Lecture: Shoulder Basic Science: Biomechanics of the Shoulder Stabilizers
Monday, March 10, 2003 • Aotea Centre, Kupe/Hauraki Room

Eiji Itoi, MD
Professor and Chairman
Department of Orthopedic Surgery
Akita University School of Medicine
Akita, Japan

KUPE/HAURAKI

Introduction

Shoulder stability depends on static and dynamic stabilizers of the shoulder. The static stabilizers include the capsuloligamentous structures such as the glenohumeral ligaments, the intra-articular negative pressure, glenoid concavity and the labrum, inclination of the scapula, etc. The dynamic stabilizers include all the muscles around the shoulder, but mainly the rotator cuff muscles, the deltoid, and the biceps. When one or combination of some of these stabilizers is damaged, then the shoulder stability is impaired. I would like to focus on some of the stabilizers related to recurrent dislocation of the shoulder.

Glenohumeral Ligaments

The inferior glenohumeral ligament is known to be the essential stabilizer to prevent anterior dislocation of the shoulder. Strain measurement of the IGHL complex revealed that not only the anterior band of IGHL but also the axillary pouch (6 o'clock position of the capsule) showed the greatest strain with the arm in abduction and external rotation. The posterior band on the other hand showed the greatest strain with the arm in flexion and internal rotation, suggesting its role as a posterior stabilizer (1).

Anterior dislocation of the shoulder usually causes detachment of the IGHL complex from the glenoid, known as a Bankart lesion. It is known from our experience that the more the shoulder dislocates, the more elongated the anteroinferior capsule. This has not been confirmed in the clinical setting. We measured the length of the capsule on MRI taken after injecting Gd into the glenohumeral joint. The anteroinferior and inferior portions of the shoulder capsule were elongated an average 19% in shoulders with recurrent anterior dislocation (2).

Glenoid Concavity

The glenoid socket also provides stability to the shoulder. When dislocation occurs, the humeral head needs to override the rim of the glenoid. Therefore, the deeper the glenoid socket, the more stable the shoulder. Fracture or erosion of the anteroinferior rim of the glenoid is sometimes observed in recurrent anterior dislocation of the shoulder. How much bony defect is acceptable without bone grafting when performing Bankart repair? We created osteotomy of the anteroinferior rim of the glenoid stepwise and measured the force necessary to translate the humeral head in anteroinferior direction after the Bankart lesion was repaired. When we created an osteotomy with the width equal to or greater than 21% of the glenoid length, both the stability and the range of motion were significantly decreased. Therefore, a bony defect whose width is greater than 21% of the glenoid length needs to be bone grafted when repairing the Bankart lesion (3).

This "critical size" of the bony defect needs to be determined in the clinical setting. We used plain x-rays (axillary view and West Point view) and CT images to evaluate how the critical size appeared in these images. Surprisingly, the axillary view revealed almost nothing. The West Point view demonstrated the critical size as a 20% defect of the glenoid socket. The CT image at the inferior 1/4 level of the glenoid showed the critical size as a 50% defect of the glenoid socket. These views are helpful in making a decision whether the bone grafting is necessary (4).

References

1. Urayama M, Itoi E, Hatakeyama Y, Pradhan RL, Sato K: Function of the 3 portions of the inferior glenohumeral ligament: A cadaveric study. J Shoulder Elbow Surg 2001; 10: 589-594.
2. Urayama M, Itoi E, Sashi R, Minagawa H, Sato K: Capsular elongation in shoulders with

recurrent anterior dislocation: quantitative assessment with magnetic resonance arthrography. Am J Sports Med 2003; 31: 64-7.
3. Itoi E, Lee SB, Berglund LJ, Berge LL, An KN: The effect of a glenoid defect on anteroinferior stability of the shoulder after Bankart repair: a cadaveric study. J Bone Joint Surg [Am] 2000; 82-A: 35-46.
4. Itoi E, Lee SB, Amrami KK, Wenger DE, An KN. Quantitative assessment of classic anteroinferior bony bankart lesions by radiography and computed tomography. Am J Sports Med 2003; 31: 112-8.

Lecture: Shoulder Anatomy
Monday, March 10, 2003 • Aotea Centre, Kupe/Hauraki Room

Glenn C. Terry, MD, USA

NOTES

Lecture: MR Imaging of the Shoulder
Monday, March 10, 2003 • Aotea Centre, Kupe/Hauraki Room

Gregory R. Applegate, M.D.

I: PRINCIPLES OF MAGNETIC RESONANCE IMAGING

Following clinical examination, routine radiography remains the initial screening study of choice when imaging the shoulder. Magnetic Resonance Imaging, however, is proving to be the imaging study of choice for further evaluation due to MRI's superb ability to evaluate soft tissues of the rotator cuff, glenohumeral articulation, tendons, as well as evaluation of radiographically occult conditions. The advantages of MRI include the lack of ionizing radiation, the ability to scan clearly in a multi-plantar fashion, and the superb soft tissue contrast. MRI has the ability to distinguish fat, ligamentous structures, tendons, bone marrow, and fluid. Excellent high resolution imaging of the shoulder and surrounding soft tissues can be easily obtained.

The magnetic resonance phenomenon involves an atomic nucleus. Usually hydrogen nuclei are imaged, consisting of a single proton, which are most abundant in water. A strong magnetic field is used to align these protons uniformly. A frequency selective pulse can quickly be applied that changes the orientation of the aligned protons. After the pulse, the protons relax back to their initial state. This relaxation process may be described by two time characteristics: T1 and T2. We can emphasize either one of these two characteristics by altering two variables: TE(echo time) and TR(repletion time). Images with a short TE and TR are relatively T1 weighted; and those with long TE and TR are T2 weighted. The third type of image is a "balanced", or intermediate-weighted image with a long TR and short TE, also referred to as proton density weighted image. These characteristics apply to spin echo imaging, the most frequently used sequence. There are other modifications to these sequences, which are of value for imaging the shoulder including fat saturation sequences. On proton density or T2 weighted sequences with fat saturation, fluid will be bright and fat will be dark, enhancing the contrast between fluid and adjacent fat.

II: QUALITY

In MRI, quality makes the difference, as all MRI's are not created equally. There is a tremendous variability in the quality of MRI studies currently being performed. Various factors contribute to MRI quality including the equipment, protocols and radiologic interpretation.

A: EQUIPMENT issues include the field strength of the magnet, the software and coils. Currently **1.5 Tesla** is the highest field strength routinely available for clinical imaging. The signal, which is used to generate the MR image, is directly proportional to field strength. Therefore, a high field strength magnet can help to optimize imaging of the shoulder. A dedicated shoulder coil must also be utilized. The coil acts as an antenna to receive the signal from the local anatomic part being imaged. Therefore a coil specifically engineered and designed for imaging of the shoulder is mandatory to optimize image quality. Currently **phased array coils** offer the optimal signal to noise ratio, further enhancing image quality.

B. MRI SHOULDER PROTOCOL. To optimize image quality, a field of view of 12 to 16 cm is utilized. We utilize thin 3-4 mm sections in three planes. Both proton density and T2 weighted (T2W) information is needed in all three planes. On a T2W image, fluid, edema and tears are of increased signal intensity (white). Additionally fat saturation techniques may be utilized to improve depiction of osseous contusional injuries. The shoulder is positioned with the arm in a neutral or slightly external rotated position. The radiologist and technologists must work as a team to ensure that the protocol is optimized to thoroughly evaluate the shoulder. The axial sequences must include the top of the acromioclavicular joint and extend well below the glenohumeral joint. From the axial images the oblique coronal plane is selected along the anterior leading edge of the supraspinatus or the central tendinous band of the supraspinatus. The oblique sagittal images are selected perpendicular to the oblique coronal images and must extend medial to the glenohumeral joint and well lateral to the rotator cuff insertion site .The average high-quality MR examination of the shoulder currently at can be scheduled in approximately 45 minutes.

C. THE RADIOLOGIC INTERPRETATION plays a large part in overall MRI quality. It is difficult to quantitate expertise in shoulder MR interpretation; however formal fellowship training in MRI or musculoskeletal imaging is helpful. A large experience in shoulder imaging with clinical feedback is helpful. VIEW YOUR RADIOLOGIST AS A CONSULTANT AND SEND THEM A COPY OF ALL RELATIVE OPERATIVE REPORTS.

III: INDICATIONS

Regarding the rotator cuff, MRI is useful in the evaluation of rotator cuff tears, and the evaluation of tendinitis/tendinosis/tendinopathy. MRI is also helpful in evaluation of the post-operative rotator cuff.

For shoulder imaging in instability, Acute Classic TUBS (traumatic, unidirectional, Bankart, surgery) category patients may not require imaging. Except after acute injury most patients have little native joint fluid. In the non-distended glenohumeral joint capsulo-ligamentous structures are contiguous with the labrum making imaging assessment more difficult. MR is useful if the diagnosis of instability is uncertain, in multi-directional instability or if there may be accompanying pathology including rotator cuff pathology, biceps tendon pathology, SLAP lesions and isolated ligamentous or capsular injuries.

IV: NORMAL ANATOMY AND NORMAL VARIANTS

A. ROTATOR CUFF

The tendinous portions of the rotator cuff are usually of low signal intensity on T2 weighted sequences. The biceps tendon within the intertubercular groove may have a round, elliptical or slightly flattened configuration. The intraarticular segment of the biceps tendon although difficult to evaluate in the absence of joint fluid, is also of low signal intensity.

