulin dependent diabetes tends to be associated with more severe symptoms which follows a prolonged time course and may not resolve in the normal fashion.

Passive range of motion is compared with the contra-lateral shoulder with the scapula stabilised. External and internal rotation are the first to be restricted and the last to recover.

10.4 Pathology: The obvious pathological changes are of a global capsular contracture, including the coracohumeral ligament. Microscopically, both inflammatory and fibrotic processes have been described. The specific cellular basis for the pathology seen in frozen shoulder has yet to be identified. Lundberg showed that there was an increase in glycosaminoglycans and a decrease in glycoproteins, specifically hyaluronic acid. Rodeo et al suggested that transforming growth factor beta and platelet-derived growth factor may be involved and act as a continual stimulus in the inflammatory and fibrotic process. Bunker et al examined the expression of growth factors, cytokines and matrix metalloproteinases (MMPs) in patients with frozen shoulder and compared this with Dupuytren's tissue and a control group. They recorded an imbalance between aggressive healing, scarring, contracture and a failure of remodelling which may lead to the protracted course of the disease. Immunological studies have failed to identify successfully a link with HLA-B27 or any specific autoimmune or arthritic process.

10.5 Arthroscopic findings: The glenohumeral joint capsule is very thickened and tight. The joint space has a markedly reduced volume. The arthroscope is inserted using the standard posterior portal. Entry into the joint can be difficult with a considerable increase in force required to gain access. Once entered manoeuvrability is tricky and the joint surfaces may be inadvertently damaged. The diagnosis is confirmed by the appearance of angiogenesis, many new blood vessels budding and spreading over the synovium, with a villous synovitis in the rotator interval, petechial haemorrhages and a thickened, scarred capsule.

The superior part of the joint is inspected, but at this stage entry into the inferior part of the joint will be impossible as the joint is just too contracted. A second portal is introduced by means of an inside out technique. Extra care is required to avoid damage to the labrum and articular surfaces. The rotator interval is covered by a sheet of dense contracted tissue. The long head of biceps is clear of contracture, but subscapularis is enclosed within the sheet of capsule, referred to by Bunker as an involucreum.

More mature frozen shoulder loses the intense vascularity, the capsule appears white and scarred.

11. ACROMIOCLAVICULAR JOINT

11.1-2 Introduction Definition: The acromioclavicular joint may be symptomatic as a result of trauma and/or degenerative changes. The pathology leads to pain arising from the acromioclavicular joint.

11.3 Symptoms and signs: Pain which may be localised to the area of the acromioclavicular joint. Pain may be referred along the clavicle or down the anterior upper arm. The acromioclavicular joint is tender to direct palpation and ballottement. Pain is reproduced by full cross arm adduction and over the last 20 degrees of abduction. Pain from diagnostic tests should be diminished by intra-articular injection of local anaesthetic. It may be advisable to undertake this with ultrasound scan guidance, and note that the joint will only accept up to 2 mls of local anaesthetic.

11.4 Pathology:

A. Dislocation: Rockwood (Rockwood 1996) added to the existing classification of Tossy. Types 1 and 2 remain the same but the classification of 'complete' ruptures is expanded to aid in management decision making, with surgery recommended for types 4-6.

Type 1 Sprain of the AC ligament

Type 2 AC joint disrupted with some vertical separation

Type 3 Complete disruption of the AC CA lig with the shoulder complex displaced inferiorly

Variants include pseudo dislocations through a periosteal sleeve, physeal injury, and coracoid process fracture

Type 4 Ligament injury as type 3 but with the clavicle displaced posteriorly into or through the trapezius muscle

Type 5 Gross disruption of the clavicle with loss of soft tissue attachments. The clavicle is palpable subcutaneously

Type 6 The clavicle is displaced inferiorly below the coracoid process

B. Osteoarthritis: I am not of an arthroscopic classification of AC joint osteoarthritis.

11.5 Arthroscopic findings Inferior osteophytes of both sides of the acromio-clavicular joint protrude into the subacromial space. Synovitis may be seen with thickening of the capsule. The capsule is thickened compared with a normal joint. There may be underlying subacromial bursitis and abrasion on the upper surface of the rotator cuff.

12 SEPTIC ARTHRITIS

12.1 Introduction: Infection of a joint by pathological organisms.

12.2 Definition: Infection of the joint and the surrounding tissues

12.3 Symptoms and signs: Patients complain of pain, particularly with the slightest movement of the joint. There may be few other signs, though more advanced cases will demonstrate swelling and redness if peri-articular tissues become involved. The joint is held in a position of greatest intra-articular volume. Passive movement of more than a few degrees is painful in any plane.

12.4 Pathology: Patients may possess a normal immune system or may be immunocompromised. The compromise may be systemic or local secondary to recent surgery, radiotherapy, poor blood supply. There may be local peri-articular joint involvement and associated osteomyelitis.

12.5 Arthroscopic findings: Stutz and Gachter (Stutz 2000) have divided the arthroscopic findings in septic arthritis into four stages. 10 of the 78 patients had involvement of the shoulder

Stage 1 opacity of fluid, redness of the synovial membrane, possible petechial bleeding, no radiological changes

Stage 2 severe inflammation, fibrinous deposition, pus, no radiological changes

Stage 3 thickening of the synovial membrane, formation of loculated compartments within the joint, appearances like a sponge. No radiological changes

Stage 4 aggressive pannus with infiltration of the cartilage, possible undermining of the cartilage radiological signs of subchondral osteolysis erosions and cysts.

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ANKLE

1. Introduction

The key point in the assessment of ankle pathology is the clinical diagnosis. By means of a clinical diagnosis, an indication is set for an arthroscopic intervention. The clinical diagnosis is based on history, symptoms, signs and standard radiographs. In this chapter, eight different clinical diagnoses of the ankle joint pathology are described. Of each clinical diagnosis a definition is given followed by a brief description of symptoms and signs, as well as the underlying process. The outcome measures, which are thought to be relevant for the diagnosis are discussed. Finally, the arthroscopic findings are presented typically belonging to the clinical picture. Concerning the typical arthroscopic findings the definition is given, followed by a classification system of this abnormality.

Clinical diagnosis and findings at arthroscopy: definitions and classifications

Intraarticular pathology is assessed in the following manner:

Clinical diagnosis	Arthroscopy
2. Asymptomatic ankle	Typically one expects to find normal anatomy. Asymptomatic lesions might be present such as; soft tissue lesions, plicae, adhesions, ossicles, spurs, chondral lesions, synovitis.
3. Anterior ankle impingement (bony/soft tissue)	Osteophytes, ossicles, soft tissue impediments, synovitis, scar tissue, plicae, congenital bands, posttraumatic soft tissue lesions
4. Synovitis	Posttraumatic synovitis, soft tissue impediments, arthritis, pigmented villonodular synovitis, charcot joint, arthrofibrosis
5. (Osteo)chondral defect	(Osteo-) chondral lesions, intraosseous ganglion, subchondral cyst
6. Loose body	Loose bodies, posttraumatic calcifications, avulsion fragments, ossicles, broken osteophytes
7. Arthrosis	Osteoarthrosis, chondromalacia, osteophytes, chronic synovitis, subchondral cysts, joint space narrowing

8. Posterior ankle impingement	Os trigonum, tendonitis, posttraumatic calcifications, avulsion fragments, loose body, fracture of the posterior process, (osteo-) chondral defects
9. Instability	Ankle joint laxity, synovitis, (osteo-) chondral lesions, spurs

2. ASYMPTOMATIC ANKLE

2.1 Definition: A patient is asymptomatic when there are no complaints and when provocative tests at physical examination are negative, regardless the fact whether any pathological findings are present in the ankle joint.

2.2 Symptoms and signs: There may be a history of intermittent or transient complaints, however there are no current symptoms. Physical examination may be normal or reveal slight abnormal range of joint motion or a positive anterior drawer test. Radiographic examination may show a normal ankle or osteophytes, intraosseous cysts or even mild osteoarthritis.

2.3 Pathology: A variety of pathological entities can remain asymptomatic. Symptoms may develop when, for example, osteophytes impinge on each other or when a reactive inflammatory response of the synovia or the joint capsule occurs. Any trivial trauma, as for example a simple sprain, can be the cause of the sudden onset of complaints.

2.4 Arthroscopy: In the asymptomatic ankle, the medial malleolar-talar articulation and the central portion of the ankle on the anterior gutter are usually normal. When present, pathology is generally limited to the syndesmosis and the lateral gutter. Patients may have a mild synovitis surrounding the anterior inferior tibiofibular ligament, both in front and behind, as well as synovitis of the anterior talofibular ligament. Fibrosis of the lateral gutter or chondromalacia of the talus and the fibula may be present. Rarely, an adhesive thick scar band, also known as a meniscoid lesion, is present, extending from the anterolateral aspect of the distal tibia to the lateral gutter. Cheng, in 1998, reported asymptomatic ankle spurs in 45% of football players and 59% of dancers.¹ Asymptomatic ankles can become symptomatic after a significant traumatic event.² In these cases, synovial and scar tissue act as painful anterior impediment.

Classification of normal anatomy

The intraarticular anatomy of the ankle as seen during arthroscopic examination has been described extensively.³ The ankle joint can be divided into anterior and posterior cavities, each of which can then be subdivided further into three compartments for methodological inspection of the ankle joint.⁴ Ferkel developed a 21-point systematic examination (table I) of the anterior, central and posterior ankle joint to increase the accuracy and reproducibility of the arthroscopic examination.^{5,6} Van Dijk reported on a new two-portal approach with the patient in the prone position, specifically for close visualization of the posterior compartment of the ankle and subtalar joint.⁷ He developed a 14-point systematic examination for the hindfoot and posterior ankle joint (table II).

Table I. The 21-point arthroscopic examination of the ankle (Ferkel and Fischer 1989)

Anterior:

- Deltoid ligament
- Medial gutter
- Medial talus
- Central talus and overhang
- Lateral talus
- Trifurcation of the talus, tibia, and fibula
- Lateral gutter
- Anterior gutter

Central:

- Medial tibia and talus
- Central tibia and talus
- Lateral tibiofibular or talofibular articulation
- Posterior inferior tibiofibular ligament
- Transverse ligament
- Reflection of the flexor hallucis longus

Posterior:

- Posteromedial gutter
 - Posteromedial talus
 - Posterocentral talus
 - Posterolateral talus
 - Posterior talofibular articulation
 - Posterolateral gutter
 - Posterior gutter
-

Table II. The 14-point hindfoot endoscopic examination (van Dijk 2000).

1. Lateral talocalcaneal articulation
2. Flexor hallucis longus retinaculum
3. Flexor hallucis longus tendon
4. Posterior talar process
5. Posterior talofibular ligament
6. Posterior tibiofibular ligament
7. Transverse tibiofibular ligament
8. Tip of the medial malleolus/medial malleolus
9. Posteromedial gutter
10. Posteromedial talus/tibia
11. Posterocentral talus/tibia
12. Posterolateral talus/tibia
13. Posterolateral gutter
14. Tip of lateral malleolus

Additional (when indicated):

Posterior tibial tendon
Flexor digitorum tendon
Peroneal tendons

3. ANTERIOR ANKLE IMPINGEMENT

3.1 Definition: Anterior ankle impingement syndrome is a clinical diagnosis which is characterised by anterior ankle pain with recognisable pain and a (slightly) limited dorsiflexion.

3.2 Symptoms and signs: Clinically, the patient complains of anterior joint pain which becomes worse after activity, walking up stairs or hills, squatting or running is especially bothersome. Pain on palpation is localized over the anterior or anteromedial aspect of the ankle joint is the most frequent symptom. Forced ankle dorsiflexion can sometimes provoke the complaints, but in most patients this test is negative. Plain radiographs may demonstrate anterior and posterior osteophytes. Additional oblique views can be helpful to demonstrate antero- and posteromedially located osteophytes. In case of anterior ankle pain with negative radiographic findings, the most likely cause of the complaints is an anterior soft tissue impediment. Congenital plicae within the ankle, posttraumatic scar tissue, adhesions or ganglions may all act as local soft tissue impediment with local swelling and circumscript pain on palpation. Relevant outcome measures for anterior ankle impingement are:

1. Pain
2. Ankle joint function
3. Giving-way

There are no specifically-designed subjective scorings systems available to assess anterior ankle impingement.

3.3 Pathology: Objective scoring systems for anterior impingement use the location (tibia or talus) and size of osteophytes as prognostic factors for postoperative success.^{8,9} Scranton and McDermott compared open resection with arthroscopic resection of painful anterior impingement spurs and developed a validated score to assess osteophytes on ankle radiographs.⁸ They categorized ankle spurs between grade 1 through 4, according to the size of spurs and degree of involvement of the ankle, and demonstrated that the treatment and recovery correlated with the grade (Table II). Grades 1, 2 and 3 spurs could be resected arthroscopically or by arthrotomy. Grade 4 spurs initially were not thought to be appropriate for arthroscopic resection. However, as experience has increased, grade 4 spurs can also be resected using great care and patience not to injure the surrounding neurovascular structures. The reproducibility of this classification system may be doubtful because the correlation was

assessed with the subjective outcome at short-term follow-up only (10 weeks postoperatively).

It has been determined that the degree of osteoarthritic changes influences the outcome of treatment.² Osteophytes without joint space narrowing are not a manifestation of osteoarthritis and subsequently a “normal” joint remains after removal of these spurs. A validated objective scoring system for anterior ankle impingement based on the degree of degeneration on the radiographs was developed by van Dijk et al. (Table III). The results at long-term follow-up show that the use of this osteoarthritic classification proved to be more discriminative than the impingement classification of Scranton and McDermott as a predicting factor for the outcome of arthroscopic surgery when dealing with anterior ankle impingement.¹⁰

3.4 Arthroscopy: The most common locations for soft-tissue impingement are the following: Anterolateral impingement; several studies report on patients with persistent pain and swelling over the anterolateral aspect of the ankle after an inversion sprain.^{14,15} Arthrotomy of these ankles reveals hyalinized connective tissue extending into the joint from the anterior inferior portion of the talofibular ligament. This is called a ‘meniscoid’ lesion. Patients generally have a synovitis surrounding the anterior inferior tibiofibular ligament, both in front and behind, as well as synovitis of the anterior talofibular ligament. In addition, a small ossicle or loose body may be hidden in the soft tissues at the tip of the fibula. Rarely, an adhesive thick scar band, previously described as a meniscoid lesion, is present, extending from the anterolateral aspect of the distal tibia to the lateral gutter. After surgical reconstruction of the lateral ankle ligaments, soft tissue impingement may be present between the reconstructed ligaments and the talus.

Syndesmotic impingement; at arthroscopy, the inflamed synovium envelops the anterior inferior tibiofibular ligament as well as the inferior articulation of the tibia and fibula. In addition, synovial nodules are frequently seen in this area. The synovitis involves the anterior and also the posterior aspects of the syndesmotic ligament, and sometimes this ligament is torn or frayed.

Anterior and anteromedial impingement: In case of anterior and anteromedial located osteophytes, an additional soft tissue impediment is most often present. During dorsiflexion

hypertrophic synovial tissue impinges between the osteophytes. Tol et al. concluded that the cause of pain is not the osteophyte itself but that a soft tissue impingement occurs between the osteophytes.¹⁰ It can be hypothesised that removal of the soft tissue impediment without removal of spurs would be sufficient. The presence of talar and tibial osteophytes reduces the anterior joint space however. After an arthroscopic intervention a haematoma will be formed postoperatively, which will subsequently develop into scar tissue. The scar tissue that fills the defect will act as a new anterior soft tissue impediment. It is therefore important to remove the osteophytes to enhance more anterior and anteromedial space and diminish the chance for a recurrence of symptoms.¹⁶

Visualization of the anterior ankle joint can be improved by bringing the ankle in a forced dorsiflexion position since in this position the anterior working are ‘opens up.’ Distraction makes the anterior capsule more tense over the osteophyte. It is important to identify the anterior and superior borders of the osteophyte, and this often requires careful elevation or peeling off soft tissues from the margins of the osteophyte.