B. GLENOID LABRUM

The wedged shaped fibrous tissue is of low signal intensity on all MR sequences. The interposed hyaline cartilage covering the osseous glenoid may be of increased signal intensity on T2 weighted images. Peripherally the labrum is joined by fibrous tissues to the capsule and glenoid rim. Usually the labrum is also attached centrally blending with the articular cartilage surface. Occasionally the labrum may be of the "meniscoid type" with the labrum detached centrally, with a free inner edge (central margin), which overlaps the articular cartilage. This variant is most often encountered superiorly but may be present anteriorly and posteriorly. The meniscoid type labrum should not be mistaken for labral tearing. With a meniscoid type labrum only a small smooth gap exists between the labrum and the glenoid rim.

C. SUBLABRAL FORAMEN

The Sublabral Foramen (present in11% of normals) is located between the labrum and the glenoid rim above the mid glenoid notch. A true Bankart lesion always occurs below the level of the mid glenoid notch although it may extend above the notch. SLAP lesions always include the biceps tendon anchor site with fraying and fragmentation of the tissues. A sublabral foramen on MR arthrography demonstrates uniformly smooth margins. A sublabral foramen is usually seen on axial images as a smooth parallel gap that must be differentiated from low-grade SLAP lesions.

D. BUFORD COMPLEX

Buford complex (present in 1% of normals) has a thick, “cord like” middle glenohumeral ligament with a high insertion. The anterosuperior glenoid is devoid of labral tissue and may mimic an anterosuperior labral tear. The smooth margins seen help distinguish this normal variant from true labral pathology.

V:IMPINGMENT SYNDRONE:

Impingement syndrome is a clinical constellation of pain and dysfunction due to entrapment of the rotator cuff, subacromial/subdeltoid bursa and the biceps tendon between the humeral head and overlying coracoacromial arch. Anatomic variations of the coracoacromial arch that compromise the supraspinatus outlet can lead to inflammation of the bursa and to tendon degeneration and tears. Impingement remains a clinical diagnosis. MRI may define the anatomy of the supraspinatus outlet and delineate anatomic findings, which may predispose, or cause impingement syndrome.

A hooked (type III) acromion may narrow the supraspinatus outlet. Acromioclavicular degenerative changes are common and frequently result in anatomic narrowing of the supraspinatus outlet. Lateral down sloping of the acromion and the congenital os acromiale are less common factors contributing to narrowing of the supraspinatus outlet.

Secondary impingement is due to subluxation of the humeral head due to ligamentous and/or capsular laxity. Posterosuperior glenoid impingement and secondary impingement may both be difficult to demonstrate with conventional MRI and may necessitate MR arthrography.

VI: ROTATOR CUFF TEARS

A. Conventional high quality MRI is usually sufficient in the diagnosis of full thickness rotator cuff tears and in the evaluation for significant partial tears. Separation of partial tearing and changes of tendinosis/tendinopathy can usually be distinguished from significant cuff tears. Most rotator cuff tears begin and occur at or near the insertion of the supraspinatus anteriorly. Small tears in this location are often best demonstrated on the oblique sagittal T2 weighted acquisitions or oblique coronal T2 weighted sequences. Larger full thickness rotator cuff tears can be demonstrated on all three imaging planes as an area of fluid that replacing the normal tendon. In viewing MRIs for rotator cuff pathology care must be taken to thoroughly evaluate all structures to look for associated pathology. Tendinosis/tendonopathy usually is seen as an area of increased signal intensity on the proton density weighted images and diminished signal intensity on the T2 weighted sequences. Larger full thickness rotator cuff tears can be demonstrated on all three imaging planes as an area of fluid (high signal intensity) that replaces the normal tendon. Significant partial tearing is seen as an area of increased signal intensity on the T2 weighted sequences with a focal or diffuse thinning of the rotator cuff. Occasionally the cuff may be thick in the area of a partial tearing.

B. ROTATOR CUFF TEAR SIZE AND ATROPHY

MRI can be useful in preoperative planning as the size of the full thickness tear component can be measured in anterior to posterior dimensions. The gap that may exist between the retracted end and its insertion site can also be accurately measured. The degree of atrophy present can be assessed best on the oblique coronal and oblique sagittal images and can also be helpful in preoperative assessment.

C. EVALUATION OF THE POST-OPERATIVE CUFF

Although evaluation of the post-operative cuff may be difficult, MRI is frequently beneficial. Knowledge as to the size of the prior cuff tear, and optimally copies of preoperative MRI's and operative reports makes evaluation of the post operative rotator cuff more accurate. For example, a 1 cm full thickness tear following repair of a large 4 cm rotator cuff tear, may represent the residual defect following the best cuff repair possible. Routine MR imaging may be sufficient, however, usually we schedule these examinations for potential intraarticular gadolinium, and based on the initial appearance of the rotator cuff on T2 weighted sequences, make a decision as to whether intraarticular contrast is necessary. Leakage of contrast from the joint into the subacromial subdeltoid bursa may be normal following rotator cuff surgery. A large amount of native fluid in the subacromial bursa and a large area of high signal intensity within the post operative cuff may indicate recurrent tearing, although further studies are necessary to support our initial observations.

The normal postoperative cuff following rotator cuff repair however may contain areas of increased signal intensity and irregularity which are within the spectrum of normal postoperatively. Care must be taken not to overestimate recurrent rotator cuff tearing based on an irregular cuff appearance on MRI. Knowledge as to the type of repair performed will be helpful in evaluation of the post-operative cuff on MRI. For example suture anchors which may be placed slightly medial to the native cuff insertion site. Encountering the post-operative tendon end medial to its normal location, may therefore be normal postoperatively. An area of tendonous debridment but may have increased signal density, irregularity and thinning, all of which may be within the spectrum of normal postoperatively. Areas of delamination following rotator cuff repair are best delineated with MR arthrography . Large full thickness rotator cuff tears are easily demonstrated with routine MR imaging.

VII: A.C. Joint

A. Posttraumatic and stress–induced osteolysis of the distal clavicle may demonstrate bone marrow edema in the distal clavicle, soft tissue swelling, cortical irregularity, subchondral cysts/erosions, joint widening and effusions. Must exclude artifactually increased signal (**coil burnout**) from poor coil positioning or selection

B. Os Acromion: his is best seen on the axial images; therefore the axials must always include all of A.C. joint.

VIII: BURSA

Fluid in the bursa may be seen with inflammation and/or bursal-sided cuff pathology. Look for an irregular interface of the bursal fluid and cuff seen with bursal cuff tearing/fraying. Artifactual increased signal may be seen along the subacromnial fat plane. T2 sequences with fat saturation eliminate this potential pitfall.

IX: GLENOHUMERAL LIGAMENTS

A. **Superior glenohumeral ligament (SGHL)**: Small, consistent structure which is always present, originating from the upper portion of the anterior humerus, just proximal to the lesser tuberosity (fovea capitis) inserting just anterior to the superior glenoid tubercle and base of the coracoid.

B. **Middle glenohumeral ligament (MGHL)**: Originates just below the SGHL on the lesser tuberosity and anatomic humeral neck and may insert directly on the anterosuperor labrum or may insert along the anterior neck of the glenoid. This scapular insertion may create a large anterior pouch. The MGHL primarily has a role in stability of the joint from 0-45 degrees of adduction. It may be absent in 15% of subjects

C. **Inferior glenoid ligament (IGHL)**: The most well defined of the glenohumeral ligaments with a thick anterior band originating from the inferior humeral neck insertion along the inferior two-thirds of the glenoid via the labrum. The anterior band and axillary pouch are important stabilizers from 45-90 degrees of abduction and external rotation. All structures of the inferior glenoid humeral ligament are important for posterior instability.

X: LABRAL PATHOLOGY

Conventional (non - contrast) MRI has yielded mixed results in the detection of labral tears with sensitivities ranging from 44 to 99% and specificities from 66 to 90%. State of the art, high field strength equipment tends to produce better results than lower field strength systems. The accuracy in the detection of labral tears is improved with MR Arthrography. We therefore perform MR Arthrography on most patients with suspected labral pathology. During the first one to two weeks following an acute dislocation there may be sufficient native fluid present within the joint to distended the joint and separate the capsolabral structures. Therefore, in the acute setting, MR arthrography may not be necessary.

XI: MR ARTHROGRAPHY

A. THEORY

With distension of the joint (MR Arthrography) the capsulolabral structures are separated allowing improved visualization and imaging assessment.

B. TECHNIQUE

For MR Arthrography the intraarticular injection is performed under fluoroscopic guidance via an anterior approach. Approximately 15-20 cc of a dilute gadolinium saline solution (1:100 dilution) is infused. Although the usual dilution cited in the literature is 1: 250, we have found that a 1.5 Tesla 1: 100 provides optimally high signal intensity on T1 weighted sequences. In addition however on T2 weighted sequences the 1:100 dilution is slightly gray in signal intensity when compared to native fluid collections, which allows the contrast to be separated from native fluid collections. At higher dilutions (1: 250) it is not possible to separate native fluid collections from the gadolinium - saline as both are of similar high signal intensity. Following the MR arthrogram T1W acquisitions in three planes are acquired in addition to an oblique coronal T2W sequence. 3D imaging or imaging with the shoulder in the abducted externally (ABER) position may also be helpful in selective cases. MR Arthrography utilizing gadolinium is currently not FDA approved and should be performed with IRB (Institutional Review Board) approval. MR Arthrography represents an "off label" use of gadolinium, which is an approved drug for intravenous administration.

XII: SPECIFIC INJURIES

A HAGL Lesion – Humeral avulsion of the glenohumeral ligaments. This lesion is best depicted on an oblique coronal T1 MR arthrogram in conjunction with the oblique coronal T2 weighted sequence. There is focal discontinuity of the capso - ligamentous complex at the insertion to the humerus insertion permitting extension of contrast beyond the confines of the capsule.

B. RAGHL lesion -- reverse (posterior) avulsion of the glenohumeral ligaments. This lesion is also best delineated on the coronal MR arthrogram. There's focal discontinuity of the capso - ligamentous complex at the posteroinferior insertion to the humerus allowing the extension of contrast beyond the confines of the capsule.

C. ALPSA-Anterior labrum periosteal sleeve avulsion. This lesion is best depicted on an axial MR Arthrograms. The anterior labral tear is accompanied with tearing of the periosteal from the anterior glenoid extending down the neck of the scapula.

D. GLAD - Glenoid labrum articular cartilage disruption. This lesion tearing there is disruption of the articular (hyaline) cartilage. This lesion may occur interiorly or posterior.

XII: SLAP LESIONS

A. SUBTYPES

1. SLAP Type I, Subtle irregularity and imbibition of gadolinium consistent with fraying along the articular surface and base of the superior labrum.
2. SLAP Type II, Gadolinium extends between the superior labrum and osseous glenoid with irregular margins. The width of the defect and irregularity allow a SLAP lesion to be differentiated a sublabral foramen. The sublabral foramen is usually a thin hole with smooth margins.

3. SLAP Type III, Bucket handle tear of the superior labrum that spares the biceps tendon.

4. SLAP Type IV Bucket handle tear of the labrum with the tear extending into the biceps tendon.

   In both type III and IV SLAP lesions the bucket handle component of the tear may not be displaced at the time of imaging with distended, and with the arm in a neutral abducted position. All SLAP lesions are best depicted on an oblique coronal MR arthrogram. The axial MR arthrogram may also be helpful any evaluation of SLAP lesions

XIII: LABRAL DEGENERATION

Labral degeneration, usually encountered in patients over fifty, may be difficult to differentiate from SLAP type I or II lesion. Gadolinium Arthrography may help in making this differentiation.

XIV: PARALABRAL CYSTS(GANGLION)

Fluid collections around the glenohumeral joint are common. We examined a series of 500 consecutive shoulder MRI studies and found that para- labral cysts were encountered

in 5.6 percent of all studies. In a subset of this population who later underwent shoulder arthroscopy labral tears were found in two-thirds of this population. Other other published series have also reported the association of ganglia around the glenohumeral joint with labral tears. In the presence of a para- labral cysts, a diligent search for the labral tear must ensue. If there is little native joint fluid present MR arthrography may be indicated. Most often however the cysts do not demonstrate direct communication with the glenohumeral joint on MR arthrography. T2 weighted sequences are an essential part of MR arthrography to ensure these ganglia are detected.
Although the ganglia may occur anywhere along the glenohumeral joint, they commonly have a bilobed configuration and extend along the supraspinatus fossa and the infraspinatus fossa .In this location the branch of the superscapula nerve to the infraspinatus may be impinged within the spinoglenoid notch. Careful inspection of the infraspinatus muscle belly evaluate for possible denervation is critical.

XV: THE POST OPERATIVE LABRUM

Postoperatively labrum may demonstrate normal blunting, attenuation or contain areas of increased signal intensity. Care must be taken in the postoperative setting not to over call labral pathology. MR arthrography may be helpful in this setting. Recurrent label tears may be demonstrated by observing gadolinium or fluid extending through the base of the labrum or within the substance of the labrum.

Making Arthroscopic Rotator Cuff Repairs Easier
My Experience and Technical Pearls

James C. Esch, M.D.
Tri-City Orthopaedics
Oceanside, CA
jesch@shoulder.com
www.shoulder.com

Surgeon
- **Must balance skill versus ego**
- **Practice on a model**
- **Know tools**
- **Know suture management**

OR team
- **Taught by the surgeon**
- **Know location of your favorite tools and anchors**
  - **Can load suture, find correct tool**
- **Knows location of Plan B and Plan C tools**
- **Tie sliding and _ hitch knots**

Bleeding control awareness
- **Inflow pressure and bag awareness**
- **Outflow control**
- **Pump nuisances**
- **Anesthesia BP awareness**
- **Is patient taking a NSAID ?**

Plan for today's patient with a cuff tear
- **Size of tear**
- **Pain versus weakness**
- **Risk of massive and superior migration**
- **What is your "mini-open" threshold? And experience?**

Tear Fix Size
- **MRI**
  - **Supraspinatus**
  - **Infraspinatus**
  - **Subscapularis**
  - **Biceps**
- **Draw the size and shape on paper**

Draw Tear
- **Tear Estimate**
  - **Size**
  - **Shape**
  - **Does it look repairable? (Full? Partial?)**
- **Repair Technique**
  - **Margin convergence**
  - **Fixation Estimate**
    - **Anchors**
  - **Suture technique**
- **See the technique steps in your head**
  - **See the anchor, suture through tendon, suture management, and tie knot.**
  - **You may need to move the scope and suture for these steps.**

Exposure
- **Portals and Cannulas**
- **Bursectomy to see**
- **Subacromial smoothening (decompression)**

Intra-operative Evaluation
- **Probe tear after bursectomy and cleaning bony bed**
- **Is your Plan now the same as preoperative plan?**
- **Start to run the play. (You are running an option formation.)**

Margin convergence
- **Handoff Techniques**
  - **A: Direct Permanent Suture**
    1. **Cuff sew #2 Ethibond to ArthoPierce**
    2. **Other permanent handoff devices**
  - **B: Shuttle with Crescent hooks to Blitz/Lasso**
- **Tie a good knot**

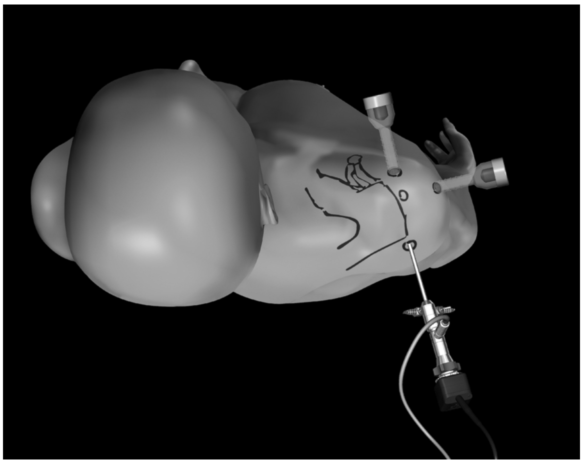
Anchor first
- **Use 18G needle to get the angle**
- **I prefer anchors down a cannula**
- **Consider double row @ tear mobility**
- **Put in all at once if able**
- **Know suture management**
- **Tag ends of each suture**

Suture through tendon
- **Direct trans-tendon grab of suture**
  - **ArthroPierce and other penetrating tools**
    - **From posterior for Infraspinatus**
    - **From anterior for some supraspinatus**
    - **From superior behind AC joint for U shaped SS tears**
- **Pass suture through tendon (if angle is good)**
  - **Direct with Cuff Sew, Penetrators, Arthrosew**
- **Shuttle suture**
  - **Use Caspari punch, crescent hooks, etc**

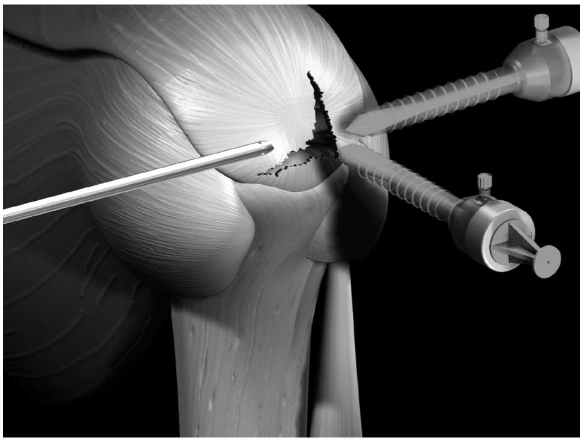
Postoperative care
- **Immobilize long enough to heal**
- **Some passive motion is good**

Rehabilitation phases
- **Immobilization**
- **Early active motion**
- **Late strengthening**

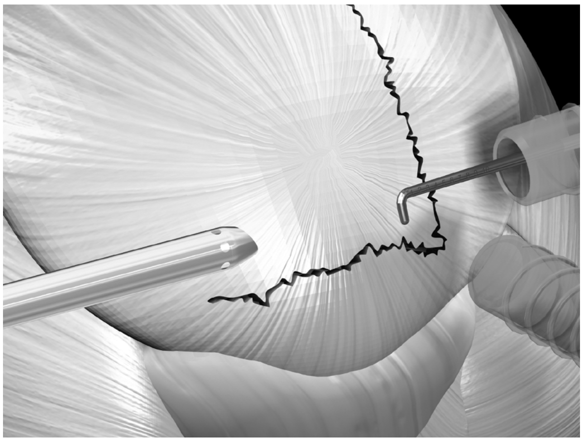
Conclusions:
- **This is hard**
- **This requires thinking out the steps**
- **This is frustrating**
- **This is rewarding**
- **This balances your skill versus your ego**