Available classification systems for anterior ankle impingement

Subjective: none available

Objective: see table II and III (below)

Table II. Radiographic classification of anterior ankle impingement (Scranton and McDermott 1992)

Type	Characteristics
I	Synovial impingement; radiographs show inflammatory reaction, up to 3-mm spur formation
II	Osteochondral reaction exostosis; radiographs manifest osseous spur formation greater than 3 mm in size. No talar spur is present
III	Significant exostosis with or without fragmentation, with secondary spur formation on the dorsum of the talus seen. Often with fragmentation osteophytes
IV	Pantaloctrural arthritic destruction; radiographs suggest medial, lateral, or posterior degenerative, arthritic changes

Table III. Radiographic classification of anterior impingement lesions (van Dijk et al. 1997, 2000)

Grade	Characteristics
0	Normal joint
I	Anterior osteophytes without joint space narrowing
II	Anterior osteophytes with joint space narrowing
III	Anterior osteophytes with total disappearance or deformation of the joint space

4. SYNOVITIS

4.1 Definition: Synovitis can be a noninflammatory, inflammatory or septic process of the synovium, which is most characterised by joint swelling and tenderness. Synovitis can be caused by trauma or previous surgery. A generalized or localized synovitis can occur, most often with fibrous bands and adhesions. Synovitis accounts for approximately 30% of pathology seen in the ankle joint.

4.2 Symptoms and signs: Patients usually have complaints of aching, swelling, tenderness, and other signs of joint inflammation. A history of trauma or injury is more likely to cause a nonspecific type of synovitis, either localized or generalized; however, trauma can also trigger an underlying specific pathologic process. Radiographs are often negative. The clinical manifestation of soft tissue lesions can be divided into:

- A. impingement, recognition of local pain and swelling
- B. diffuse pain, swelling, calor, restriction of range of ankle motion in all directions
- C. pain deep in the ankle, no recognition of local swelling or restriction of range of ankle motion
- D. absence of symptoms

Relevant outcome measures for synovitis are:

- 1. Pain
- 2. Ankle joint function
- 3. Activity level
- 4. Swelling

A well-known clinical scoring system which has been specifically designed for the evaluation of synovitis is the DAS-28 score (table IV). This system is developed for patients with synovitis due to rheumatoid arthritis. Although not specifically developed for the ankle, this system can be helpful to assess and monitor synovitis in patients with rheumatoid arthritis.

4.3 Pathology: Synovitis of the ankle may be a difficult diagnostic problem. Even after careful history, physical examination, and diagnostic testing, the diagnosis may not be readily apparent. MR imaging has been proven to be sensitive for the evaluation of synovitis of the

ankle associated with trauma, whereas it is specific for soft tissue impingement evaluation. Therefore most objective scoring systems designed for synovitis concern MRI. A well-known assessment tool is the MRI grading system of Huh et al. which describes four grades of synovial enhancement (table V). This system is well validated since it was correlated with arthroscopic findings. Another objective scoring system for synovitis has been developed specifically for histopathologic assessment. This validated system was developed by Krenn et al. (table VI). Three features of chronic synovitis (enlargement of lining cell layer, cellular density of synovial stroma, leukocytic infiltrate) are evaluated (from 0, absent to 3, strong) and each feature is graded separately. The sum provides the synovitis score, which is interpreted as follows: 0-1, no synovitis; 2-4, low-grade synovitis; 5-9, high-grade synovitis.

4.4 Arthroscopy: Localized or generalized inflammation of the synovia can be present. It may contain hemosiderin or fibrin debris. Scarring, fibrosis and adhesions are often seen in relation with the synovitis. Ferkel, in 1997, proposed a arthroscopic classification system for synovial disorders (table VII).¹⁷ In 1985 Gächter developed a staging system for septic arthritis (table VIII). It is a combined system of radiological and intraoperative findings. The system has been validated by a study of Stutz et al. in 2000. He found that stages I through III could be managed by arthroscopic treatment.

Available classification systems for synovitis

Subjective: DAS-28 (table IV)

Objective: MRI grading system by Huh et al. (table V), histopathologic system by Krenn et al. (table VI), Arthroscopic system by Ferkel (table VII), Staging system for septic arthritis developed by Gächter (Table VIII).

Table IV. DAS-28 system

DAS28 form

	Swollen	tender	Swollen	Tender
Shoulder				
Elbow				
Wrist				
MCP 1				
2				
3				
4				
5				
PIP 1				
2				
3				
4				
5				
Knee				
Subtotal				
Total	Swollen		Tender	

No disease activity

high disease activity

Swollen (0-28)

Tender (0-28)

ESR

VAS disease activity (0-100mm)

$$\text{DAS28} = 0.56*\sqrt{(t28)} + 0.28*\sqrt{(sw28)} + 0.70*\text{Ln}(\text{ESR}) + 0.014*\text{VAS}$$

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Table V. MRI grading system by Huh et al.

The ankle joint is divided into four compartments: the anterolateral gutter, anteromedial gutter, anterior recess and posterior recess.

Grade 1	No synovial enhancement
Grade 2	Linear enhancement
Grade 3	Focal nodular enhancement (can be soft tissue impingement)
Grade 4	Irregular nodular enhancement (can be soft tissue impingement)

Table VI. Histopathologic assessment system by Krenn et al.

Three features of chronic synovitis:

1. enlargement of lining cell layer
2. cellular density of synovial stroma
3. leukocytic infiltrate

are semiquantitatively evaluated (from 0, absent to 3, strong) and each feature is graded separately. The sum provides the synovitis score, which is interpreted as follows: 0-1, no synovitis; 2-4, low-grade synovitis; 5-9, high-grade synovitis.

Table VII, Arthroscopic assessment system by Ferkel et al.

- Congenital: plicae or congenital bands within the ankle; plicae, or shelves, have been demonstrated in the knee, but are difficult to find in the ankle. Congenital bands are seen as an incidental finding when examining the ankle for other types of pathology.
- Traumatic: sprains, fractures, and previous surgery;
- Rheumatic: rheumatoid arthritis, pigmented villonodular synovitis, crystal synovitis, hemophilia, and synovial chondromatosis
- Infectious: bacterial and fungal
- Degenerative: primary and secondary
- Neuropathic: Charcot joint
- Miscellaneous: ganglions, arthrofibrosis

Table VIII. Septic arthritis staged according to the criteria of Gächter (1985).

- Stage I: opacity of fluid, redness of the synovial membrane, possible petechial bleeding, no radiological alterations
- Stage II: severe inflammation, fibrinous deposition, pus, no radiological alterations
- Stage III: thickening of the synovial membrane, compartment formation ("sponge-like" arthroscopic view, especially in the suprapatellar pouch), no radiological alterations
- Stage IV: aggressive pannus with infiltration of the cartilage, possibly undermining the cartilage, radiological signs of subchondral osteolysis, possible osseous erosions and cysts.

5. OSTEOCHONDRAL DEFECT

5.1 Definition: An osteochondral defect is a separation or an inflammatory lesion of cartilage and bone causing deep ankle pain. Osteochondral lesions of the ankle joint include many pathologic entities, including osteochondritis dissecans, chondral and osteochondral loose bodies, osteophytes, chondral and osteochondral fractures of the tibia and talus, cystic lesions of the talus, fracture defects, and arthritis. Many controversies and misconceptions persist in terms of the etiology, treatment, and prognosis of osteochondral and chondral lesions of the ankle. Multiple terms are used to describe these lesions, including transchondral fractures, osteochondral fractures, osteochondritis dissecans, talar dome fractures, and flake fractures. According to Ferkel, the best term to describe this problem is osteochondral lesion.²⁰ The controversy in terminology has resulted in part from a lack of a clearly defined and universally accepted etiology.

5.2 Symptoms and signs: The presentation of osteochondral lesions can be acute after an injury, but more often it is associated with persistent ankle pain, particularly after a trauma such as inversion injury of the lateral ligamentous complex. A history of chronic lateral ankle pain is commonly noted. In addition, a history of associated injuries such as ankle or lower extremity fractures or falls from a height may be elicited. Usually, symptoms are intermittent and can include stiffness and pain of a deep aching nature aggravated by weight bearing. Swelling, catching, clicking, locking and less commonly giving-way can be present. On physical examination, pain on palpation is usually absent. Some patients have a limited painful range of motion, crepitus sometimes can be present. Different studies suggest that the average duration of symptoms before a definitive diagnosis is made ranges from 4 months to 2 years. Relevant outcome measures for osteochondral lesions are:

1. Pain
2. Giving-way
3. Ankle joint function

Plain radiographs can be negative. A heel-rise view can be helpful to detect an osteochondral lesion. CT scan and MRI can be helpful to establish the diagnosis and aid in preoperative planning.

5.3 Pathology: Osteochondral lesions on the medial aspect of the dome of the talus occur in the middle or posterior third; lateral lesions occur primarily in the middle or anterior portion.

Lateral lesions are usually shell- and wafer-shaped and are often displaced and elevated by the levering effect of the distal tibia. Medial lesions are deeper and cup-shaped and are usually not displaced.

Berndt and Harty's 1959 radiologic classification was based on plain radiographs of the ankle (Table IX).¹⁸ This classification was revised by Loomer et al. (Table X).¹⁹ More recently, Ferkel and Sgaglione have developed a CT classification, which is considered to be more accurate and to correlate better with the arthroscopic picture and with subsequent results (Table XI).²⁰ A MRI classification system was developed by Anderson et al. (table XII).

5.4 Arthroscopy: Pritsch and associates classified osteochondral lesions by the arthroscopic appearance of overlying cartilage into three grades (Table XIII): intact, firm, shiny articular cartilage; intact but soft cartilage; and frayed cartilage.²¹ They found some lesions which progressed from grade 1 to grade 3 during the course of treatment. They also noted a poor correlation between the radiographic appearance and arthroscopic findings, and considered the arthroscopic appearance to be the most important determinant for treatment. Cheng, Ferkel and Applegate developed an arthroscopic staging system (Table XIV) which correlated well with the CT classification of Ferkel and Sgaglione (1994) and the.^{20,23} Recently, a new arthroscopic staging system was developed by Taranow et al. who classified cartilage as viable and intact (stage A) or breached and nonviable (stage B) (table XV). The bone component was determined as follows: 1) stage 1 is a subchondral compression or bone bruise; 2) stage 2 lesions are subchondral cysts and are not seen acutely (these develop from stage 1 lesions); 3) stage 3 lesions are partially separated or detached fragments in situ; and 4) stage 4 represents displaced fragments. The condition of the cartilage and bone together determines the type of surgical treatment.²⁴ The Outerbridge and ICRS classification were originally designed for the knee but can be used for the ankle as well (table XVI). The Outerbridge classification is the most widely used system to describe the size of a cartilage lesion. It provides a distinction between a partial (grades 1 and 2) vs. nearly-full or full thickness cartilage defect (grades 3 and 4); between a small (grade 2) and larger (grade 3) lesion; and describes a complete loss of cartilage (grade 4). The Outerbridge classification has, however, specific limitations. For example, a large partial thickness defect with a potentially bad prognosis due to its size is classified as a grade 1 defect, whereas a direct cut or narrow fissure is classified as a grade 4 defect. These limitations have prompted the International Cartilage Repair Society (ICRS) to introduce a modified classification system that focuses on the depth of the cartilage injury. Combined with visual measurement, the

modified ICRS classification has the potential to better describe the defect macroscopically and correlates better with the clinical outcome.

Despite the existence of these classification systems, few authors base their decision for a specific treatment on these systems. A meta-analysis of Tol et al. showed that the value of preoperative radiological staging systems was of minor value in the preoperative planning since they hardly correlate with the peroperative findings. This demonstrates the shortcomings of preoperative radiological staging systems as guide for the treatment strategy.²⁵ Peroperative staging of osteochondral defects, therefore, appears more appropriate. Eventually, the most rational way of preoperative assessment of osteochondral lesions is to determine whether they are symptomatic or asymptomatic. Symptomatic lesions need surgical treatment; e.g. debridement and drilling.²⁶

Available classification systems for osteochondral defects

Subjective: none

Objective: Berndt and Harty's radiographic classification (table IX), radiographic classification by Loomer et al. (table X), CT classification by Ferkel and Sgaglione (table XI), MRI classification by Anderson et al. (table XII), Arthroscopic classification by Pritsch et al. (table XIII), arthroscopic staging system developed by Cheng et al. (table XIV), arthroscopic staging system developed by Taranow et al. (table XV), the Outerbridge classification system of cartilage lesions (table XVI), the ICRS OCD and hyaline cartilage lesion classification system (table XVII and table XVIII).

Table IX. Berndt and Harty radiographic classification of osteochondral lesions

Stage	Characteristics
1	A compression fracture of subchondral bone
2	A partial osteochondral fragment
3	A completely fractured osteochondral fragment without displacement
4	A completely fractured osteochondral fragment with displacement into the joint

Table X.1 Revised Berndt and Harty radiographic classification of osteochondral lesions by Loomer et al.

Stage	Characteristics
1	Compression of subchondral bone
2	Partially fractured but undisplaced
3	Completely fractured but undisplaced
4	Displaced fracture
5	Radiolucent (fibrous defect)

Table XI. CT classification of osteochondral lesions by Ferkel and Sgaglione (1994).

Stage	Characteristics
1	Cystic lesion with dome of talus, intact roof on all views
2A	Cystic lesion with communication to talar dome surface
2B	Open articular surface lesion with overlying nondisplaced fragment
3	Undisplaced lesion with lucency
4	Displaced fragment

Table XII. MRI classification of osteochondral lesions by Anderson et al. (1989).

Stage	Characteristics
1	Subchondral trabecular compression. Plain radiograph normal, positive bone scan, marrow edema on MRI
2	Incomplete separation of fragment
2A	Formation of subchondral cyst
3	Unattached, undisplaced fragment with presence of synovial fluid around ligament
4	Displaced fragment

Table XIII. Arthroscopic classification by Pritsch et al. (1986).

Stage	Characteristics
1	Intact, firm, shiny, articular cartilage
2	Intact but soft cartilage
3	Frayed cartilage

Table XIV. Arthroscopic classification by Cheng and Ferkel (1995).

Stage	Characteristics
A	Smooth, intact but soft or ballotable
B	Rough surface
C	Fibrillations/fissures
D	Flap present or bone exposed
E	Loose, undisplaced fragment
F	Displaced fragment

Table XV. Arthroscopic staging by Taranow et al. (1999).

Cartilage:	A: viable and intact B: breached and nonviable
Bone:	Stage 1: subchondral compression or bone bruise Stage 2: subchondral cysts Stage 3: partially separated or detached fragments in situ Stage 4: displaced fragments

Table XVI. The Outerbridge classification (1961).

Grade 0	Normal cartilage
grade I	cartilage with softening and swelling
grade II	a partial-thickness defect with fissures on the surface that do not reach subchondral bone or exceed 1.5 cm in diameter
grade III	fissuring to the level of subchondral bone in an area with a diameter more than 1.5 cm
grade IV	exposed subchondral bone

Table XVII. ICRS OCD classification system (1998).