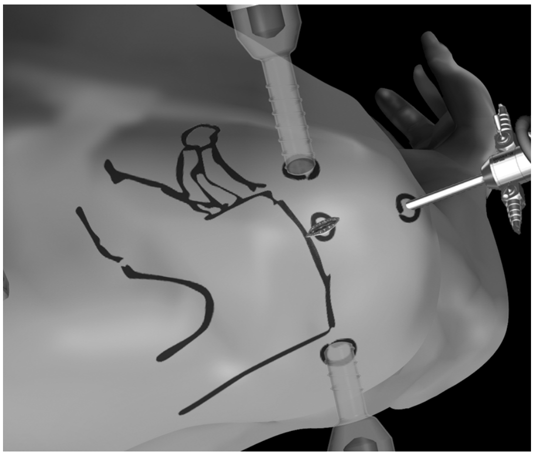
1. Three Portals, scope posterior



2. Inside view. Scope and tools are moved as needed to repair the cuff tear.



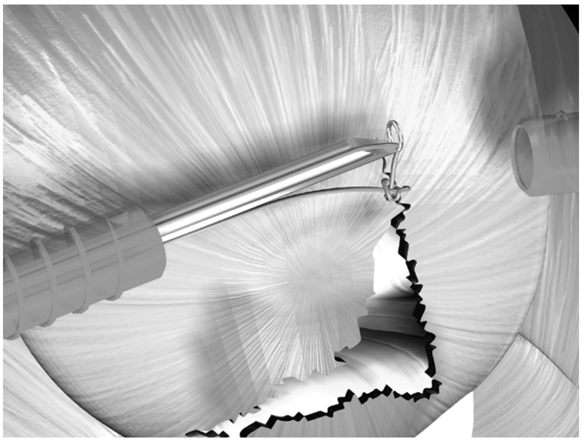
3. Estimate the steps necessary for repair.



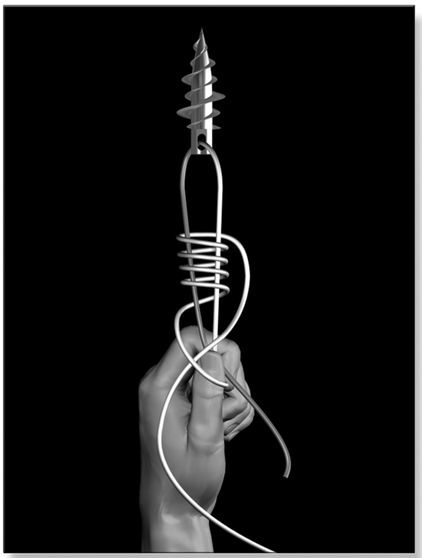
4. The scope is now in the lateral portal providing the "50-yard line" view. ***Note the anchor insertion portal adjacent to the acromion***.

5. Margin convergence with a single pass cuff sew tool.

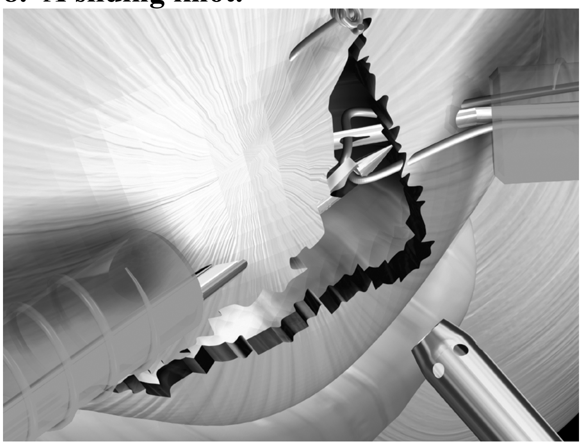
6. Retrieve the suture.



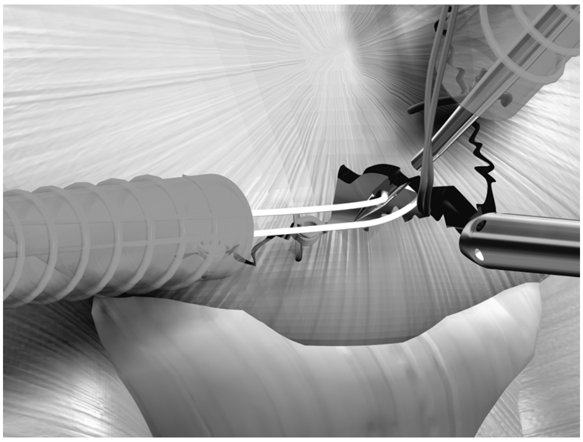
7. Tie the knot



8. A sliding knot.



9. A suture "handoff" from the ArthroPierce to the straight Cuff Sew.



10. Retrieving the suture off of the anchor with the ArthroPierce.

11. Final repair with two margin convergence sutures and two anchors.

Illustration from Esch: <u>Arthroscopic Rotator Cuff Repair</u> for Smith+Nephew Endosocpy.

Symposium: Shoulder Instability – Limits of Arthroscopic Surgery: Bone Deficiency, Shrinkage, Acute Instability
Monday, March 10, 2003 • Aotea Centre, Kupe/Hauraki room

Chairmen: W. Jaap Willems, MD, Netherlands and Philippe P. Hardy, MD, France
Faculty: Stephen Burkhart, MD, USA and Joe De Beer, MD, South Africa, Mario Larrain, MD, Argentina

Introduction
P.Hardy J.Willems 5 mn

Bony lesions
S.Burkhart the glenoid side 8 mn
P.Hardy the humeral side 8 mn

Capsular thermal shrinkage
J.Willems 8 mn

High risk sports
J de Beer 8 mn

Conclusion and discussion
M.Larrain 7 mn

Bone Loss in Instability: A Contra-Indication to Arthroscopic Repair?

S.S.Burkhart San Antonio USA

Current Series

- 194 Arthroscopic Bankart Repair
- Consecutive Series of Traumatic Anterior Instability
- 2 Surgeons
- July 1992 to June 1998
- Average Follow-up : 27 months

Results

- Group 1 :

–No Significant Bone Defect (173 patients)
–Recurrence rate : 4 %

- Group 2 :

–Significant Bone Defect (21 patients)
–Recurrence rate : 67%

Contact Athletes

- If bone defect, 87% recurrence
- If no bone defect, 6.5% recurrence
- A. glenoid side

-"INVERTED PEAR"

KUPE/HAURAKI

- Inferior glenoid narrower than superior glenoid (> 25% Loss of Inferior Glenoid Arc)

“Inverted Pear”

Quantifying the“Inverted Pear”

Biomechanical & Geometric Factors

●“ Dish Effect of Glenoid ”

●Shallower Dish Less shearing resistance

“Inverted Pear”

Biomechanical & Geometric Factors

●Effect of glenoid arc length

●Shorten the arc Less resistance to Humeral Axial Forces

Conclusion

Arthroscopic Bankart results equal to Open Bankart results if no significant bone defects

Contraindication Contact Athletes with Bone Deficiency

BONY LESIONS INFLUENCE ON THE RESULT OF THE ARTHROSCOPIC TREATMENT OF GLENO-HUMERAL INSTABILITY

Ph.HARDY. Paris France

Introduction

The arthroscopic treatment of glenohumeral instability has the reputation to present a more important failure rate than open treatment. We were interested in the influence of bony lesions and more especially Hill-Sachs lesions on a possible pejoration of clinical results.

Material and methods

We have made a retrospective study on 60 patients operated between February 1993 and December 1998 by an arthroscopic bankart for chronic anterior glenohumeral instability.The average patients age was 28 year. We have calculated on preoperative computed tomography the volume and the location of the Hill-Sachs lesion if present. Six measures have been used in order to get a volume that we assimilated to the half volume of a revolution ellipsoid. The ratio of the Hill-Sachs lesion was compared to the volume of the humeral head assimilated to a sphere. Glenoidal bony lesions were also evaluated in four types : normal, abrasion, fracture and amputation. Clinical results were evaluated using the Duplay score. The average follow-up was 42 months. None of the patients was lost for follow-up.

Results

Global results were : 24 excellent results, 14 good, 7 fair, 5 bad including recurrence of the instability (apprehension) and 10 failures needing reoperation by an open approach. The rate of operated recurrence was 16.6%. Hills-Sachs lesion volumes were from 0 to 4792 mm^3 (average :1019 mm^3 , standard deviation :1253 mm^3).The average ratio was 2.28% (max 10.5, min 0 , standard deviation 2.63). Only 12 patients didn't present any humeral bony lesion (20%) in this group 11 had an excellent or a good result. The average volume of the Hill-Sachs lesion in group A (excellent, good and fair results) was 639.72 mm^3. The average volume in group B(failures) was 2158.11 mm^3 . In group A glenoidal bony lesions were 17 normals, 20 abrasions, 8 fractures and 0 amputation. In group B, 2 normals, 9 abrasions, 2 fractures and 2 amputations. Using Spierman’s test and average comparison the size of the notch appears highly corelated to clinical results. We were not able to show the same relation between glenoidal lesions and clinical results. However we have noted that major glenoid rim when the Hills–Sachs volume was lower than 500mm3. Considering a volume inferior to 1000mm3 the failure rate was 7.7% whatever the bony lesions of the glenoid were and 2.5% if we excluded amputations and fractures of the glenoid rim.

Conclusion

Arthroscopic treatment should be reserved to Hills-Sachs lesions with a minor volume (< 1000mm3) and wihtout bony lesions on the glenoid side (amputations and fractures).

THERMAL SHRINKAGE IN SHOULDER ARTHROSCOPY

W.JAAP WILLEMS, M.D.,Ph.D., Amsterdam, The Netherlands

INTRODUCTION

Since the Radiofrequency Energy (RF) was introduced , thermal energy became an increasingly popular method to treat pathology of the shoulder. Thermal energy was first used with lasers and most of the research has been performed with this laser equipment, but mainly due to its costs the RF systems have replaced the laser systems.

THERMAL ENERGY

With the LASER (Light Amplification by Stimulated Emission of Radiation) system the light energy, when in contact with the tissue and with adequate power, is converted to heat and subsequently the water and tissue are vaporized.

The Radiofrequency Energy (RF) transmit a high frequency alternating current from their tips to the tissue. The subsequent ionic agitation results in frictional heating within the tissue.
The alternating current is passed between the probe and a ground plate (monopolar systems: Oratec) or between 2 points on the probe tip (bipolar systems: Mitek, Arthrocare).
The heat at the lesion is dependent on a) distance, b)current intensity, c) time of exposure.
Both monopolar and bipolar systems create collagen shrinkage in the same way.
There is sofar no objective method , besides the macroscopic appearance during the procedure, to measure the effect of the applied energy.

BIOLOGICAL AND HISTOLOGICAL EFFECTS

In fibrous connective tissue the collagen fibers (built up from collagen type I fibrils) are joined together in a triple helix structure.The 3 chains are interlinked by intermolecular bonds. At about 60^0 C these bonds break and the collagen structure begins to unwind. The diameter of the collagen fibrils increases.
In in vitro animal studies, where 10% shrinkage of fibrous tissue was obtained, the following effects have been shown:

Immediately after the shrinkage diffuse hyalinisation as well as fusion of collagen fibers was seen.
After 1 week fibroblast proliferation occurred in the hyalinized areas.
After 1 month fibrosis was seen with disorganised connective tissue. Reactive fibroblasts start collagen repair on the larger denatured collagen fibrils, using them as a scaffold.
After 3 to 6 months the tissue looked histologically normal .
Tissue culture analysis showed a peak of collagen synthesis 2 weeks after shrinkage and return to normal level 1 month after shrinkage.

Inadequate exposure of thermal energy causes necrosis of tissue, with either disappearance of the capsule or scar formation.

BIOMECHANICAL EFFECTS

As long as the proper amount of energy has been applied no changes in the tensile stiffness or failure strength occur. With increasing shrinkage (more than 10% of tissue length) too much energy will be applied to the tissue, with as result an irreversible loss of tissue stiffness. Uncontrolled application leads to tissue ablation. No ideal endpoint of the amount of shrinkage has been defined.

SURGICAL TECHNIQUE AND INDICATIONS

Thermal energy , either with laser or RF has been used in multidirectional instability as well as adjuvant procedure in unidirectional instability, after labral repair and /or capsular shift. One study showed the effect in posterior impingement.
In the beginning the anterior, inferior, posterior and sometimes the rotator interval was treated in multidirectional cases. As an adjunct procedure in anterior instability only the anterior capsule was shrinked.
Recently an animal study showed that treatment with linear passes (grid pattern) showed greater healing properties and better mechanical effect than the treatment with uniform application of the heat (paintbrush pattern).

REHABILITATION

In animal studies the mechanical properties of the capsule are not essentially changed with the proper use of the heat energy ; however there are no data on the effect of loading of the capsule after the in vivo shrinkage procedure, so for this reason, although evidence is lacking, some form of initial immobilisation (2 weeks immobilisation, with another 4 weeks of passive motion) is recommended.

CLINICAL RESULTS

Most series , published sofar show good to excellent results .
However there are several weaknesses in the reports:
- The follow-up is in most series is not longer than 2 yrs.

-The indications are variable, with sometimes mixing patient with multidirectional instability or multidirectional laxity. In series where the shrinkage is an adjunct to the labral repair, a clear effect of the shrinkage cannot be determined.
-The technique is not always used in a standard way: the duration of application can vary, as well as the applied intensity.

COMPLICATIONS

Several complications have been described:

- axillary nerve lesions ; most described axillary nerve lesions were reversible.
- secondary frozen shoulder is mostly reversible.
- recurrent instability; in some series up to 40% recurrence is reported.
- irreversible tissue changes: tissue ablation, tissue necrosis with even complete loss of the capsule is described.

CONCLUSION

Thermal energy might be a valuable tool in treating some shoulder problems.
Biological and biomechanical studies have shown, that if properly used, an adequate shrinkage can be achieved without altering the biomechanical properties of the tissue.
Longterm randomised studies in clearly defined indications are needed to evaluate the ultimate value of this treatment modality.

Literature:

Hayashi K et al. The biological response to laser thermal modification in an in vivo sheep model. Clin Orthop 2000;373:262-276.
Medvecky MJ et al. Thermal capsular shrinkage. Arthroscopy 2001;17:624-635.
Wall MS et al. Thermal modification of collagen. J Shoulder Elbow Surg 1998;8:339-344.

Shoulder Instability: High Risk Sports

Joe de Beer Cape Town

Introduction
Sports with repetitive overhead motion carry a risk of shoulder instability due to damage to the labro-ligamentous tissues. Contact sports like rugby, on the other hand, carry a risk due to the force of impact. The latter group can be prone to failure of surgical repair due to the excessive forces applied to these shoulders.

High-risk sports:
1. Repetitive overhead motion: baseball pitchers, tennis players and swimmers suffer shoulder instability due to repetitive overhead motion and the shoulders being placed into ”at risk” positions. The pathological lesion is usually stretched out ligamentous tissue with or without Bankarts lesions.

2. High impact sports: rugby, football and others are good examples. The mechanism of injury is often different from group 1. There might be direct impact to the humeral head with forced dislocation or horizontal extension injuries. The pathology in these shoulders is often different: glenoid bony lesions, HAGL and GLAD lesions found more often than in group 1. In this group failure of surgical treatment is either due to not addressing bony lesions as well as repeated high impact forces.

Surgical Management:

1. In this group arthroscopic stabilisation carries a high success rate and avoids the stiffness of the open repairs –an advantage to the overhead sportsman.

2. In this group open surgical management is often required: the Latarjet procedure for glenoid lesions and open repair for HAGL lesions. In those cases without bony deficiency arthroscopic repair is a viable option, contact sports and young age not being a contra-indication to arthroscopic repair..

Summary:

Arthroscopic stabilisation is successful for a large proportion of unstable shoulders in high-risk sports. With adequate selection and using the appropriate open repair a satisfactory outcome is usually achieved.

Debate: Acute Stabilization of Primary Anterior Dislocation of the Shoulder
Monday, March 10, 2003 • Aotea Centre, Kupe/Hauraki Room

Moderator: James C. Esch, MD, USA
Faculty: Metin Lufti Baydar, MD, Turkey and Robert A. Arciero, MD, USA

Arthroscopic Fixation Technique of Acute Anterior Shoulder Dislocation
Metin Lütfi Baydar, MD, Professor, Chairman of Department of Sports Medicine,
Süleyman Demirel University 03200 Isparta / TURKEY

INTRODUCTION
Arthroscopically assisted repair of the anterior aspect of the labrum with use olived and grooved k-wire was performed in forty-nine young patients who had acute anterior traumatic dislocation of the shoulder. The average age of the patients was twenty-two years (range, sixteen to twenty-seven years). The etiology of the dislocation was a military training in twenty-nine patients, sports injuries in fifteen patients and fallen of daily activity in the others five patients. This method is a simple, effective and chip way.

MATERIAL and METHOD
We treated 49 cases that had acute anterior shoulder dislocation with arthroscopy between January 1996 – June 2002. All cases were male and their mean age was 22. After standard diagnostic arthroscopy, the labral ligamentous lesions are fixed with two olived and grooved 3.0 K-wires through an anterior portal. We immobilized with Welpau bandage and cold application after operation. The K-wire is disengaged in postoperative 3 weeks and a special rehabilitation program is applied. As a control group, 49 cases were treated conservatively. Shoulder was kept in shoulder sling for 3 weeks then it removed and rehabilitation programme applied .

Surgical Technique: Lateral recumbent position was used for all operations. After determining bony landmarks, standard arthroscopic diagnose was made by using posterior portal. After forming anterior portal, debritman and abrasion were performed at the separation area of labral ligamentous complex from glenoid. Attachments around separated labrum were debrited with shaver and continuity of glenoid labral complexes were provided by two 3.0 mm K-wires. When the arthroscopic examination and surgical manipulation were finished, all portals were closed.