OCD I	Stable lesion with a continuous but softened area covered by intact cartilage
OCD II	Lesion with partial discontinuity that is stable when probed
OCD III	Lesion with a complete discontinuity that is not yet dislocated (“dead in situ”)
OCD IV	Empty defect or defect with dislocated fragment or loose fragment within the bed
Subgroups I – IVB	Defects that are > 10 mm in depth

Table XVIII. ICRS hyaline cartilage lesion classification system (2000).

Grade 1	Superficial lesions, cracks, and indentations
Grade 2	Fraying, lesions extending down to <50% of cartilage depth
Grade 3	Partial loss of cartilage thickness, cartilage defects extending down >50% of cartilage depth as well as down to calcified layer
Grade 4	Complete loss of cartilage thickness, bone only

6. LOOSE BODY

6.1 Definition: A loose body floats within the ankle joint or is capsulated in scar tissue. The loose body can be bony, chondral or osteochondral. It arises from osteophytes or defects in the talus or tibia.

Ossicles, broken osteophytes, chondral or osteochondral fragments arising from defects in the talus or tibia can all be considered as loose bodies in the ankle joint.²⁷ Sometimes, such a loose body is attached with scar tissue to the capsule or other structures and is then called a ‘corpus liberum pendulans’.

6.2 Symptoms and signs: A small loose body may cause catching symptoms with joint motion along with pain, swelling, and limitation of motion. Symptoms of internal derangement may resolve if a small loose body becomes fixed to the synovial lining, ceasing to cause joint irritation. A loose body may grow by proliferation of chondroblasts/osteoblasts or may shrink due to the action of chondroblasts/osteoclasts.

The physical examination may not be very revealing, with vague areas of tenderness, possible limitation of motion and catching. Rarely a loose body is palpable. As with all ankle problems, a careful physical examination must rule out extraarticular pathology, which can cause symptoms similar to intraarticular lesions. Peroneal subluxation, posterior tibial tendon attrition or rupture, tarsal tunnel syndrome, sinus tarsi syndrome, stress fracture, and tendinitis must be carefully excluded by both physical examination and ancillary studies. There are no subjective scoring system available, which are specifically designed for intraarticular loose bodies.

Clinical relevant outcome measures for loose bodies are:

1. Locking
2. Instability
3. Pain
4. Activity Level

Plain radiographs usually reveal an osseous loose body, but chondral loose bodies are not visible on routine studies. A CT can be helpful to disclose a loose body. Bone scans are rarely informative, MRI is capable of showing chondral lesions not seen on other types of studies. The plain radiographs, CT scan, or MRI scan should be scrutinized to discover the origin of the loose body, such as a defect of the talar dome, tibial plafond, or osteophyte.

6.3 Pathology: There are no standardized classification systems available for the radiological or arthroscopic assessment of loose bodies. Often, they may result from major trauma to the ankle joint, or from a relatively innocent injury such as a lateral ligament sprain. In either case, an unsuspected chondral or osteochondral lesion may occur and results in a loose body floating within the joint. There are six important parameters with regard to the radiological and arthroscopic assessment of loose bodies:

1. Number: Single or multiple
2. Attachment: (semi) –attached or loose
3. Location: intra- or extraarticular and intra- or extracapsular, or is it embedded in the cartilage layer?
4. Aspect: bony or chondral, is the fragment secondary to damage in the joint or is it a calcification?
5. Size: small fragments cause complaints of locking since they are able to travel through the joint whereas large fragments cause impingement
6. Synovium: Are the loose bodies produced by a synovial disorder?

Lesions, which appear on routine radiographs to be loose bodies may actually be intraarticular, intracapsular, or extracapsular in location, particularly in the posterior ankle joint compartment. The location of the lesions should be determined preoperatively to avoid the embarrassment of performing an arthroscopic examination for loose body for removal, only to find the joint free of any abnormality. A CT or MRI study is best suited to make the distinction between an intraarticular versus an extraarticular or intracapsular abnormality.

6.4 Arthroscopy: The arthroscopic approach to loose bodies is straight forward. Loose bodies localized to the anterior compartment, particularly in patients with ligamentous laxity, can be approached with a routine set-up using anteromedial and anterolateral portals. However, the posterior joint should also be examined for the presence of loose bodies, which can hide in the posterior recess of the joint. A posterolateral portal can be made. A posterior ankle joint approach has been described using two portals in a prone position.⁷

Available classification systems of loose bodies

Subjective: none

Objective: none

7. OSTEOARTHRITIS

7.1 Definition: Osteoarthritis is a degenerative joint disease characterized by sclerosis, osteophytes chondromalacia, subchondral cyst formation and subsequent narrowing of the joint space. Arthroscopic treatment of osteoarthritis of the ankle was initially considered effective, but has recently generated much disappointment. With proper selection of patients a few indications can be mentioned however.

7.2 Symptoms and signs: Osteoarthritis may be symptomless, but severe osteoarthritis of the ankle joint consist of symptoms of chronic pain and stiffness with limitation of range of ankle motion. There is diffuse pain on palpation, sometimes with swelling and calor when the synovium is involved. There are no clinical scoring systems for ankle osteoarthritis. The most relevant clinical outcome measures are:

1. Pain
2. Ankle joint function
3. Activity level

Patients presenting with ankle having some limited motion due to synovitis; a minimal to moderate degree of arthrofibrosis, osteophytes, chondral defects, or loose bodies; or only minimal instability can be candidates for arthroscopic surgery.

7.3 Pathology: Degenerative changes with osteophytes and synovitis are the most important causes of residual complaints after an ankle injury. Bargon, in 1978, developed a radiological staging system (table XIX). Van Dijk, in 1997, developed a radiological classification system which was validated by arthroscopic assessment (table XX). In this system, the observation of joint space narrowing is crucial in order to determine whether there is ankle joint degeneration,

7.4 Arthroscopy: According to Ferkel et al. the ankles that should be excluded from arthroscopic intervention are those with advanced destruction, marked joint-space narrowing, extensive fibrosis, and significant instability or deformity.¹³ However, this has not been validated in a follow-up study. It has been determined that osteoarthritic ankles have with clinical manifestation of anterior ankle impingement have 50% good/excellent outcome for arthroscopic removal of osteophytes at long-term follow-up.¹⁶

Available classification systems of osteoarthritis

Subjective: None

Objective: Radiographic system by Bargon (table XIX), radiographic classification by van Dijk (table XX)

Table XIX. Radiographic assessment of osteoarthritis by Bargon et al (1978).

Grade	Characteristics
0	No radiological changes
I	Early osteophytes
II	Increased bone density adjacent to the osteophytes
III	Narrowing of the joint space, subchondral sclerosis and cyst formation

Table XX. Radiographic classification for ankle joint degeneration (van Dijk et al. 1997, 2000)

Grade	Characteristics
0	Normal joint
I	Anterior osteophytes without joint space narrowing
II	Anterior osteophytes with joint space narrowing
III	Anterior osteophytes with total disappearance or deformation of the joint space

8. POSTERIOR ANKLE IMPINGEMENT

8.1 Definition: In a posterior ankle impingement syndrome; os trigonum, tendonitis of the flexor hallucis longus, posterior tibial or peroneal tendons; posttraumatic calcifications; bony avulsions; osteochondral defects; ankle and subtalar arthrosis; synovitis; loose bodies and their combinations can all be the cause of posterior ankle pain. Posterior ankle impingement is a clinical diagnosis, based on posteriorly located ankle pain, swelling and limited, painful plantarflexion. Because of its nature and deep location, posterior ankle problems pose a diagnostic and therapeutic challenge.

8.2 Symptoms and signs: In contrast to patients with anterior ankle pain, the structures of the hindfoot have a deep location, which makes direct palpation difficult. Moreover, the anatomic structures in the posterior ankle compartment are located closely to each other.

It is difficult to distinguish these various causes of posterior ankle pain by medical history and clinical examination alone. The patient experiences pain on the posterior aspect of the ankle joint, which is mainly present on forced plantar flexion. In some patients, forced dorsiflexion is also painful. In this dorsiflexed position, traction is applied to the posterior joint capsule and posterior talofibular ligament, which both attach to the posterior talar process. On examination, there is pain on palpation of the posterior aspect of the talus. This posterior talar process can best be palpated posterolateral in between the peroneal tendons and achilles tendon. Posteromedially, the neurovascular bundle and flexor tendons cover the talus. Posteromedial pain on palpation therefore does not automatically equal to impingement pain. The passive forced plantarflexion test is the most important test. A negative test rules out a posterior impingement syndrome. A positive test in combination with pain on posterolateral palpation should be followed by a diagnostic infiltration. If the pain on forced plantarflexion disappears, the diagnosis is confirmed. The AP radiographs typically do not show any abnormalities. On the lateral view often a prominent posterior talar process or os trigonum can be recognised. In post-traumatic cases there may be signs of non-union of for instance posterior talar fracture in this region. The posterior talar process or os trigonum is located posterolaterally. On the lateral view, this posterolateral part is often superpositioned onto the medial talar tubercle. Detection of a non-union on a standard lateral view therefore often is difficult. Calcifications for the same reason sometimes can not be detected by this standard lateral view. In posttraumatic cases therefore a bone scan must be performed if the radiographs do not show any abnormality. A positive bone scan can be followed by a CT-

scan. Especially in posttraumatic cases a CT-scan is important to determine the extent of the injury and the exact location of calcification or fragments.

8.3 Pathology: The most prominent posterior part of the talus is located posterolateral. Apart from the posterior joint capsule the posterior talofibular ligament attaches to this posterior talar process. Medial from this bony prominence the flexor hallucis longus tendon is located. This tendon thus separates the posterior talar process from the medial talar tubercle. The posterior talar process forms the roof of the posterior facet of the subtalar joint. Posterior ankle impingement can be caused by overuse or trauma. Distinction between these two appears important since posterior impingement through overuse has a better prognosis. A posterior ankle impingement syndrome through overuse is mainly found in ballet dancers and runners. The forceful plantarflexion during the “en-pointe” position or the “demi-pointe” position produces compression at the posterior aspect of the ankle joint.

Forced plantarflexion causes compression of the posterior talar prominence between the tibia and calcaneus. In the presence of an os trigonum, this can lead to a displacement of the os trigonum. In case of a prominent posterior talar process a fracture can occur. Compression of the posterior joint capsule can lead to calcification. Combined supination and plantar flexion (leading to a lateral ankle ligament lesion) in some patients also leads to compression of posteromedial joint structures. The post-traumatic calcifications in these cases most often are located posteromedially.

8.4 Arthroscopy: Once the diagnosis is made, a 2-portal endoscopic hindfoot approach can be used by means of which the above mentioned pathology can be visualized and treated.

Removal of a symptomatic os trigonum or a nonunion of a fracture of the posterior talar process involves partial detachment of the posterior talofibular ligament and release of the flexor retinaculum, both of which attach to the posterior talar prominence.

Arthroscopic evaluation of posterior ankle problems by means of routine ankle arthroscopy using an anteromedial, anterolateral, and posterolateral portal is difficult because of the shape of the ankle joint. Only in cases where the ligaments are very lax, it is possible to visualize and treat posterior pathology of the ankle joint by means of anterior portals. Pericapsular and extracapsular posterior ankle pathology cannot be treated by means of routine ankle arthroscopy. The 2-portal posterior endoscopic approach with the patient in the prone position offers excellent access to the posterior ankle compartment.

Classification of posterior ankle impingement

There are no classification systems available for the assessment of pathology in the posterior ankle compartment. Van Dijk and co-workers, however, developed a systematic arthroscopic examination which is mentioned previously in this section.⁷

9. ANTEROLATERAL ANKLE INSTABILITY

9.1 Definition: Chronic anterolateral ankle instability is defined as a situation in which a patient experiences recurrent episodes of giving-way with a duration of more than six months. It can be caused by either functional or mechanical instability or a combination of both. Functional instability is the most common residual disability after acute, lateral ligament ruptures and is a description of the subjective symptoms of the patient, e.g., repeated giving-way, in some cases combined with pain. Laxity, on the other hand refers to an objective measurement, e.g., standardized stress radiographs, or clinical measurement of the anterior drawer sign. Repeated ankle sprains may lead to chronic ankle instability with chronic pain secondary to repeated chondral and osteochondral injuries, especially on the medial side of the ankle joint.

9.2 Symptoms and signs: The clinical evaluation of chronic ankle joint instability is primarily based on the assessment of anterior drawer sign and the inversion (supination) test, always in comparison with the contralateral side. Increased anterior translation of the talus in the talocrural joint is due to rupture or elongation of the ATFL. Increased inversion is due to rupture or elongation of the CFL, or a combination of ATFL and CFL insufficiency, which is more common. There are no standardized arthroscopic classification systems for the assessment of ankle instability available. The two radiographic tests which are used are the lateral instability/laxity test (Talar Tilt, TT) and the anterior instability/laxity test (Anterior Talar Translation, ATT). Increased laxity can be defined either as a single value of ATT >10 millimetres or TT >9 degrees. Another way of defining increased laxity is a difference of ATT >3 millimetres, i.e. the difference in ATT between the functionally unstable ankle and the contralateral ankle and/or TT >3 degrees in patients with unilateral instability. A good correlation between functional and mechanical instability has been shown in some studies, but this correlation is highly variable, since several factors other than mechanical instability can be responsible for the development of functional instability. Several studies have questioned

the reliability of stress radiographs, especially the measurements of TT, and it must be born in mind that radiographs alone can never be used to establish the indication of surgery.

9.3 Pathology: Functional instability is a complex syndrome, in which mechanical, neurological, muscular and constitutional factors interact. The etiological factors are not exactly known and in several cases there is a combination of factors. Elongation of the ruptured ligaments, i.e. increased laxity, proprioceptive deficit, peroneal muscle weakness and subtalar instability are all documented etiological factors of functional instability, either alone or in combination. In some studies a correlation between functional instability and increased laxity using standardized stress radiographs, has been shown. Although these radiographic stress tests may be useful, the reliability of these tests is rather low and there is no definite correlation between functional instability and increased laxity.

Several researchers have demonstrated that delayed proprioceptive response to sudden angular displacement of the ankle may be an important cause of functional instability of the ankle joint. Functional instability, therefore is caused by increased laxity, inhibition of proprioceptive function, peroneal muscle weakness or a combination of these factors. The specific cause of functional instability in each individual case has to be analysed separately.

9.4 Arthroscopy: At arthroscopy, patients demonstrate an attenuated anterior talofibular ligament with scarring of the lateral gutter and syndesmosis. There may be associated loose bodies or ossicles. With recurrent instability or after a severe inversion injury, additional abnormalities can be seen on the medial side as the talus tilts, abutting the medial dome against the surface of the tibia. Pathology and synovitis are sometimes seen in the posterior ankle as well.

Arthroscopic ligament stabilization can be an alternative to open reconstructions. The ATFL is shortened and reinserted by the use of a percutaneous staple. Only a few reports on this technique are found in the literature and it is probably very little used today. There might be a specific indication for this technique in children in order to avoid damage to the growth plate of the fibula. The major drawback of the arthroscopic stabilization is that this technique makes it possible to reconstruct only the ATFL. The arthroscopic technique for lateral ankle ligament stabilization is demanding, and the procedure should be performed only by surgeons with very good experience in ankle arthroscopy. Future development, especially the development of biodegradable implants and capsular shrinkage techniques might make this

procedure more useful, but as things stand today, further studies are needed to define the exact role of arthroscopic technique in stabilisation of the unstable ankle.

Classification of anterolateral ankle instability

There are no standardized classification systems available for the assessment of chronic anterolateral ankle instability. Karlsson and co-workers developed criteria for the assessment of stress radiographs in 1991. Increased laxity can be defined either as a single value of anterior talar translation >10 mm or talar tilt >9 degrees. Another way of defining increased laxity is a difference of anterior translation >3 mm in comparison with the unaffected ankle and a difference of talar tilt >3 degrees in comparison with the unaffected ankle.²⁹

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KNEE

1. Introduction

The objectives of this monograph are to provide standard terminology, definitions and classification systems in the field of knee arthroscopy and to provide guidelines for the assessment of normal and pathologic aspects of the knee joint at arthroscopy.