Postoperative Care: We immobilized shoulder with Velpau bandage and Ezy-Wrap (for cold application) in postoperative 48 hours and after that shoulder by a shoulder sling. All suture materials were removed in the twelfth day. K-wires were disengaged at the and of the postoperative third week and a special rehabilitation programme was applied.

Codman's pendular exercises are started at the end of the third week. When the patient thus not have any more pain, isotonic exercises may be started. Later isotonic exercises that enhance deltoid rotator cuff and scapular muscle power are started. Patient should continue this exercises until they gain back their normal activities.

RESULTS
The results of our cases are satisfying. The normal shoulder had a Constant score of nearly 100, as expected. In our group it was 94 and control group it was 75. As a complication, redislocation was seen in 17 cases of the control group whereas only in one case of the K-wire fixation group in the postoperative third month. Moreover, during the follow-up period of sixteen months, we observed that the K-wire separated from fixation area and slided to pectoral muscles group in axillar region in one case and this K-wire was extracted.

CONCLUSIONS & SIGNIFICANCE
One of the most important lesions which cause anterior glenohumeral instability is labroligamentous avulsion lesion(1,4,5,6,9). Thisl lesion, which had some difficulty in diagnosis because of conventional diagnostic methods, can be easily diagnosed and treated in the same session with arthroscopy (3,5,6,8). Also olived and grooved K-wire fixation under arthroscopic control is less invasive and has low morbidity rate compared to conventional open surgical procedure (2). We believed that the treatment of labral lesions observed under 25 years old with arthroscopy would prevent recurrence of shoulder dislocation and we strongly recommend this method for treatment of traumatic shoulder dislocations which acute first time or reoccurred less than three times.

We believe that using K-wire fixation in the treatment of Bankart lesions is easy, cheap and useful in selected cases. Number of previous dislocations, etiology, direction of dislocation, a bone defects are important in patient selection. We hope that with improvement of this technique, much better results can be obtained in the future.

REFERENCES:

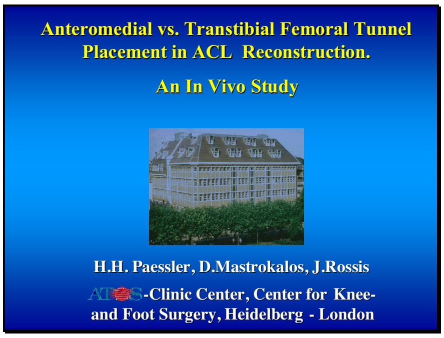
1. Bankart, A.S.B.: The pathology and treatment of recurrent dislocation of the shoulder joint. British J. Surg. 26: 23-29, 1938.
2. Büktel,H., Yanmı_,_., Yaman, H., Aydo_an, N., Baydar, M.L., Gür, E., Cerulli, G.: The artroscopic treatment of acute anterior ligamentous labral lesions with K-wire fixation. 3rd.Turkish Sports Traumatology Congress. Abstract Book p. 60, Ankara 1996.
3. Caspari, R.B., Savoie, F.H., Meyers, J.F.: Arthroscopic shoulder recostruction. Orthop Trans. 13: 559-560, 1989.
4. Caspari, R.B., Savoie, F.H.: Arthroscopic reconstruction of the shoulder : The Bankart repair: In Operative Artroscopy, pp 507-516. Edited by Mc Grinty. New York, Raven Press, 1991.
5. Johnson, L.L.: Shoulder arthroscopy. In Arthroscopy Surgery: Principles and Practice, p.180. Edited by L.L.Johnson, St Louis, C.V. Mosby, 1986.
6. Neviaser, T.J.: The anterior labroligamentous periostal sleeve avulsion lesion: A cause of anterior instability of the shoulder. Arthroscopy 9: 17-21, 1993.
7. Rowe, C.R., Zarins, B.: Recurrent transient subluxation of the shoulder. J. Bone and Joint Surg. 63-A: 863-872, July 1981.
8. Speer, K.P., Warren, R.F., Pagnani, M., Warver, J.: Arthroscopic stabilisation of anterior shoulder instability using a bioabsorbable Tac. J. Bone and Joint Surg. (In press)
9. Wiley, A.M.: Artroscopy of shoulder instability and technique for arthroscopic repair. Arthroscopy 4: 25-30, 1988.

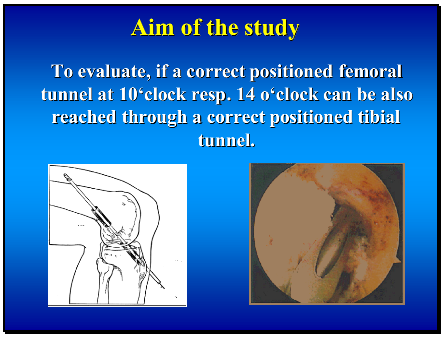
TUNNEL POSITIONING IN ACL RECONSTRUCTION (PATELLAR TENDON & HAMSTRING)

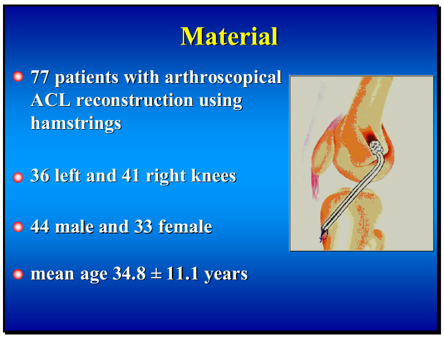
Tuesday, March 11, 2003 • Aotea Centre, Kupe/Hauraki Room

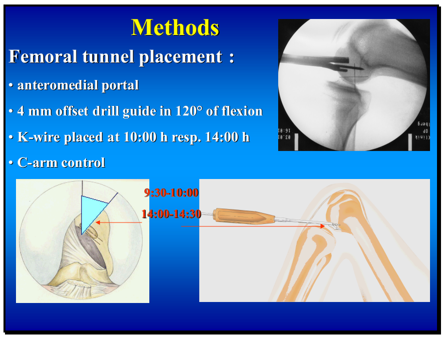
Chairman: Hans Paessler, MD, Germany

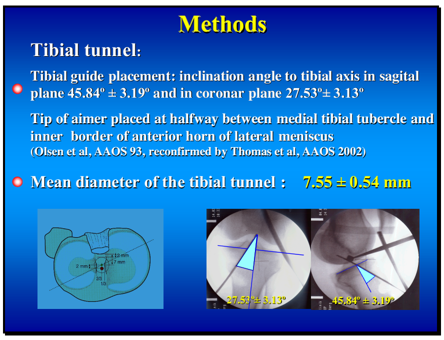
Faculty: Ponky Firer, MD, South Africa, Kazunori Yasuda, MD, PhD, Japan

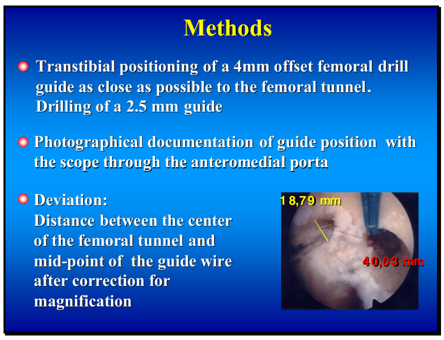










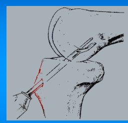


Results

- **In 74 knees (96.1%)** the femoral tunnel entrance could not be reached
- **In 3 knees (3,9 %)** the superomedial edge of the femoral tunnel was reached
- Mean deviation (distance) between center of the femoral tunnel and mid-point of the transtibial guide wire : **4.50 ± 1.54 mm** (p< 0.001)

Results

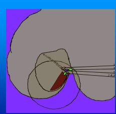
- No statistical relationship between deviation and tibial tunnel inclination angle in **coronar** and **sagital plane**
- No statistical relationship was observed between deviation and tibial tunnel diameter



tibial tunnel Ø	mean deviation ± std
7-7.5 mm	4.6 ± 1.5 mm
8 - 8.5 mm	4.0 ± 1.6 mm

Conclusion

- In our study we did not reach the anatomic side of ACL insertion even with larger tibial tunnels (for hamstrings grafts up to 8.5mm) by using the transtibial femoral tunnel technique
- Cour study confirms the results of a recent cadaveric study (Arnold, M.P. et al., KAOSM 2001)



References

- Giron F, Buzzi R, Aglietti P.: Feoral tunnel position in anterior cruciate ligament reconstruction using three techniques. A cadaver study. Arthroscopy 1999 Oct; 15(7): 750-6
- Aglietti P, Zaccherotti G, Menchetti PP, De Biase P.: A comparison of clinical and radiological parameters with two arthroscopic techniques for anterior cruciate ligament reconstruction . Knee Surg Sports Traumatol Arthrosc 1995; 3(1): 2-8
- Arnold MP, Kooloos J, van Kampen A.: Single-incision technique misses the anatomical femoral anterior cruciate ligament insertion : a cadaver study. Knee Surg Sports Traumatol Arthrosc. 2001 Jul; 9(4): 194-9
- Sgaglione NA, Schwartz RE.: Arthroscopically assisted reconstruction of the anterior cruciate ligament: Initial clinical experience and minimal 2-year follow-up comparing endoscopic transtibial and two-incision techniques. Arthroscopy 1997 Apr; 13(2): 156-165
- Howell SM, Wallace MP, Hull ML, Deutsch ML.: Evaluation of the single-incision arthroscopic technique for anterior crucite ligament replacement . A study of tibial tunnel placement, intraoperative graft tension and stability. Am J Sports Med. 1999 27(3): 284-293
- Torbjörn Gröntvedt, Fernando Pena, Lars Engebretsen.: Accuracy of femoral tunnel placement and resulting graft force using one- or two-incision drill guides. A cadaver study on ten paired knees. Arthroscopy 1996 Apr; 12(2): 187-192

ISAKOS
Auckland, NZ
March, 2003

Guidelines for Tunnel Placement for ACL Surgery

Christopher D. Harner, MD
Blue Cross Professor of Orthopaedic Surgery
Medical Director, Center for Sports Medicine
University of Pittsburgh Medical Center

ACL Surgery

Overview

- **I. The Experts: Global Perspectives on ACL Reconstruction**
- **II. ACL Tunnel Placement**
- **III. Summary**

ACL Surgery **May 4-6, 2000**

Panther Sports Medicine Symposium

- **Global Panels**
  - *ACL Reconstruction*
  - Revision ACL Reconstruction
  - Return to Play Following ACL Reconstruction
  - PCL Reconstruction
  - Osteotomy
  - Mensical Transplant
  - Multiple Ligament Injured Knee
  - Posterolateral Corner Reconstruction

ACL Surgery

2000 Panther Sports Medicine Symposium

- **14 surgeons**
  - North America
  - Europe
  - Africa
  - Australia
  - Asia
- **21 years experience (13 - 30 yrs)**
- **133 ACL reconstructions / year**

ACL Reconstruction in the New Millennium

- **6 min talk**
- **Indications**
- **Techniques**
  - Graft selection
  - *Tunnel Placement*
  - Graft fixation
  - Post-operative rehab
- **Complications**
- **Outcomes**

Harner et al, KSSTA, Dec. 2001

ACL Surgery

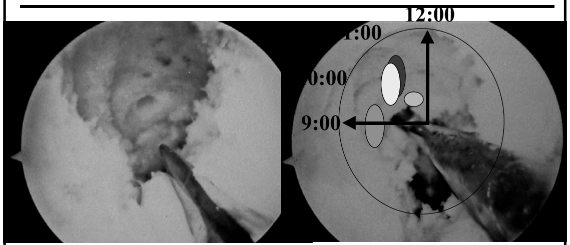
ACL Global Panel

Femoral Tunnel:

- **At posterior cortex ("blow-out") 6 / 14**
- **Off-set up to 7 mm from cortex 6 / 14**
  - *11:00 / 1:00 Placement 7*
  - *10:30 / 1:30 Placement 5*
- **Over-the-Top 1 /**

ACL Surgery

ACL Global Panel



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ACL Global Panel

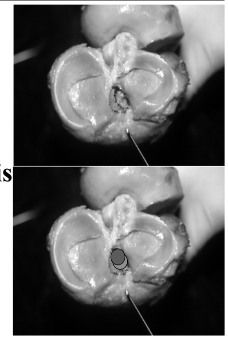
Tibial Tunnel:

- **Posterior/posteromedial**

  11 / 14

- **Off ant. horn of lat. menis**

  3 / 14



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Summary

Femoral tunnel -- most variable!!

- **Insertion is difficult to define**
- **Placement is dependent on guide / approach**

Tibial tunnel -- most consistent!!

- **Insertion is obvious**
- **Guides are consistent**

ACL Surgery

Tunnel techniques - CDH

Endoscopic

- **Harner, et al, Arthroscopy, 10:502, 1994**

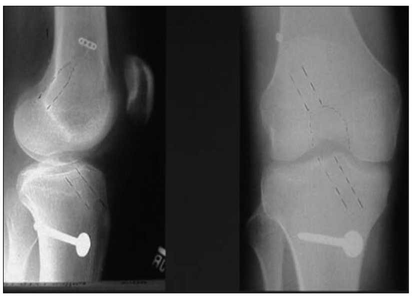
Tunnel placement

- **Know your insertion sites!**
- **Harner, et al, Arthroscopy, 15:741, 1999**

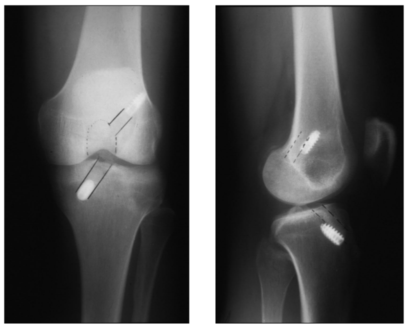
Graft selection

- **Allograft vs. autograft**

ST/Gr autograft



P.T. autograft



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Tunnel Placement - Criteria

- •Arthroscopic
- •Radiographic
  - » You need both

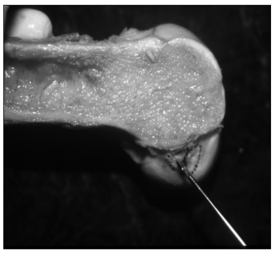
ACL Surgery

Arthroscopic Criteria

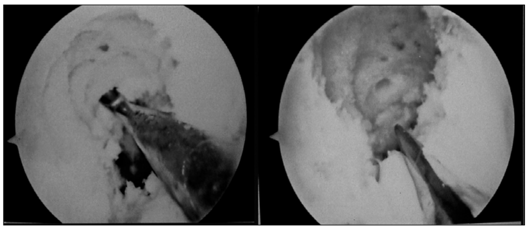
Femoral tunnel

- "Over-the-top"
- O'clock position 1:00/ 11:00

  2:00 - 3:00



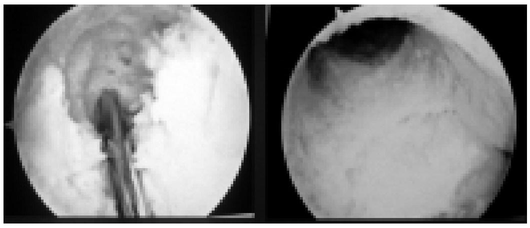
ACL Tunnel Placement - Femur



Most common errors:

- Too anterior!
- Too central (12:00)

ACL Tunnel Placement Femur



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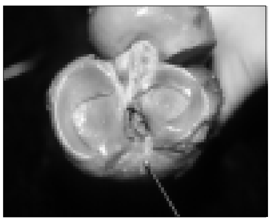
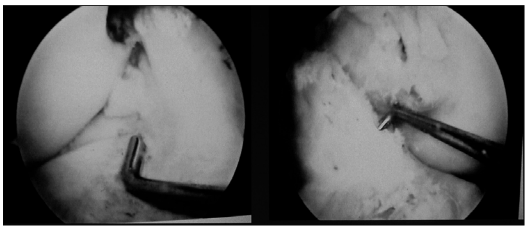
Arthroscopic Criteria

Tibial tunnel

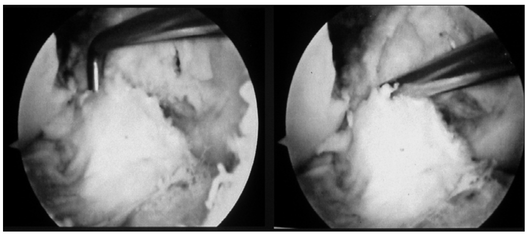
- "Footprint" - post. 1/2 vs. central
- Medial tibial spine
- *Ant. horn lat. meniscus*



ACL Tunnel Placement Tibia



ACL Tunnel Placement
Tibia



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Radiographic Criteria

- Intra-op
- Post-op

Obtain one or both!

ACL Surgery

Radiographs - AP

Femoral

- ICN
- "O'clock"

Tibial

- Medial tibial spine

ACL Surgery

Summary

1) Know your insertions - arthroscope and x-ray!
2) Femoral tunnel is more problematic!
3) Beware of "guides"!!!!

It is the surgeon, not the

Thank You

UPMC Sports Medicine Complex

BONE TUNNELS IN ANTERIOR CRUCIATE LIGAMENT RECONSTRUCTION : UNDERSTANDING THE VARIABLES

Ponky Firer, MD, South Africa

A. THE TIBIAL TUNNEL

This has both an entrance point on the tibial cortex and an exit point in the joint.

B. THE FEMORAL TUNNEL

This may have an entrance point on the lateral femoral condyle and an exit point in the joint.

Or

It may be drilled purely from inside and be a blind ended tunnel with an opening inside the joint.

I. <u>TIBIAL TUNNEL</u>

a) Tunnel obliquity

Because the tibial tunnel is drilled obliquely to the intra articular surface of the bone, the AP diameter of the tunnel at this point is wider than the mediolateral diameter which more closely matches the size the drill bit used.

b) The degree of verticality of this tunnel determines the extent of the AP diameter variance with respect to the mediolateral diameter. A more vertical tunnel has a smaller AP diameter than a more horizontal tunnel.

c) This causes graft tunnel mismatch at this tunnel entrance in the joint.

Ideal position for tibial tunnel in the joint is in the anatomical footprint of the old anterior cruciate ligament

a) In the coronal plane it lies in the same plane as the normal anterior cruciate ligament and is 70 - 75° from the line drawn parallel to the surface of the tibial plateau and is centred between the tibial spines. In the sagittal plane the drill hole should run parallel to the intercondylar roof in extension exiting behind the line of the roof. This takes the tunnel in the joint to the posterior part of the ACL foot print.

   i) If this tunnel is too anterior there is impingement on the notch.
   ii) If this tunnel is too posterior the ligament will lie too vertical and not function correctly.