Assessment of knee pathology begins with the clinical diagnosis, which is based upon history and physical examination. The clinical diagnosis might be confirmed by radiological investigations. This monograph describes nine different clinical diagnoses. For each diagnosis a definition is given, followed by a brief description of the symptoms and signs as well as the underlying pathologic process. Typical arthroscopic findings are presented, together with classification systems that are currently in use for those findings. The monograph concludes with a brief discussion of instruments that are currently used to measure outcomes in the research of knee pathologies and treatments.

Clinical Diagnosis and findings at arthroscopy: definitions and classifications

Clinical Diagnosis	Arthroscopy
2. Asymptomatic knees	Asymptomatic lesions, plicae, adhesions, ossicles, spurs, or synovitis
3. Loose bodies	Osteophytes, ossicles, synovitis, scar tissue, osteochondrosis dissecans, osteochondral fracture, chondromatosis
4. Synovitis	Posttraumatic synovitis, soft tissue impediments, arthritis, pigmented villonodular synovitis, inflamed plica, arthrofibrosis, septic synovitis
5. Osteochondrosis dissecans (OCD)	Cartilage flaps, loose bodies, inflammatory synovitis, persistent or intermittent joint effusion, secondary joint degeneration
6. Osteochondral defects	Loose bodies, posttraumatic calcifications, avulsion fragments, chondral fractures
7. Osteoarthritis	Osteoarthritis, chondromalacia, osteophytes, chronic

	synovitis, subchondral cysts, joint space narrowing
8. Meniscal injuries	Degenerative or traumatic lesions, flap, radial or longitudinal tears, distance to periphery, zone classification
9. Instability	Instability is not an arthroscopic diagnosis
10. Patellofemoral disorders	Patellar instability is not an arthroscopic diagnosis

2. ASYMPTOMATIC KNEES

2.1. Introduction: In an examination of the knee, extra- as well as intraarticular pathologic findings do not always produce symptoms. Thorough knowledge of the arthroscopic anatomy is necessary to distinguish between normal and abnormal findings.

2.2. Definition: A patient has an asymptomatic knee when there are no complaints and when tests and physical examination are negative, regardless of whether there is any pathologic finding present at arthroscopy.

2.3. Symptoms and Signs: There may be a history of intermittent or transient complaints but there are no current symptoms. Physical examination may be normal or reveal slightly abnormal range of motion, swelling, clicking, or crepitation. Radiographic examination may show a normal knee with osteophytes, intraosseous cysts, or even mild arthrosis.

2.4. Pathology: A variety of pathologies can be asymptomatic. Meniscal degeneration or joint surface changes might not cause symptoms, and then suddenly become symptomatic after a trivial sprain.. Likewise synovial adhesions or synovial duplications (plicae) usually are not symptomatic unless the patient develops acute synovial inflammation..¹⁻³ Patella baha or a flat trochlear groove might also be asymptomatic.

2.5. Findings at Arthroscopy: In the asymptomatic knee, the suprapatellar pouch, the lateral gutter, and patellofemoral articulation are usually normal. In the medial gutter, a medial plica is frequently found that can be of varying size and thickness. In very rare cases the plica might become pathologic after a contusion or other traumatic event, resulting in thickening and inflammation. The articular surfaces of the femoral condyles and tibiae are usually shining and intact, but might be eroded, irregular, or even split without causing symptoms. The appearance of the menisci can vary from shining, elastic, and flexible to grayish, dull, and calcified. The size of the menisci can also vary, especially the lateral meniscus.⁴⁻⁵ Patients may have mild synovitis in the anterior gutter of the knee or around the posterior cruciate ligament.

3. LOOSE BODIES

3.1. Introduction: Loose bodies, a common condition, may occur in any joint due to a variety of pathological processes. Awareness of the different conditions that may cause intraarticular loose bodies is necessary for proper medical and surgical treatment.

3.2 Definition: Loose bodies are fibrous, bony, cartilaginous and osteocartilaginous fragments in a synovial joint.

3.3. Symptoms and Signs: Intra-articular loose bodies may present with similar signs and symptoms despite being secondary to different pathologic conditions. Although other findings may be present, depending on the underlying pathology, loose bodies grow over time; a dramatic enlargement has been observed in some cases.⁶ Patients most commonly report the sensation of a loose body as something moving in the knee. Clinical findings include the presence of swelling, stiffness, and mechanical symptoms such as locking or catching. An antalgic gait may be observed.

3.4. Pathology: Major causes are osteochondritis dissecans, synovial chondromatosis, osteophytes, fractured articular surfaces, and damaged menisci.

3.4.1 Osteochondritis dissecans: Osteochondritis dissecans (OCD) is an acquired, potentially reversible idiopathic lesion of subchondral bone that may result in instability of the fragment and cartilage. Knee OCD has been classified according to the anatomical location, surgical appearance, age of the patient (juvenile or adult), and stability of the fragment. Bony,

cartilaginous, and osteocartilaginous loose fragments are quite common. The clinical presentation occasionally includes the finding of a loose body. Specifically, on palpation, maximal tenderness can be elicited over the anteromedial aspect of the knee, which is the most common site of OCD lesions.

3.4.2 Synovial chondromatosis: Synovial chondromatosis is a rare and benign metaplasia of the synovial membrane that results in the formation of multiple intraarticular cartilaginous bodies. Synovial chondromatosis is known by several other names, including articular chondrosis and synovial chondrosis. This self-limited and nonaggressive condition occurs most commonly in the knee followed by the hip, shoulder, and elbow. It occurs most commonly during the third to fifth decade, twice as often in men as women, and not at all in children. Synovial chondromatosis occurs as the gradual onset of monoarticular pain and stiffness. If allowed to continue, progressive symptoms can result in a decreased range of motion, effusion, crepitation, and eventual locking of the joint. Secondary synovial chondromatosis may be present after long-standing osteoarthritis.

3.4.3 Osteophytes: Bone spurs, or osteophytes, are bony projections that form along joints; they are often seen in conditions such as arthritis. Bone spurs can cause pain and are largely responsible for limitations in joint motion. Osteophytes can also become loose bodies secondary to small fractures. In such cases, common clinical findings of loose bodies can overlap with symptoms of arthritis.

3.4.4 Fractured articular surfaces: Tibial plateau fractures or tibial bone fragments from detached insertions of the cruciate ligaments may become intraarticular loose bodies. The clinical presentation depends upon the etiology of these loose bodies.

3.4.5 Damaged menisci: Some meniscal tears can produce loose bodies. Clinical findings may include typical signs and symptoms of meniscal tears and loose bodies.

3.5 Findings at arthroscopy: Loose bodies can be located in any compartment of the knee joint. Due to gravity, they quite often are located in the posterior compartment. Loose bodies in the posterior compartment have been classified in 6 sections:⁷ posteromedial, posterolateral, around the posterior septum, posterior supracondylar, posterior popliteal hiatus, and intrameniscal recess. A case of locked knee caused by a loose body in the fabellofemoral

joint has been described.⁸ Loose bodies also can be found in the anterior, medial, lateral, and suprapatellar compartments. In synovial chondromatosis loose bodies can be located in all of the compartments, as can cartilaginous bodies not yet separated from the synovium.⁹ In some cases hundreds of brilliant white loose bodies are found in the joint; the term “snow storm knee“ has been proposed for this condition.¹⁰

3.6 Outcome measures: The International Knee Documentation Committee (IKDC) Current Health Assessment Form (SF-36),¹¹ Subjective Knee Evaluation Form,¹²⁻¹³ and Knee Examination Form¹⁴ are recommended to evaluate the outcomes of loose body removal and treatment for the underlying pathology.

4. SYNOVITIS

4.1 Introduction: The synovial membrane lines all of the inner surfaces of the joint except the area of the cartilage. It is normally very thin and produces synovial fluid. The synovial membrane carries nutrients to and lubricates the cartilage. In some diseases this membrane is the primary target of inflammation.

4.2 Definition: Synovitis is not a disease per se. It is the way the synovial membrane reacts during inflammation. The mediator of a specific disease starts the inflammatory process. Synoviocytes proliferate and inflammatory cells infiltrate from the vessels. Macroscopic characteristics are hypertrophy of the synovial membrane and proliferation of vessels leading to increased vascularity.

The arthritis is classified as acute (lasting less than 3 weeks), subacute (lasting 4 to 6 weeks) or chronic (lasting more than 6 weeks). When the arthritis is present in only one joint it is called monoarthritis, when present in a few joints it is called oligoarthritis, and when present in many joints it is called polyarthritis. Polyarthritis can begin as monoarthritis or oligoarthritis.

Examples of monoarthritis:

Septic arthritis, trauma, crystal arthropathies, reactive arthritis, haemarthrosis, osteoarthritis

Examples of oligoarthritis:

Psoriatic arthritis, enteropathic arthritis, ankylosing spondylitis, reactive arthritis, osteoarthritis

Examples of polyarthritis:

Viral arthritis, rheumatoid arthritis, osteoarthritis, systemic lupus erythematosus

Rheumatoid arthritis and other chronic diseases will sometimes be undiagnosed for a long time because the symptoms and the histologic picture present as unspecified synovitis/arthritis.¹⁵

4.3 Symptoms and Signs

4.3.1 Clinical classification: Symptoms and the grade classification vary with the activity of the synovitis and the type of disease. The following symptoms are common in connection with synovitis:

- Soft tissue swelling in the joint, hypertrophy of the joint capsule
- Joint effusion
- Pain and ache
- Stiffness in and around the joint, especially after rest
- Muscle weakness near the joint
- Extraarticular symptoms and involvement of other joints, depending upon the specific disease

In longstanding chronic cases extension and/or flexion lag, muscle atrophy, or bone loss may occur.

4.3.2 Classification system of septic arthritis according to Tan et al¹⁶: Joint name (glenohumeral, elbow, hip, knee, etc.)

- Anatomic type
 - I. Periarticular soft-tissue infection without pyarthrosis
 - II. Isolated septic arthritis
 - III. Septic arthritis with soft-tissue extension, but no osteomyelitis
 - IV. Septic arthritis with contiguous osteomyelitis
- Host class (according to Cierny and Mader¹⁷⁻¹⁸):
 - A. Normal immune system
 - B. Compromised system
 - B^L: Local tissue compromise
 - B^S: Systemic immune compromise
- Clinical setting
 - A. Less than 5 days of symptoms and nonvirulent organism
 - B. Symptoms for 5 days or more, or a virulent organism
- Clinical stage for the septic joint

Anatomic type + host class + clinical setting = stage

4.4. Pathology

4.4.1 Radiologic classification: Some signs are typical, but in most types the signs of synovitis will be seen only in cases of chronic and/or aggressive synovitis. In those you can see erosion of the bone and degenerative changes in the joint that are most often not disease-specific. Thinning of cartilage in the knee joint will be seen in all compartments.

Specific radiologic signs:

Chondrocalcinosis (pyrophosphate arthritis)

Osteochondromatosis: multiple chondral or osteochondral lesions

4.4.2 Radiograph classification according to Larsen, Dale, & Eek¹⁹:

- **Grade 0:** Normal conditions. Abnormalities not related to arthritis, such as marginal bone deposition, may be present.
- **Grade I:** *Slight abnormality.* One or more of the following lesions are present: periarticular soft-tissue swelling, periarticular osteoporosis, and slight joint space narrowing. When possible, use for comparison a normal contralateral joint or a previous film of the joint in the same patient. Soft-tissue swelling and osteoporosis may be reversible. This stage represents an early uncertain phase of arthritis or a later phase without destruction. Compatible appearances may occur without arthritis in old age, traumatic conditions, Sudeck atrophy, etc.
- **Grade II:** *Definite early abnormality.* Erosions and joint space narrowing corresponding to the standards. Erosions must be present and joint space narrowing except in weight-bearing joints.
- **Grade III:** *Medium destructive abnormality.* Erosions and joint space narrowing corresponding to the standards. Erosions must be present in both weightbearing and nonweightbearing joints.
- **Grade IV:** *Severe destructive abnormality.* Erosions and joint space narrowing based on the radiographic criteria. Bone deformation is present in the weight-bearing joints.
- **Grade V:** *Mutilating abnormality.* The original articular surfaces have disappeared. Gross bone deformation is present in the weight-bearing joints. Dislocation and bony ankylosis, being late and secondary, should not be considered in the grading. If present, the grading should be made according to the concomitant bone destruction or deformation.

4.4.3 MRI classification: Pigmented villonodular synovitis (PVNS) is easy to diagnose because of the typical content of hemosiderin. Pathologic anatomical diagnosis (PAD) is specific for PVNS. In most cases the PAD is unspecified synovitis.

4.5. Findings at Arthroscopy

4.5.1 Classification and stages: There are no good systems at the moment for macroscopic evaluation of different stages of synovitis. In most cases there are nonspecific changes on the histological examination.²⁰

In osteoarthritis the characteristic finding is cartilage degeneration; the inflammation is confined to areas of the synovial membrane near the cartilage. That means that the major part of the joint cavity is not affected. Microscopically the synovitis is indistinguishable from those seen in rheumatoid arthritis.²¹ RA has Russell's bodies, Allison – Ghormley bodies, fibrinous exudate, etc.)

Pyrophosphate-synovitis: Calcification in menisci and chondrocalcinosis

4.5.2 Synovial histology in different diagnoses:

Nonspecific synovitis

- Immune-mediated disease, such as rheumatoid arthritis, systemic lupus erythematosus, and spondyloarthropathies (ankylosing spondylitis, psoriatic arthritis, reactive arthritis, enteropathic arthropathies)
- Arthritis caused by bacterial or viral infection in the joint
- Arthritis caused by trauma/microtrauma
- Arthritis as a paramalignant phenomenon
- Osteoarthritis

Specific synovitis (sometimes)

- Crystal arthritis, such as gout
- Arthritis caused by haemarthrosis (for example, haemophilia)
- Arthritis caused by osteochondromatosis

Specific synovitis (always)

Pigmented villonodular synovitis (PVNS). PVNS usually has a typical arthroscopic picture with thick brown-red synovitis with bulky villi (villonodular).

4.5.3 Arthroscopic stages of septic arthritis according to Gächter²² and Stutz et al.²³:

Stage I: Opacity of fluid; redness of the synovial membrane; possible petechial bleeding, no radiologic alterations

Stage II: Severe inflammation; fibrinous deposition; pus; no radiologic alterations

Stage III: Thickening of the synovial membrane; compartment formation (“spongelike” arthroscopic view, especially in the suprapatellar pouch); no radiologic alterations

Stage IV: Aggressive pannus with infiltration of the cartilage, possibly undermining the cartilage; radiologic signs of subchondral osteolysis; possible osseous erosions and cysts.

4.6 Outcomes Measures: No outcomes measures have been validated specifically for synovitis.

5. OSTEOCHONDRITIS DISSECANS (OCD)

5.1 Introduction: Osteochondritis dissecans is a condition of the joint that appears to affect subchondral bone primarily, with secondary effects on articular cartilage. With progression, this pathology may present clinically with symptoms related to the integrity of the articular cartilage.²⁴ It is found more frequently in children who are active athletically and involved in organized sports and it is twice as common in males as in females. Although the etiology of these lesions is unclear, it is believed that repetitive microtrauma may contribute to the development of osteochondritis dissecans lesions.²⁵

5.2 Definition: Osteochondritis dissecans is a focal injury or condition affecting the subchondral bone. If the bone fails to heal, progressive changes occur in the articular cartilage, including softening, swelling, early separation, partial detachment, and complete osteochondral separation of the loose bodies.

5.3 Symptoms and Signs: Early symptoms, associated with intact cartilage, may be related to softening and alteration in the mechanical properties of cartilage. Later stages, because of the lack of underlying support of the cartilage, can present with symptoms of articular cartilage separation, cartilage flaps, loose bodies, inflammatory synovitis, persistent or intermittent joint effusion, and, in severe cases, secondary joint degeneration.²⁴

5.4. Pathology: Trauma may be caused by direct force, such as impaction fracture, or repetitive microtrauma, such as excessive normal compressive stress. The pathology of OCD can be described in three stages.

- In the first stage (acute injury), thickened and edematous intraarticular and periarticular soft tissues are observed. Often, the adjacent metaphysis reveals mild osteoporosis resulting from active hyperemia of the metaphysis.
- In the second stage, the epiphysis reveals an irregular contour and thinning of the subcortical zone of rarefaction. On radiography, the epiphysis may demonstrate fragmentation. Blood vessels within the epiphysis are incompetent because of thrombosis or microfractures of the trabeculae, which results in poor healing.
- The third stage is the period of repair in which granulation tissue gradually replaces the necrotic tissue. Necrotic bone may lose its structural support, which results in compressing and flattening of the articular surface.

In the knee joint, the medial femoral condyle is the most commonly involved site. Potential locations are the lateral aspect of the medial femoral condyle (75%), the weight-bearing surface of the medial (10%) and lateral (10%) femoral condyles, and the anterior intercondylar groove or patella.^{26,27} Rarely, OCD occurs in the medial tibial plateau.

5.5 Findings at Arthroscopy: See Section 6.5

5.6. Outcome Measures: See Section 6.6

6. OSTEOCHONDRAL DEFECTS

6.1 Introduction: In the knee, several distinct lesions of the articular cartilage exist. These include idiopathic chondromalacia, degenerative chondrosis, osteochondritis dissecans, osteochondral fractures, chondral flaps, and chondral fractures. The goals of this text are to create an accurate description of the true nature of osteochondral defects and to establish a common understanding of their etiology and pathology, as well as expected arthroscopic findings. The first section deals with a brief biologic description of the cartilage in the knee joint, which is intended to facilitate recognition of the true nature of these lesions and their sequela. The second part discusses the description of the lesions as reported in the literature.

6.1.1 Cartilage biology in the knee: One of the most extensive studies in the literature that provides ample information as to the incidence of chondral damage that can be seen upon arthroscopic examination of the knee is the study done by Curl et al²⁸ with a review of 31 516 knee arthroscopies which revealed a total of 53 569 hyaline cartilage lesions. The prevalence of chondral injury in this series of arthroscopies was as follows: 0.7% OCD lesions; 1.3% articular fractures; 9.7% Grade I chondromalacia (CM); 28.1% Grade II CM; 41.0% Grade III CM; and 19.2% Grade IV CM.

Hyaline cartilage makes up the protective covering of the femur, tibia, and the undersurface of the patella. It provides a shock-absorbing property that can withstand compression, tension and shearing forces, and at the same time dissipate load during weight-bearing. Hyaline cartilage also provides an almost frictionless articulating surface for mobility.

Articular cartilage in the knee must withstand repetitive mechanical forces that can sometimes reach 65 times body weight. It is well adapted for this through its biochemical and biophysiological properties: it is composed of a network of collagen fibers and a proteoglycan

matrix within which lie cartilage cells. The collagen is responsible for the tensile strength, while the proteoglycan matrix (consisting of 80% water) resists compressive forces.²⁹ Articular cartilage derives its nutrition primarily from the synovial fluid and, to some extent, from the adjacent bone. The lack of blood supply has implications with regard to healing, since superficial lesions rely solely on very slow and unsatisfactory cell mitosis and regeneration for repair. Deeper lesions that, together with the articular cartilage, involve the underlying bone, heal better because the repair cells in the bone have direct access to the cartilage defect. Repair by these cells, although better than that seen with superficial lesions, is far from ideal since the type of cartilage produced is not the original hyaline cartilage but 'fibrocartilage,' which is not as well adapted to the mechanical forces generated on weight bearing. It is important to understand the difference between these types of repair as it forms the rationale behind the treatment options for such injuries.²⁹

6.2 Definition

6.2.1 Traumatic chondral injury: Traumatic chondral lesions generally occur as a result of compression and rotational shearing forces.³¹ These injuries are solitary lesions with acutely angled margins that occur in the deep layers or at the surface of the cartilage. Often these lesions are partial-thickness or full-thickness tears, with associated bone marrow changes. Chondral or osteochondral fragments may be avulsed and cause locking, similar to displaced meniscal tears.^{29,30}

Unstable osteochondral lesions are completely loose fragments or lesions that are covered by cartilage that is torn and shows no continuity with neighboring cartilage.

Stable lesions, on the other hand, are covered by cartilage that is continuous with the normal surrounding articular cartilage.

6.2.2 Chondral flaps/chondral fractures: Chondral flaps and fractures are traumatic injuries usually associated with ligamentous tears. They are very difficult to diagnose clinically because the history and clinical findings frequently mimic those of meniscal tears, which are more common.

6.2.3 Chondromalacia/chondrosis: Chondromalacia is described as softening or fissuring of the articular hyaline cartilage that may result from an excessive load on the patellofemoral joint. There are several stages:

1. Swelling and softening of the cartilage
2. Fissuring of the softened areas

3. Fasciculations of the articular cartilage almost to the level of the subchondral bone
4. Destruction of cartilage with exposed subchondral bone

6.3 Symptoms and Signs: The presenting features of articular cartilage damage are nonspecific. These include intermittent pain and swelling. The patient may also present with locking or giving way if the fragment has displaced into the joint. On examination, there may be muscle atrophy, a reduced range of motion, and tenderness over the articular cartilage lesion. Most commonly though, patients present with a chief complaint of pain in the respective compartment (i.e., medial, lateral, or anterior). Direct weight bearing may aggravate tibiofemoral symptoms, whereas stair climbing and squatting typically increase patellofemoral symptoms. Other common symptoms include localized pain, effusion, catching, and occasional locking, or mechanical symptoms.³¹

In cases of acute trauma, the patient may have a hemarthrosis. Blood within the knee suggests that the cartilage defect may extend into the subchondral bone. A fracture in the knee should always be included in the differential diagnosis of hemarthrosis. The other common causes of hemarthrosis are an anterior cruciate ligament (ACL) tear, a peripheral meniscal injury, and a severe bone bruise.

6.4. Pathology: A single high-impact force may cause chondrocyte death, matrix damage, shearing of cartilage fragments, subchondral bone injury, and fracture. Abrasive injuries from chronic meniscal tears or loose bodies can also damage articular cartilage. Repetitive overloading with forces exceeding the biomechanical carrying capacity of articular cartilage from obesity or malalignment may cause irreparable damage.

Biochemical injuries result from joint sepsis, inflammation, and chondrocyte impairment. Bacteria frequently produce collagenases and other degradative enzymes. Immune response to articular cartilage antigens can result in extensive cartilage and joint destruction. Chondrocyte impairment because of genetic deficiencies or aging can change the quality of the matrix and alter the critical balance between matrix synthesis and degradation to favor degradation, resulting in articular cartilage damage and destruction.²⁷

Although obvious to most surgeons, it is not obvious to most patients that joint pain may be, in fact, not directly related to articular cartilage pathology ascertained from imaging studies or following arthroscopic evaluation. The relationship of pain to the extent of articular cartilage injury is at best indirect. Because articular cartilage lacks nerve supply, the pain originates from either the subchondral region (bone, nerve, and vascular network) or from the soft

tissues stimulated by the local biochemical/mechanical effects of flaps or desquamated cartilage.

Damaged articular cartilage has an extremely limited capacity for intrinsic repair. A single laceration to articular cartilage that does not extend into subchondral bone does not heal. Within the cut, chondrocytes are killed and the matrix is disrupted. Imprisoned in undamaged matrix, neighboring chondrocytes are unable to migrate into the cleft. Some may temporarily increase metabolic activity and a thin layer of new matrix may even cover the surface. However, this repair is transient and without structural integrity.²⁸ With time, cumulative damage leads to accelerated loss of functional articular cartilage. As the cartilage thins out, forces in the underlying bone increase. The bone responds by becoming stiffer. It may also attempt to increase its surface area in response to higher mechanical loads and form osteophytes. Small subchondral fractures allow for cyst formation. The joint progressively degenerates, descending into the all too prevalent final common pathway of debilitating osteoarthritis.

The limited repair potential of articular cartilage has been attributed to the absence of a blood supply with its attendant repair cells and healing factors. Without a vascular response to injury, there is no fibrin clot formation, no inflammatory response, and no migration of undifferentiated repair cells into the defect. Given this set of circumstances, cartilage procedures such as arthroscopic debridement and thermal chondroplasty that do not penetrate subchondral bone cannot be expected to affect articular cartilage repair.

6.4.1 The role of diagnostic imaging modalities: Since all the above signs and symptoms can be present in a number of different knee conditions it can be quite difficult to confidently reach a diagnosis on clinical findings only.

Radiographs will only show cartilage damage if the damage is associated with underlying bone injury and, even so, small lesions can easily be missed. Lesions appear as a line of demarcation around a small area of bone if the lesion is still attached, and as a 'crater' or loose body if separation has occurred.

A more detailed mode of investigation is MRI scanning. This will not only show the osteochondral lesion clearly but will also provide information about the surrounding bone, menisci and the ACL. However, MRI scanning still compares poorly to direct inspection of the joint surface at the time of an arthroscopy. MRI is an excellent way to obtain precise information on rotational abnormalities of the femur and tibia, and on patellar orientation.^{32,33}

TABLE 6.1 MR Classification System in Grading Cartilage Lesions Described by Yulish et al.³³ (Based on the Arthroscopic Examination Described by Outerbridge³⁴)

Arthroscopic Classification		MR Classification	
Grade 0	Normal	Grade 0	
Grade 1	Softening	Grade 1	Normal contour ± abnormal signal
Grade 2	Superficial blistering or fraying	Grade 2	Superficial fraying
Grade 3	Surface irregularity and thinning	Grade 3	Surface irregularity and thinning
Grade 4	Ulceration and bone exposure	Grade 4	Full thickness cartilage loss

6.4.2 Classification of articular cartilage defects: The International Knee Documentation Committee (IKDC) form^{12,13,14} and the Lysholm score³⁵ are acceptable outcome instruments. The IKDC Current Health Assessment Form (SF-36)^{11,14} is a helpful quality-of-life survey.

6.5. Findings at Arthroscopy: Diagnostic arthroscopy of the knee joint may reveal pertinent information regarding the location and extent of the osteochondral damage. An adequate history, complete physical examination, and diagnostic arthroscopy minimizes the chance of underdiagnosis or missed diagnosis of articular cartilage defects.

According to Hjelle K et al,³⁶ in their study of articular defects in 1000 knee arthroscopies, the main focal chondral or osteochondral defect can found on the medial femoral condyle in 58% of cases, patella in 11%, lateral tibia in 11%, lateral femoral condyle in 9%, trochlea in 6%, and medial tibia in 5% of cases. It has been suggested that cartilage repair surgery may be most suitable in patients younger than 40 to 50 years old.

6.5.1 Arthroscopic classification: Arthroscopy allows direct visualization of the cartilage. Operative reports can include videos, pictures, and precise mapping of the cartilage. The French Society of Arthroscopy (SFA) has developed a precise scoring and grading scale.³⁷ The American College of Rheumatology proposed a Knee Arthroscopy Osteoarthritis Scale (ACR/KAOS).³⁸ This scale is less reliable but easier to use.³⁹

Several observations can be recorded:

- *Defect thickness.* The Outerbridge classification is the most accepted method, modified by Brittberg.⁴⁰⁻⁴¹ Cartilage changes are classified as normal (grade 0), softening (grade 1), fibrillation (grade 2), fissuring (grade 3) and bone exposure (grade 4). Grades 1 and 2 are partial lesions; grades 3 and 4 are full-thickness lesions. Evaluation of the tidemark is also important.⁴²
- *Size.* Measurements are usually made with a probe. Defects < 2cm² are considered small, 2 to 10 cm² moderate, and >10 cm² large.
- *Degree of containment.* A lesion can be more or less shouldered or contained. Poor containment leads to loss of joint space
- *Location.* Determination of the precise site of the defect is mandatory. Mono- or multipolarity of the lesion can be evaluated.
- *Associated lesions.* Meniscal, synovial, and ligamentous pathologies can be thoroughly evaluated.
- *Status of repair.* Articular cartilage stiffness can be evaluated with a probe and biopsies can be done. Second-look arthroscopies are relatively invasive and cannot be used as a routine outcome measure.

Cartilage lesions in weight-bearing areas require different treatment and have worse prognoses than lesions in non-weight-bearing areas. The location of articular cartilage lesions may be described as in Table 5.2. In addition to sign, depth, and location, associated subchondral and ligamentous lesions should be reported.

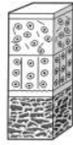
TABLE 6.2.. Location Descriptions for Cartilage Lesions

Location of Cartilage Lesions			
Medial femoral condyle	Anterior 1/3	Central 1/3	Posterior 1/3
Lateral femoral condyle	Anterior 1/3	Central 1/3	Posterior 1/3
Medial tibial plateau	Anterior 1/3	Central 1/3	Posterior 1/3

6.6 Outcome Measures: The ICRS articular cartilage injury classification system: The International Cartilage Repair Society (ICRS) has devised a system to describe the depth and location of articular cartilage lesions (Figures 5.1–5.3).⁴⁶ However, in the United States most surgeons report articular cartilage lesions using a modified Outerbridge^{40,41} grading system. Confusion can occur if researchers do not state which classification system is used. In the modified Outerbridge system, grade 3 represents deep fissuring of 50% depth with palpable but not exposed bone, and grade 4 represents exposed bone without subchondral involvement. (See Table 5.3) In contrast, the ICRS has four subcategories of grade 3, with 3c corresponding to the modified Outerbridge grade 4 (i.e., exposed bone) whereas ICRS grade 4 represents bony involvement.

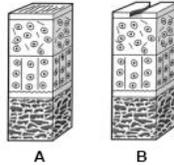
The ICRS system focuses on objectively measurable parameters of the lesion's extent and not its surface appearance.⁴⁰ The system was developed during the ICRS standards workshop at Schloss Munchenwiler, Switzerland in January 2002 and further discussed during the 3rd ICRS meeting in GoteborgSweden.⁴⁶

ICRS Grade 0 - Normal



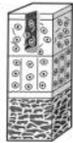
ICRS Grade 1 – Nearly Normal

Superficial lesions. Soft indentation (A) and/or superficial fissures and cracks (B)



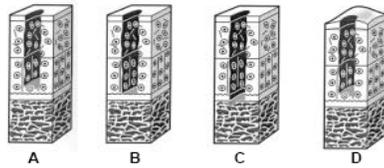
ICRS Grade 2 – Abnormal

Lesions extending down to <50% of cartilage depth

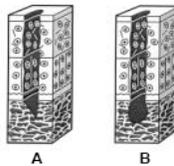


ICRS Grade 3 – Severely Abnormal

Cartilage defects extending down >50% of cartilage depth (A) as well as down to calcified layer (B) and down to but not through the subchondral bone (C). Blisters are included in this Grade (D)

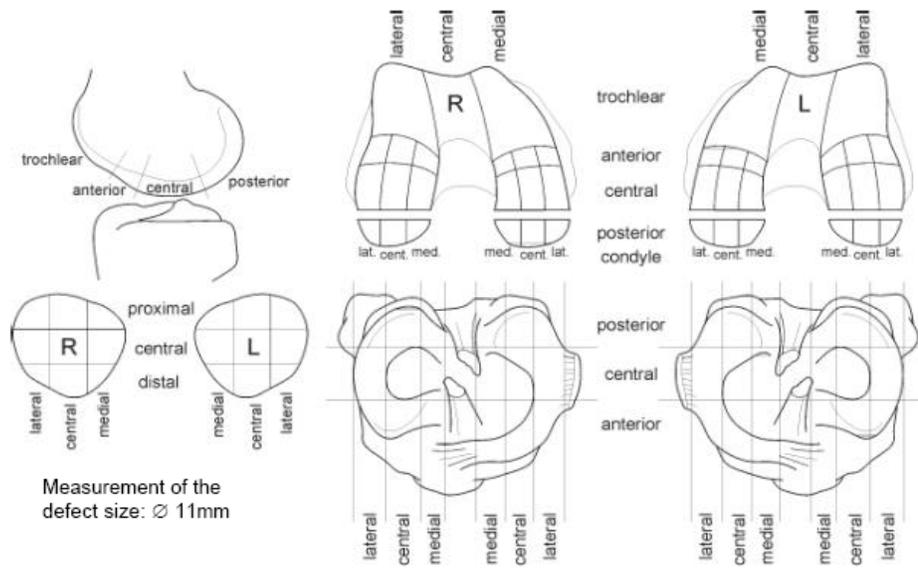


ICRS Grade 4 – Severely Abnormal



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FIGURE 6.1. *The International Cartilage Repair Society (ICRS) system for describing the depth of articular cartilage lesions*

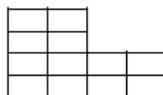


Femur

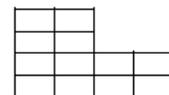
Side Right Left
 Condyle medial lateral
 Sagittal plane trochlear anterior
 Frontal plane lateral central

central posterior
 medial

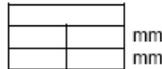
First lesion



Second lesion



Cartilage lesion (Grade) (*)
 Defect size pre-debridement
 Defect size post-debridement

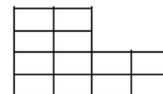


Tibia

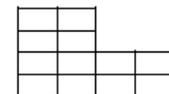
Side Right Left
 Plateau medial lateral
 Sagittal plane anterior central
 Frontal plane lateral central

posterior
 medial

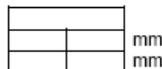
First lesion



Second lesion



Cartilage lesion (Grade) (*)
 Defect size pre-debridement
 Defect size post-debridement



Patella

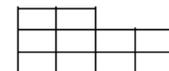
Side Right Left
 Sagittal plane distal central
 Frontal plane lateral central

proximal
 medial

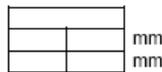
First lesion



Second lesion



Cartilage lesion (Grade) (*)
 Defect size pre-debridement
 Defect size post-debridement



Diagnosis: Traumatic cartilage lesion OD OA AVN Others

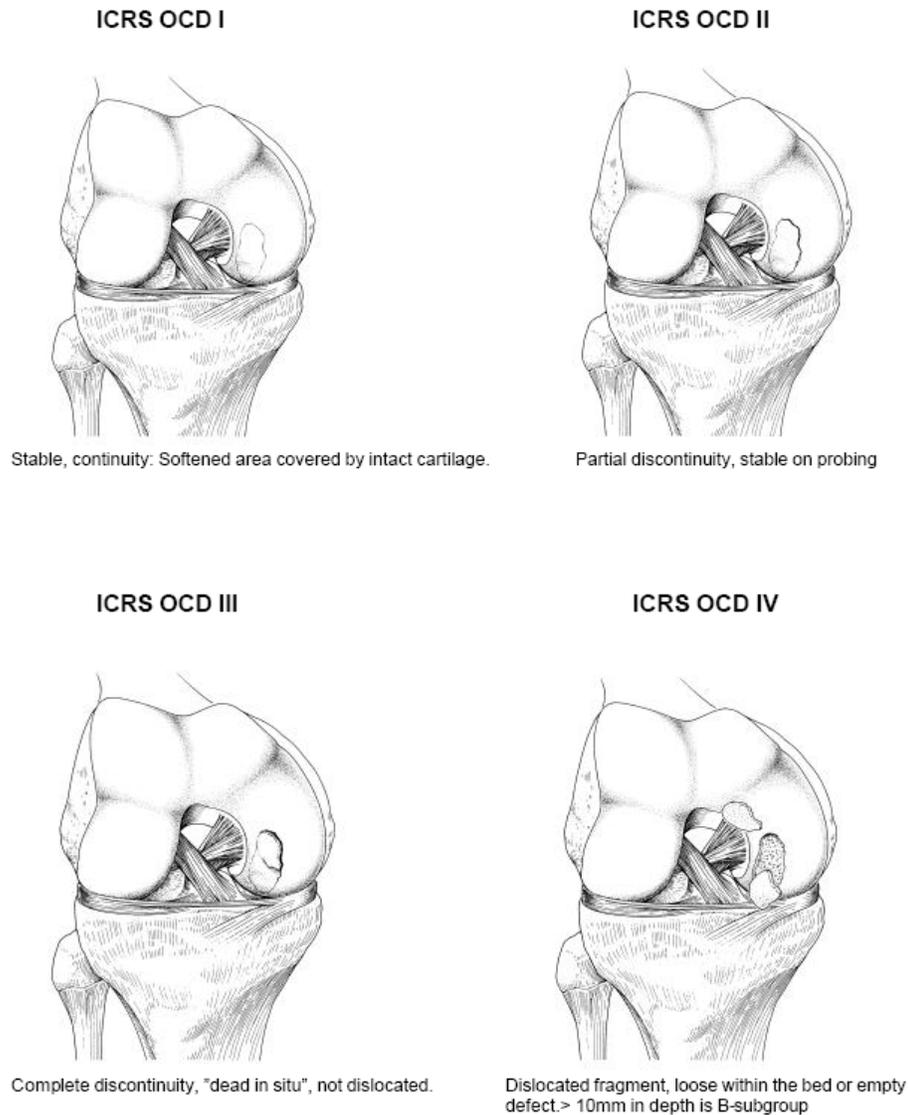
Biopsy/Osteochondral Plugs: Location: Number of Plugs: Diameter of Plugs:

Treatment: Shaving Drilling Others:
 Mosaic-Plasty Microfracture
 Autologous Chondrocyte Implantation (ACI)

Notes:

FIGURE 6.2 The ICRS Articular Cartilage Injury Mapping System

ICRS Classification of OCD-Lesions (Osteochondritis-Dissecans)



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FIGURE 6.3. *The ICRS Classification of OCD Lesions*

7. OSTEOARTHRITIS

7.1. Introduction: A more appropriate term for this condition would be arthrosis, since inflammation is not always present. It is the result of the biomechanical failure of the joint cartilage due to physical forces, ending in loss of integrity of articular cartilage. Primary osteoarthritis is progressive⁴⁷ and increases after 50 years of age. It is estimated that 25% to 30% of those aged 45 to 64 years of age and more than 85% of those older than 65 years of age have radiographically detectable osteoarthritis.⁴⁸ Secondary osteoarthritis may occur earlier after significant injuries resulting in either varus or valgus malalignment, osteochondral fractures or ligamentous and meniscal deficiency.⁴⁹ Rangger et al.⁵⁰ reported an increase in osteoarthritis after partial arthroscopic medial or lateral meniscectomy (38% and 24% respectively). Normally the lateral meniscus carries about 70% of the lateral compartment load and the medial meniscus carries 50% of the medial compartment load when the knee is fully extended.⁵¹ Loads increase up to threefold in the involved compartment after meniscectomy.⁵² Independent of the cause of osteoarthritis, cartilage degeneration is associated with a loss of glycosaminoglycans and progressive intolerance to compressive joint forces.⁵³ Articular cartilage becomes thinner and degenerates with superficial fraying, deep fissuring, ulceration, and full thickness loss of the joint surface. The subchondral bone also undergoes proliferative changes, including bone spur formation, cyst formation, and sclerosis.

7.1.1 Inflammatory arthritis: Cartilage thinning is uniform and diffuse, usually without focal defects, except where pannus erodes the cartilage and bone. Inflammatory changes occur in the subchondral bone, synovium and paraarticular soft tissues.

7.2 Definition: Osteoarthritis is a slow, progressive degeneration of articular cartilage of noninflammatory origin. Cole defined osteoarthritis as a spectrum of clinical entities, ranging from focal chondral defects to established arthrosis resulting from biologic and biomechanical hyaline cartilage failure.⁵⁴

7.3 Symptoms and Signs: Osteoarthritis (OA), previously called degenerative joint disease, is the most prevalent form of arthritis in the United States.⁵⁵ Clinically, patients with OA of the knee have pain in and around the knee that is typically worse with weight bearing and improved with rest, morning stiffness, and gel phenomenon. On physical examination,

patients often have tenderness to palpation, bony enlargement, crepitus on motion, and/or limitation of joint motion.

Unlike rheumatoid arthritis and other inflammatory arthritides, inflammation, if present, is usually mild. Although the causes of OA of the knee are not always known, biomechanical stresses affecting the articular cartilage and subchondral bone and biochemical changes in the articular cartilage and synovial membrane are important in its pathogenesis.⁵⁶⁻⁵⁸

TABLE 7.1. *American College of Rheumatology Classification Criteria for Osteoarthritis of the Knee*⁵⁹

Traditional format

Knee pain and radiographic osteophytes and at least 1 of the following 3 items:

- Age >50 years
- Morning stiffness \leq 30 minutes in duration
- Crepitus on motion

Classification tree

- Knee pain and radiographic osteophytes
 - Knee pain and age \geq 40 years and morning stiffness \leq 30 minutes in duration and crepitus on motion
-

Doubts have been expressed about the performance of the American College of Rheumatology (ACR) clinical classification criteria for osteoarthritis (Table 6.1) when applied in the general population. In a population-based cross-sectional study, 819 adults aged \geq 50 years who reported knee pain in the last 12 months were clinically assessed by research therapists using standardized protocols and blinded to radiographic status. Two hundred thirty-eight participants (30%) fulfilled the ACR clinical criteria for knee osteoarthritis. Agreement between the ACR clinical criteria and symptomatic radiographic knee osteoarthritis was low (sensitivity 41%; specificity 75%; positive predictive value 44%; negative predictive value 72%). Sensitivity and specificity did not vary markedly between population subgroups, although they were influenced by the underlying severity of radiographic osteoarthritis. Peat et al.⁶⁰ concluded that the ACR clinical criteria seem to

reflect later signs in advanced disease and other approaches may be needed to identify early, mild osteoarthritis.

Investigating the reliability of the American Knee Society (AKS) score (Figure 6.1), Liow⁶¹ found interobserver reference intervals at 16 points. The more experienced observers had greater intraobserver reproducibility, and they concluded that reliable use of the AKS score would necessitate repeated evaluation by an experienced observer.

American Knee Society Score

Pain (Maximum 50)

Walking

None	35
Mild or occasional	30
Moderate	15
Severe	0

Stairs

None	15
Mild or occasional	10
Moderate	5
Severe	0

R.O.M. (Maximum 25)

8°= 1 point

Stability (Maximum 25)

Medial/Lateral

0-5 mm	15
5-10 mm	10
> 10 mm	5

Anterior/Posterior

0-5 mm	10
5-10 mm	8
> 10 mm	5

Deductions

Extension lag

None 0	0
--------	---

	<4 degrees	-2	
	5-10 degrees	-5	
	>11 degrees	-10	
Flexion Contracture			<input type="text"/>
	< 5 degrees	0	
	6-10 degrees	-3	
	11-20 degrees	-5	
	> 20 degrees	-10	
Malalignment			<input type="text"/>
	5-10 degrees	0	
	More	(5° = -2 points)	
Pain at rest			<input type="text"/>
	Mild	-5	
	Moderate	-10	
	Severe	-15	
	Symptomatic plus objective	0	
Knee Score (Maximum: 100)			<input type="text"/>

FIGURE 7.1 American Knee Society Score

7.4. Pathology: Repetitive overloading with forces exceeding the biomechanical carrying capacity of articular cartilage from obesity or malalignment generally results in irreparable damage. Ideally, the knee is centered on the load-bearing axis (LBA), the hip-knee-ankle (HKA) angle shows zero deviation from linearity and the quadriceps-patella-tendon (Q angle) is aligned at 11° laterally. A common deformity is a varus knee (bow-leggedness), in which the knee center is lateral to the LBA. This can cause knee instability and subluxation when the ACL is lax. Subluxation is accentuated in the varus oblique knee in which the joint surfaces have an exaggerated inward tilt.^{62,63}

Arthritic knee patterns relate to alignment through cause or effect or both. In osteoarthritis (OA), varus knee degradation is focused medially.⁶⁴ Surveys of OA patients reveal that femoral deformity is responsible more frequently for the varus condition than is tibial

deformity. Valgus deformity (knock-knee) is uncommon in OA; it is linked to inflammatory arthritis, renal rickets, and various dysplasias.^{39, 65}

7.4.1 The role of diagnostic imaging modalities: The most accepted quantitative assessments of articular cartilage lesions are the Kellgren-Lawrence system⁶⁶ (Table 6.2) and the Ahlbäck⁶⁷ system to quantify the degree of joint space narrowing, subchondral sclerosis, osteophytes and cysts. Knee alignment is best assessed by radiography of the hip, knee, and ankle when the subject is standing upright to support body weight. Standardization of the subject's positioning is needed to get results that are reproducible, with special care to have the legs rotated correctly so that the flexion-extension plane is lined up in the anterior-posterior direction. The 45° flexion weight-bearing posteroanterior radiograph may demonstrate loss of joint space that is indicative of early chondrosis. Traditional extension views fail to show subtle joint space loss.⁶⁸

TABLE 7.2. Kellgren-Lawrence Osteoarthritis Scale

Grade	Description
0	None. No osteophytes
1	Doubtful
2	Minimal severity. Some osteophytes, possibly with narrowing, cysts, and sclerosis
3	Moderate severity. Definite osteophytes with moderate joint space narrowing
4	Severe. Large osteophytes and definite joint space narrowing

Staging of osteoarthritis (OA) of the knee is commonly based on the Ahlbäck⁶⁷ radiographic classification. A study comparing radiographic classification to visual inspection of the bone pieces removed during arthroplasty revealed an acceptable sensitivity in both medial OA (67-95%) and lateral OA (43-86%), but the specificity was low (medial: 11-67%; lateral: 25-75%). The authors concluded that conventional radiographs do not provide sufficient information for correct grading.⁶⁹

7.5 Findings at Arthroscopy: Arthroscopy does not play a role in the diagnosis or treatment of osteoarthritis of the knee.⁷⁰⁻⁷³ The arthroscopic findings can be graded according to the Outerbridge classification (see Table 5.3), but radiographic grading systems such as the Kellgren-Lawrence⁶⁶ and the Brandt⁷⁴ grading scales were equally effective in defining the presence of and estimating the severity of OA of the tibiofemoral joint.⁷⁵

7.6. Outcome Measures: The Western Ontario and McMaster Osteoarthritis Index (WOMAC) is widely accepted.^{76,77} The Knee Injury and Osteoarthritis Outcome Score (KOOS) was developed for younger and/or more active patients with knee injury and knee OA. KOOS is a valid, reliable, and responsive outcome measure in osteoarthritis. When compared to the WOMAC, the KOOS improved validity and may be at least as responsive.⁷⁸⁻⁸⁰ The IKDC Subjective Knee Evaluation Form¹²⁻¹⁴ and the Knee Outcomes Survey⁸¹ are also valid, reliable, and responsive outcome measures for osteoarthritis.

8. MENISCAL INJURIES

8.1. Introduction: Arthroscopic treatment of meniscal injuries is one of the most common orthopedic surgical procedures. Although our knowledge and understanding of the anatomy, function, and treatment of meniscal pathology has evolved, meniscal tears continue to cause significant symptoms and long-term impairment of the knee. Therefore, it is important for the treating physician to have a full understanding of the anatomy and biomechanics of menisci, symptoms of meniscal tears, findings at arthroscopy, techniques for surgical treatment, and outcome measures for reporting results.

8.1.1 Anatomy: The menisci are semilunar shaped structures on the medial and lateral sides of the knee. They are composed of collagen and cells of either fibroblast or chondrocyte origin. Water accounts for 75% of the weight of the menisci. The organic matrix is composed of three-quarters collagen, with type 1 collagen predominating. The collagen fibers are oriented in a characteristic fashion. The most superficial fibers are oriented radially. Most of the collagen fibers, however, are found in the deep layer and are arranged in a circumferential orientation, following the periphery. The radial fibers are woven between the circumferential fibers, which helps to provide structural integrity. The arrangement of fibers enables them to resist the hoop stresses that are produced in the meniscus during weight bearing.⁸²⁻⁸⁴ In cross section, the menisci are triangular, being thicker at the periphery and tapering to a thin free edge centrally. The superior surfaces are concave to accommodate the convexity of

the femoral condyles. The medial meniscus is semilunar in shape and is thinner and narrower anteriorly. The posterior horn is thicker and wider, averaging approximately 10.6 mm. The anterior and posterior horns are attached to the intercondylar eminence with an additional slip from the posterior horn attaching to the posterior cruciate ligament. The peripheral circumference is firmly attached to the capsule by the coronary ligaments. The medial meniscus is also firmly attached to the posterior oblique ligament. The medial meniscus covers approximately 64% of the medial tibial plateau. The lateral meniscus covers approximately 84% of the lateral tibial plateau. It is more circular than the medial meniscus and is also more uniform in width (average 12 to 13 mm).

The anterior and posterior horns of the lateral meniscus also attach to the intercondylar eminence, but in closer proximity to the ACL than the medial meniscus. The peripheral attachment of the lateral meniscus to the capsule is thinner and looser than on the medial side. In addition, there is no attachment in the region of the popliteal hiatus, and there is no attachment of the lateral meniscus to the lateral collateral ligament.^{82,87}

Vascular Anatomy

The lateral, medial, and middle geniculate arteries form a parameniscal capillary plexus along the entire rim of the menisci. The blood supply consists of vessels that are circumferential with radial branches that end in small capillary loops. Prenatally, the vessels transverse the entire meniscus, but the vascularity decreases because of vertical load bearing.

In the adult, the capillary loop extends no deeper than 10 to 25% of the width of the lateral meniscus and 10 to 30% of the width of the medial meniscus. The lateral meniscus in front of the popliteus tendon is avascular.^{88,89} The vascular synovial tissue is important in meniscal healing. Rasping these tissues can stimulate vascular overgrowth into the menisci and cause migration of pluripotential mesenchymal cells that play a role in healing after meniscal repair.

8.2 Biomechanics

Load Transmission

The medial meniscus transmits 50% of the load on the medial side and the lateral meniscus transmits 70% of the load on the lateral side in extension. At 90° of flexion the lateral meniscus transmits 85% of the load.⁹⁰⁻⁹¹ The ability of the menisci to transmit load comes from their shape, which bridges the incongruity between the spherical femoral condyles and flat tibial plateau.⁹²

Partial meniscectomy of the inner third of the menisci decreases contact area by 10% and increases peak load by 65%. A total meniscectomy decreases contact area by 75% and increases peak load by 235%.⁹³

Shock Absorption

The intact menisci can dissipate forces because they are biphasic structures. The solid phase consists of proteoglycans and collagen and the liquid phase is water. They function under compression similar to articular cartilage. The unique architecture allows transmission of vertical forces into tangential and radial forces as the menisci are loaded. For a meniscus to provide a protective function, it must have structural continuity throughout. Loss of the menisci results in a 20% reduction in shock absorption.⁹⁴

Joint Lubrication

The biphasic composition of the meniscus is also important for joint lubrication. Water is forced out of the menisci and into the joint space with compression; the water is reabsorbed when the load is removed. This sponge phenomenon not only dissipates force but also circulates cellular nutrients. There is a 20% increase in the friction coefficient after meniscectomy.^{85,86}

Joint Stability

The medial meniscus plays an important function in joint stability. Markolf⁹⁵ found that after medial meniscectomy there is an increase in anteroposterior translation at 90° of 1.82 mm in varus-valgus translation and flexion is increased 1.36 mm. In the ACL-deficient knee, elasticity after medial meniscectomy is increased 18% to 20% in extension and 58% at 90° of flexion. In addition, in situ graft forces in an ACL replacement graft are increased by 33% to 50% after a medial meniscectomy.

8.3. Symptoms and Signs:

8.3.1. Acute meniscal tear: A patient with a recent meniscal tear typically presents with complaints of pain and swelling. Physical examination often reveals tenderness over the joint line, a decreased range of motion, and an effusion.

8.3.2 Chronic meniscal tear: A patient who has had a meniscal tear for several months or longer typically presents with intermittent pain, catching, or locking symptoms. Physical examination also reveals tenderness over the joint line, pain with forced hyperflexion, and occasionally muscle atrophy and effusion.

8.4 Pathology: Meniscal tears can be either traumatic or degenerative in nature. Meniscal tears are uncommon in persons under 10 years of age, but become increasingly common during and after adolescence. Degenerative tears can be found in as much as 60% of people over age 65. The majority of these tears, however, are asymptomatic and occur in association with degenerative joint disease. The changing patterns of meniscal injury with chronological age most likely correlate with alteration in collagen fiber orientation with aging, as well as with increasing intrasubstance degeneration.

MRI can be used to confirm the clinical diagnosis. The accuracy rates of MRI range from 92% to 98%. MRI can also be used to evaluate meniscal degeneration. A grading system has been developed to describe abnormal intrameniscal signal: Grade 1 is oval or globular in appearance and does not communicate with any meniscal surface. A Grade 2 signal is more linear, but similarly does not communicate with the articular surfaces. Grade 3 signals within the meniscus are linear and communicate with either superior or inferior articular surfaces. Grades 1 and 2 signals are consistent with intrasubstance myxoid degeneration, whereas a Grade 3 signal is consistent with a tear.

8.5. Findings at Arthroscopy

8.5.1 Development of standard terminology: Knee surgeons have been aware of the difficulty created by absence of standard terminology and classification of meniscal pathology.

Consequently, under the auspices of the International Society of Arthroscopy, Knee Surgery, and Orthopaedic Sports Medicine (ISAKOS) Knee Committee, a meniscal documentation committee was formed in 2006 to develop and adopt a standardized international meniscal documentation system. The meniscal documentation committee, consisting of experts from the continental sports medicine societies, adopted the following terms and definitions:

I. Tear length

Tear length indicates the length of the meniscal tear that reaches the surface of the meniscus. It does not include contained tears (MRI Grade II) that do not reach the surface of the meniscus.

II. Tear depth

Tear depth mirrors the MRI classification of 0 to 3. A tear depth of 3A is a partial tear that extends through either the superior or inferior surface. A horizontal tear may also be a 3A partial tear. A 3B tear is a complete tear that extends through both the superior and inferior surfaces of the meniscus.

III. Location

The committee adopted a zone classification system. Zone 1 includes tears at the meniscosynovial junction and tears with a rim width <3 mm. Zone 2 tears have a rim width of 3 to <5 mm. Zone 3 tears have a rim width ≥ 5 mm.

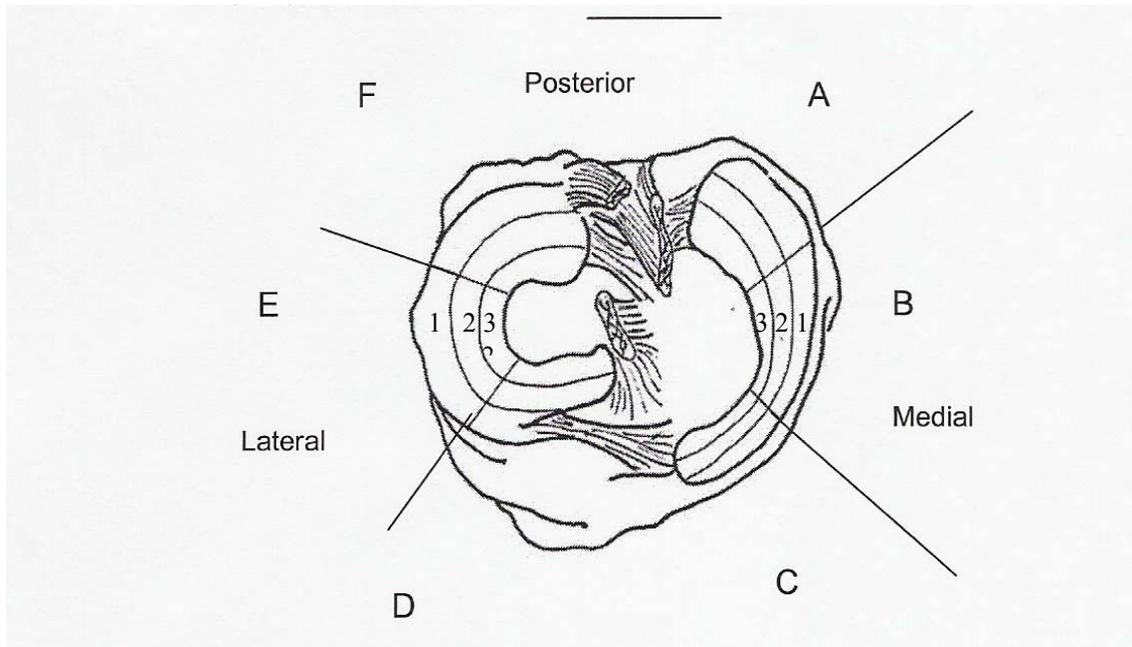
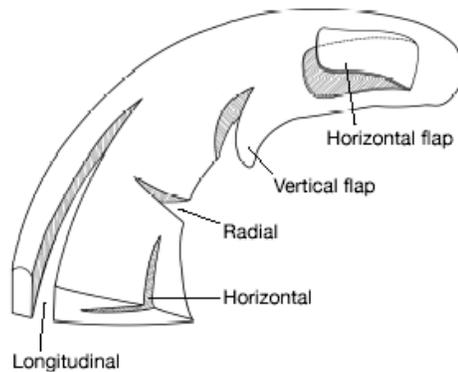


FIGURE 8.1. The Meniscal Zone Classification System

The committee discourages the use of the terms red-red, red-white and white-white to describe the zone of meniscal tears. The vascular supply of the menisci varies and therefore cannot be precisely determined arthroscopically by meniscal tear rim width.

IV. Tear pattern

The committee agreed to the use of a set of terms to describe different types of meniscal



tears.

FIGURE 8. Terms for meniscal tears as defined by the ISAKOS Meniscal Documentation Committee.

Longitudinal-vertical tear

The longitudinal-vertical tear as pictured may be located anywhere along the meniscus. The extension of this tear may result in a bucket-handle tear.

Horizontal tear

The horizontal tear begins at the inner margin of the meniscus and extends toward the capsule.

Radial tear

The radial tear also begins at the inner margin and extends towards the capsule. This type of tear is typically located at the junction of the middle and posterior thirds of the lateral meniscus. These tears may extend completely through the meniscal rim, transecting the meniscus.

Flap tear

A flap tear may be either vertical or horizontal. The vertical flap tear extends through both the inferior and superior surfaces of the meniscus. The horizontal flap tear is an extension of the horizontal tear. Either the inferior or superior surface the meniscus may remain intact in a horizontal flap tear.

Complex tear

This term describes complex patterns that demonstrate tearing in several planes.

Discoid meniscus

The discoid meniscus is a congenital variance that usually occurs laterally. Watanabe⁹⁶ classified this abnormality into three types. The incomplete discoid type is larger than a normal meniscus and has normal attachments. The complete discoid type covers the entire tibial plateau, but also maintains normal attachment. The third type of discoid meniscus lacks a posterior capsular attachment and is more often symptomatic than the other two types.

Three large reviews have documented the relative incidence of various meniscal tear patterns (Table 8.1).

TABLE 8.1. Relative Incidence of Various Meniscal Tear Patterns

Type of Tear	R. Metcalf⁹⁷	G. Poehling⁹⁸	French Society of Arthroscopy (SFA) 99-101
Oblique/flap	45	21	28.5
Vertical longitudinal	36	26	41
Peripheral			
All Types			
Degenerative or complex	12	30	21
Radial	3	9	3
Horizontal	3	12	6.5
Discoid	1	1	

Meniscus cysts commonly occur in the peripheral portion of the middle third of the lateral meniscus. These cysts are in direct continuity with the meniscus.

8.6 Outcome Measures: The disease-specific outcome measures that have been validated for evaluation of meniscal treatment include the Lysholm,³⁵ Cincinnati,¹⁰² and Mohtadi¹⁰³ scales. The region-specific scales that have been validated for evaluation of meniscal injury include the IKDC Subjective Knee Evaluation Form,¹²⁻¹³ the Knee Outcomes Survey,⁸¹ and the KOOS.⁷⁸⁻⁸⁰

Although any of these forms may be used, ISAKOS has formally adopted the IKDC system. Consequently, the IKDC Current Health Assessment (SF-36),¹¹ Subjective Knee Evaluation Form,¹²⁻¹³ and Knee Examination Form¹⁴ are recommended to evaluate the results of treatment for meniscal injuries.

9. INSTABILITY

9.1 Introduction: Historically, the most difficult problems in comparing outcomes of different methods of treatment for knee ligament injuries were due to the absence of generally accepted terminology and the use of outcome instruments that were neither valid nor comparable.

Under the auspices of the American Orthopaedic Society for Sports Medicine (AOSSM) and the European Society of Sports Traumatology Knee Surgery and Arthroscopy (ESSKA), the IKDC was formed to standardize terminology and develop subjective and objective documentation systems. An international group of surgeons developed a consensus on terminology and the IKDC forms were developed and revised to include the advances in the science of outcomes measurement.¹⁰⁴⁻¹⁰⁵ These forms were officially adopted by AOSSM, ESSKA, and ISAKOS.

9.2 Definition:

Development of Standard Terminology

The discrepancy in the implied meaning of terms used in the literature has been an impediment to international communication. To improve communication, the committee critiqued, revised, and adopted a standard set of definitions. The following definitions are among those adopted:¹⁰⁴⁻¹⁰⁵

Motion: The act or process of changing position. Motion is described as the rate and direction of change.

Displacement: The net effect of motion; a change in position between two points without regard to the path followed. Displacement may be described by a change in translation or in rotation, each of which has 3 degrees of freedom.

Translation: Motion of a rigid body in which all lines remain parallel to their original orientation. By convention, knee translation is described as motion of the tibia relative to the femur. Translation of the tibia may be mediolateral, anteroposterior, or proximodistal. Translation is measured in millimeters. The reference point normally used to measure translation is midway between the medial and lateral margins of the joint.

Rotation: A type of motion or displacement in which all points move about an axis. Rotations of the knee may be flexion-extension, internal-external, and abduction-adduction.

Range of motion: The displacement occurring between two limits of movement for each degree of freedom. Range of motion does not indicate the extremes of motion. For motion other than flexion-extension, range of motion depends upon the angle of knee flexion.

Limits of knee motion: The extreme positions of movement possible for each of the 6 degrees of freedom. The term limits of knee motion is more specific than range of motion. It indicates where motion begins and ends and includes range of motion. There are 12 limits of motion, 2 for each 6 degrees of freedom. Ligament injury increases the limits of knee motion. The IKDC knee examination describes the limits of flexion and extension with 3 numbers: the maximum extension, neutral position, and flexion.

Coupled motions: A displacement or motion in one or more degrees of freedom caused by a load applied in another degree of freedom. Coupled motions occur during the clinical examination. An anterior displacement force applied during the Lachman test causes anterior translation and internal rotation of the tibia. A posterior displacement force results in posterior translation and external rotation. The amount of motion depends on the force applied and the constraints of the coupled motion. For example, constraint of rotation during the Lachman test significantly diminishes anterior translation.

Laxity: A lack of tension; looseness, referring to a normal or abnormal range of motion. In the first context, laxity is used to describe a lack of tension in a ligament, and in the second, as looseness of a joint. This ambiguous term should be used to indicate lack of tension in the ligament. The degree of laxity should be specified as either normal or abnormal. Laxity should not be used in the context of looseness of a joint; the motion should be specified. The term anterior translation is preferable to anterior joint laxity.

Instability is another ambiguous term that has been used in two ways. First, it is used to describe the symptoms of giving way, and second, to describe increased joint motion. Rather

than use instability to refer to symptoms, it is preferable to describe the event (i.e., giving way with activity). It is incorrect to designate a specific anatomic structure as the cause of ACL instability. Instability should only be used in the general sense to indicate excessive motion of the tibia as the result of traumatic injury.

9.3. Symptoms and signs: The symptoms and impairments caused by ligament injury are variable and difficult to quantitate. The symptoms of pain, swelling, and giving way that frequently occur may be evaluated with a subjective outcome instrument that has been validated for knee ligament injuries.

9.3.1 Limits of knee motion evaluation: The methods of examination that have been used to determine the limits of knee motion are qualitative and clinical-specific. Even experienced examiners can produce and perceive appreciable differences in displacement. Accurate assessment of translation and rotation is more demanding in ligament injuries that increase more than one limit of motion. In these circumstances, clinicians have difficulty identifying either the starting or ending positions for the tibia.

The IKDC determined the validity of the clinical tests and conditions for testing. The consensus was that reproducibility depends upon specifying the conditions of the tests. Clinical and laboratory studies confirm that the position of the knee at the initiation of testing affects displacement. The site of measurement must be identified and the magnitude, direction, and point of application of force should be specified. Measurements in translation should be reported in millimeters and rotation in degrees. Changes in any of these conditions will result in different interpretations of the tests.¹⁰⁴

The Lachman test, total anteroposterior translation at 70° degrees, and medial and lateral joint opening may be assessed with manual, instrumented, or stress radiograph examination. Only one method of assessing these tests should be graded, preferably a "measure displacement." Displacement with instrumented examination of both knees is recorded with a force of 134 newtons (30 lbs) and the maximum manual test. Only the measured displacement at the standard 134 newtons is used for grading. The numerical values for side-to-side differences are rounded off and recorded on the IKDC Knee Examination Form

The end point is assessed in the Lachman test. The end point affects the grading of the IKDC form when the index knee has 3.5 mm more anterior laxity than the normal knee. In this case, a soft end point results in an abnormal grade rather than a nearly normal grade.

The 70° degree posterior sag is estimated by comparing the profile of the injured knee to the normal knee and palpating the medial femoral-tibial step-off. It may be confirmed by noting whether contraction of the quadriceps pulls the tibia anteriorly.

The external rotation tests are performed with the patient prone and the knee flexed to 30° and 70°. Equal external rotational torque is applied to both feet and the degree of external rotation is recorded.

9.3.2 The pivot shift tests: Examiners constrain knee motion when performing the pivot shift test. The coupled knee motions of anterior translation and internal tibial rotation are induced to produce anterior subluxation. The pivot shift and reverse pivot shift are performed with the patient supine, with the hip in 10° to 20° of abduction and the tibia in neutral rotation.. The greatest subluxation, compared to the normal knee, should be recorded. The variability between examiners indicates that the pivot shift test can only be considered a qualitative test. Presently, in vivo measurement devices are not available to quantitate displacement in mm. Consequently, the pivot shift test should be graded in the following way: negative; 1+, glide; 2+, clunk; 3+, gross.

In summary, even under the best circumstances, large variations can exist in clinicians' estimates of displacement. Consequently, instrumented or stress radiography measurements should be used to report quantitative instrumented or radiographic measurements of the Lachman test at 25° of flexion, total anteroposterior translation at 70° of flexion, and medial and lateral joint opening at 20° of flexion.

9.4 Pathology

9.4.1 Compartment and radiographic findings: Restoration of stability and prevention of degenerative changes are long-term goals of knee ligament surgery. Evaluation of success in attaining these goals is difficult. Early degenerative changes cannot be accurately evaluated without visual inspection, and radiographic changes occur late in the course of traumatic arthritis. Assessment of crepitation is used to detect early compartment changes.

Unfortunately, only limited conclusions can be drawn from the evaluation of crepitation. The collection of data is subject to bias and crepitation may not indicate articular cartilage abnormality.

A bilateral, double-leg posteroanterior weight-bearing radiograph at 35° to 45° degrees of flexion (tunnel view) is used to evaluate narrowing of the medial and lateral joint spaces. The

Merchant view at 45° degrees is used to document patellofemoral narrowing. Radiographic changes are also qualitatively graded. A mild grade indicates minimal changes (i.e., small osteophytes, slight sclerosis, or flattening of the femoral condyles), and narrowing of the joint space, which is just detectable. A moderate grade may have those changes and joint space narrowing (e.g., a joint space of 2 to 4 mm or up to 50% joint space narrowing). Severe changes include a joint space of less than 2 mm or greater than 50% joint space narrowing. The evaluation of compartment and radiographic findings are qualitative and influenced by investigator bias.

9.5 Findings at Arthroscopy: Arthroscopy is not usually needed to make the diagnosis of knee ligament injury. An exception may be a partial tear of the ACL, which may be more fully evaluated with arthroscopic probing.

9.6 Outcome Measures: The disease-specific scales that have been validated for evaluation of treatment for knee ligament injuries included the Lysholm,³⁵ Cincinnati,¹⁰² and Mohtadi¹⁰³ scales. The region-specific scales that have been validated for evaluation of ligament injury include the IKDC Subjective Knee Evaluation Form,^{12-13,14} the Knee Outcomes Survey,⁸¹ and the KOOS.⁷⁸⁻⁸⁰

ISAKOS has formally adopted the IKDC system. Consequently, the IKDC Current Health Assessment (SF-36),¹¹ Subjective Knee Evaluation Form,^{12,13} and Knee Examination Form¹⁴ are recommended.

10. PATELLOFEMORAL DISORDERS

10.1. Introduction: Patellofemoral disorders are a group of very different lesions, symptom complexes, or multifaceted disabilities. The term has been used to cover different entities like anterior knee pain, patello-femoral chondropathy, patello-femoral dysplasia or combinations. Anterior knee pain is not a well described entity but rather a diagnosis describing a condition—not a lesion or an illness. The condition occasionally can be linked to malalignment, patellar tendinopathy or Osgood-Schlatter disease. Chondromalacia is not correct terminology and has too many negative connotations. Chondritis implies an inflammation, which is not present. Chondropathy is a term better suited to metabolic disorders.

There are many parameters to consider in classifying patellofemoral disorders: the status of the patellofemoral articular surface, the three-dimensional position of the patella relative to

the trochlea, abnormalities of the extensor mechanism above and below the patella, and structural abnormalities of the patellofemoral joint.

10.2 Definition: The patellofemoral disorders can be divided into the following:

- Patellofemoral pain with no instability and no pathology demonstrated at arthroscopy
- Patellofemoral pain with subjective and/or objective mechanical instability
- Patellofemoral pain with no instability but a chondral lesion of the patellofemoral joint
- Patellofemoral instability that can be either atraumatic or posttraumatic with no pain
- Patellofemoral chondral disorders with no pain or instability (the asymptomatic knee)

Here we will deal only with patellofemoral instability, posttraumatic or atraumatic, with and without pain.

10.3. Symptoms and Signs: Traumatic first-time patella dislocation is a severe injury that will lead to disability in the form of patella instability, recurrent dislocations, anterior knee pain, and even patellofemoral osteoarthritis in more than half of the patients if untreated or treated nonoperatively.¹⁰⁶⁻¹¹⁰ The natural history of nonoperatively treated patellar dislocation includes redislocation in 1 in 6 cases, other residual symptoms in 2 in 6 cases; 3 in 6 will be asymptomatic.¹¹¹

Atraumatic instability is often linked to malalignment causing dyskinesia and pain.

Patellofemoral incongruity is only one cause of instability, although trochlear dysplasia (shallowness of the femoral sulcus) is often associated with patellar stability. Lateral trochlear dysplasia may contribute to chronic lateral tracking of the patella thereby exacerbating pre-existing instability.

Schutzer¹¹² has the only validated clinical classification system currently available. Hinton and Sharma¹¹³ found the following questions useful in classifying patellofemoral stability

1. Is the situation one of a mechanically normal knee subjected to misuse/overuse, or does the patient have significant underlying mechanical risk factors?
2. Is the problem an aggravation or disability gauged by lost practice time, presence of quadriceps atrophy, or loss of explosive ability?

3. Has there been adequate compliant nonoperative care? If so, what was the response?
4. Are there signs or symptoms of concurrent knee injury, such as osteochondral fracture, extensor mechanism overuse, or undiagnosed ligamentous injuries?
5. Is the instability acute, recurrent, or chronic?
6. Is the patella grossly unstable during clinical examination or daily activities?
7. What are the patient's age, activity level, and athletic potential?

10.4 Pathology: Radiographic, MRI and CT imaging provides important information about the anatomical factors that may predispose to patellofemoral instability. Radiographs to evaluate the patellofemoral joint include a standard anteroposterior view, 45 degree PA weight bearing view, true lateral view at 20° of flexion and an axial view at 30° of flexion. The Insall-Salvati index,¹¹⁴ which compares the length of the patella to the length of the patella tendon, has limited application because the length of the patella and the length of the tendon do not necessarily correspond to how the patella articulates with the trochlea. The Blackburne-Peel index¹¹⁵ provides a more useful measure of the relationship of the patella to the trochlea; it relates the length of the articular surface of the patella to the distance of the patella's inferior margin from the tibial plateau. A consistent relation of patella height to risk of patella instability has not been established.

Trochlear dysplasia can be demonstrated in a standard true lateral view when the trochlear groove crosses both femoral condyles (the crossing sign).¹¹⁶⁻¹¹⁷ The shape of the trochlea can be classified according to the Dejour classification system:

Type 1. The crossing is symmetric at the upper area of the trochlea

Type 2. The crossing is asymmetric, first with the medial condyle, and then above with the lateral condyle

Type 3. The crossing occurs at the lower area of both condyles

Intermediate trochleae

Type a1. The trochlear groove ends near the anterior border of the condyles but without crossing

Type b2. The trochlear groove crosses the anterior border of the medial condyle, but does not cross the lateral condyle

The Dejour classification system has been studied for reproducibility, and the interobserver and intraobserver agreement was only slight to fair.¹¹⁸⁻¹¹⁹ The researchers who evaluated the Dejour classification system found that the reliability of the system improved when the Type

2 classification was changed to only include 5 mm or more a between the intersections with the medial and lateral femoral condyles. Trochlea dysplasia however is only one factor that may predispose to patellofemoral instability. Patients should be evaluated for other predisposing factors, including an abnormal tibial tubercle-trochlear groove (TT-TG) relationship, medial patellofemoral ligament deficiency, and functional factors (such as excessive internal hip rotation, patella alta, and ligamentous laxity). In general, these inter-related factors may create a pathologic condition that is unique to each patient.

MRI, CT scan, and ultrasound can provide reliable information about the trochlear anatomy. The sulcus angle is measured between a line drawn from the lateral condyle to the trochlea and a line drawn from the trochlea to the medial condyle with the knee in 15° of flexion at the widest part of the patella. Most important, however, is the TT-TG relationship as described by Neyret.¹²⁰

The lateral patellofemoral angle is measured between a line drawn through the two condyles and a line drawn through the apex of the patella and the lateral patella joint surface. The angle is abnormal if the angle does not open laterally.

Schutzer¹¹² has compared CT data in patients with symptoms caused by patellofemoral instability and normal subjects. He found abnormal tilt and subluxation of the patella at 15° degrees knee of flexion in the symptomatic patellofemoral patients.

10.5 Findings at Arthroscopy: Arthroscopy is not needed to diagnose patellar instability. The articular cartilage of the patella and trochlea can be evaluated with arthroscopy and classified according to ICRS criteria.

10.6 Outcome measures: The IKDC Subjective Knee Form^{12,14} and the Knee Outcomes Survey⁸¹ have been validated for patellofemoral pathology.

Knee Outcome Measures

The validity of a study (i.e., the degree to which one can make cause-effect conclusions) depends upon the design and implementation of the study and the validity of the outcomes instrument used in the study. A crucial feature of outcomes research is selection of an outcomes instrument that has been validated for its intended use. It is also important for an outcomes instrument to be validly translated into the language of the people using it. Health-related quality-of-life measures are of two types: general and specific.

General measures are applicable to diverse populations. They usually measure multiple aspects of health status, including physical, emotional, and social well being. The SF-36 is the

most common example of a general measure. The advantage of general health measures is that they permit comparisons across populations with different health conditions. General health measures are also more likely to detect unexpected effects of intervention. The disadvantage of general health measures is that they are less responsive to specific measures of health status. They are also susceptible to ceiling effects.

Specific health measures focus on aspects that are specific to the conditions or populations of interest. Specific measures include only those aspects of health-related quality of life that are relevant to the condition or population being studied. The advantage of specific health measures is improved responsiveness. They tend to be short and easy to interpret, and the content is more relevant to the particular condition. The disadvantages are that they do not measure all aspects of health status and do not allow for comparison between different disease stages or populations. Specific measures include disease- and region-specific scales. The disease-specific scales that have been validated for evaluation of ligament pathology include the Lysholm,³⁵ Cincinnati¹⁰² and Mohtadi¹⁰³ scales. The Lysholm and Cincinnati scales have also been validated for evaluation of meniscal pathology. The WOMAC^{76,77} is a disease-specific scale that has been validated for osteoarthritis. The region-specific scales that have been validated include the IKDC Subjective Knee Evaluation Form,¹²⁻¹⁴ the Knee Outcomes Survey,⁸¹ and the KOOS.⁷⁸⁻⁸⁰ The IKDC Subjective Knee Evaluation Form and Knee Outcomes Survey are valid for ligament injury, meniscal injury, patellofemoral pain and osteoarthritis. The KOOS has been validated for ACL insufficiency, meniscus tears, and cartilage damage.⁸¹

Outcomes instruments that are disease-specific may not be appropriate for evaluation of treatment outcomes when another condition, other than what the outcomes instrument was validated for, occurs concomitantly. A single form that is valid for patients with a variety of conditions affecting the knee simplifies data collection because the same form may be used for all patients. The alternative would be to use multiple forms. Although any of the above-mentioned validated forms can be used, ISAKOS formally adopted the IKDC forms. Consequently, a general health measure, the IKDC Current Health Assessment (SF-36) and the region-specific IKDC Subjective Knee Evaluation Form are recommended for the evaluation of knee pathology. The IKDC Knee Examination Form can be used to document objective results.

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