The bone tunnel positioning is important to avoid impingement on

a) The roof of the notch.
b) The lateral wall of the notch.
c) The posterior cruciate ligament.

Thus avoiding tenting in these areas which puts stress on the graft and also changes the tensions in the graft through movement.

Anatomical landmarks for positioning of the guide wire for the tibial tunnel.

a) On the line between the anterior horn of the lateral meniscus and the medial tibial spine.

b) Approximately 6mm anterior to the posterior cruciate ligament.

c) In the posterior half of the old foot print.

II. FEMORAL TUNNEL

This tunnel should exit in the joint 1 – 2mm from the over the top position. It should be set at 10.30 on the clock for right knees and 1.30 on the clock for left knees.

Methods of drilling the femoral tunnel are :

a) Outside in technique using the rear entry guide system.

b) An all inside blind tunnel technique, of which there are various forms of instrumentation.

TUNNEL SIZE

The tunnel size should most closely match the size of the graft being used to within 0.5mm. This is especially important for hamstring grafts.

METHODS OF CREATING TUNNELS

a) Using sequential drill bits increasing the size of the drill tunnel until the correct size has been reached.

b) Dilation in which a small drill hole is first made and a series of dilators are used to dilate the tunnel to the correct size. The theoretical advantage of this is compacting the cancellous bone for better interference screw fixation.

c) Using a core reamer to core out a tunnel. This allows use of the cored bone for accessory fixation.

Guides available for the tibial drill hole

a) Simple point guides uses anatomical landmarks.

b) PCL based guides that key off the posterior cruciate ligament.

c) Intercondylar roof based guides that key off the roof avoiding impingement.

Femoral guides

a) Free hand drilling.

b) Offset guide that sets the guide wire the right distance to leave a 1 – 2mm rim in the over the top position.

c) Rear entry guide.

(a) and (b) can be drilled through the tibial tunnel OR via the medial arthroscopy portal.

Symposium: Tunnel Positioning in ACL Reconstruction

A Novel Procedure for Anatomical Posterolateral and Anterolateral Bundle Reconstruction for the ACL

Kazunori Yasuda, MD, PhD
Professor and Chairman
Department of Medical Bioengineering and Sports Medicine
Hokkaido University School of Medicine, Sapporo, Japan
(E-mail: yasukaz@med.hokudai.ac.jp)

1. Introduction

(1) ACL reconstruction with single bundle procedures
- Clinical outcome has been much improved
- However, knee functions after reconstruction are not completely normal
- Why?

(2) Normal ACL
- Composed of the anteromedial and posterolateral bundles (AMB and PLB).
- In single bundle procedures, these two bundles cannot be reconstructed

(3) ACL reconstruction with 2-bundle procedures
- Developed to mimic the AM and PM bundles
  - Mott (1983): 2 tibial tunnels and 2 femoral tunnels
  - Rosenberg (1994): 1-tibial tunnel and 2 femoral tunnels
  - Muneta et al (1999): 2 tibial tunnels and 2 femoral tunnels
  - Hamada et al (2001): 1-tibial tunnel and 2 femoral tunnels
- In the literature, these procedures did not anatomically reconstruct the PLB

(4) Where is the footprint of the PLB on the lateral wall of the intercondylar fossa?
- Anatomical observation of the ACL footprint
  - Girgis et al (1975):
- Nevertheless, orthopaedic surgeons have not understood where it is in the arthroscopic visual field in ACL reconstruction
  - No arthroscopy-based studies on the PLB anatomy

(5) Based on the arthroscopic PLB anatomy, we have developed a novel procedure for anatomical PLB and AMB reconstruction (Submitting to the JARS).

2. Basic studies for the anatomical PLB and AMB reconstruction

(1) Anatomical observation of the ACL footprint

(2) Arthroscopic observation of the ACL footprint
- Where is the femoral and tibial footprints of the PLB?
- What is the function of the PLB?
- Can we identify the PLB footprint, specifically on the femur, in arthroscopy?
  - How?

(3) Functional anatomy of the anatomical PLB and AMB reconstruction
- Can we create tibial and femoral drill holes that pass through the PLB footprints without MCL injury?
  - Principle of the one-incision technique (2 tibial tunnels and 2 femoral tunnels)

- Functional anatomy of the 2 reconstructed bundles

3. Surgical procedure for the anatomical AMB and PLB reconstruction

(1) Arthroscopy-assisted ACL reconstruction
(2) Autogenous doubled hamstring tendons
- AMB: SemiT (1/2) + Gr
- PLB: SemiT (1/2)

(3) Arthroscopic navigation for the tibial tunnels
- Development of a "guide wire navigator"

(4) Tibial tunnels that pass through each normal footprint
- PLB: 6- or 7-mm diameter
- AMB: 7- or 8- or 9-mm diameter

(5) Femoral tunnels that pass through each normal footprint
- Trans-tibial tunnel technique
- PLB: 6- or 7-mm diameter
- AMB: 7- or 8- or 9-mm diameter

(6) Tunnel positioning: For the AMB, we can represent that a guide wire (Kirschner) was inserted at the 2 or 10 O'clock orientation. Importantly, however, we cannot represent the insertion position for the PLB with the clock (not at 3 or 9 O'clock), because the footprint of the PLB in not located on the 2-dimensional clock. We will show the 3-dimensional position in pictures in my presentation.
(7) Fixation: Endobutton – Polyester tapes – double staples
(8) Initial tension: 40N for each bundle (total 80N)
(9) Standard postoperative management

4. Intra-operative Evaluation of the anatomical AMB and PLB reconstruction

(1) Intraoperative simultaneous measurement of the "length pattern" of the 2 bundles
- "Isometer" (Smith & Nephew Co, Norwood, MA)

(2) Intraoperative simultaneous measurement of the "tension pattern" of the 2 bundles
- "Tensiometer" (Custum-made load-transducer)

(3) Significant differences in both the 2 patterns between the two bundles
(4) These patterns were similar to those of the normal AMB and PLB (Kurosawa and Yasuda, CORR, 1987)

5. Clinical Results of the anatomical AMB and PLB reconstruction

(1) Semi-randomized clinical trial
G-I: Single bundle procedure
G-II: 2-bundle procedure (1 tibial and 2 femoral tunnels)
G-III: 2-bundle procedure (anatomical AMB & PLB)

(2) Minimum 1-year follow up
(3) Objective evaluation: range of motion, KT2000, and IKDC evaluation.
(4) Concerning the side-to-side anterior laxity, G-III was significantly less than G-I.
(5) Spearman's analysis showed that there was a significant correlation between the anterior laxity and the group.

6. Conclusion

(1) We have developed a novel anatomical AMB and PLB reconstruction procedure

for the ACL-deficient knee, based on the basic studies.
(2) There is a high possibility that this procedure improve the clinical results after ACL reconstruction with the hamstring graft.

Lecture: HOW I DO AN ACL RECONSTRUCTION
Tuesday, March 11, 2003 • Aotea Centre, Kupe/Hauraki Room

Freddie H. Fu, M.D., D.Sc. (Hon) and Volker Musahl, M.D.

Freddie H. Fu, M.D., D.Sc. (Hon), David Silver Professor and Chairman of the Department of Orthopaedic Surgery, Kaufmann Building Suite 1011, 3471 Fifth Avenue, Pittsburgh, PA, 15213
www.orthonet.upmc.edu, email: ffu@uoi.upmc.edu

INTRODUCTION

Approximately 250,000 ACL reconstructions/year in the USA alone
80-90% success
More then 20 different techniques available
Global panel: equal use of patella tendon and hamstring tendons,
60% of surgeons use multiple grafts [3]

Purpose of this Instructional Course Lecture
Review current *technique for ACL reconstruction*

BACKGROUND

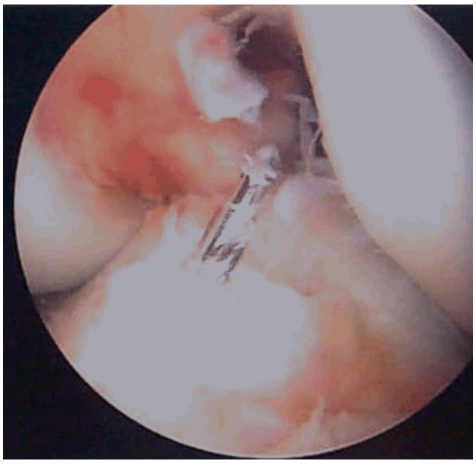
The Perfect graft
should reproduce insertion site anatomy and biomechanics,
should have complete biological incorporation
should resume neuromuscular control
does not yet exist [2]

Historically
B-PT-B grafts since late 1970s
hamstring tendon grafts since late 1980s
Quadriceps tendon grafts
synthetic graft materials
allografts
clinical outcome has not been proven significantly better in one or the other technique [1] [6]

SURGICAL TECHNIQUE:

Preparation
Graft harvest, systematic diagnostic arthroscopy
One-incision technique
Minimal notch plasty provides an impingement-free placement of the ACL graft
(After placement of the tunnels and testing for impingement utilizing a tunnel dilator, a more extensive notch plasty can be performed if impingement is present)

Tibial tunnel placement



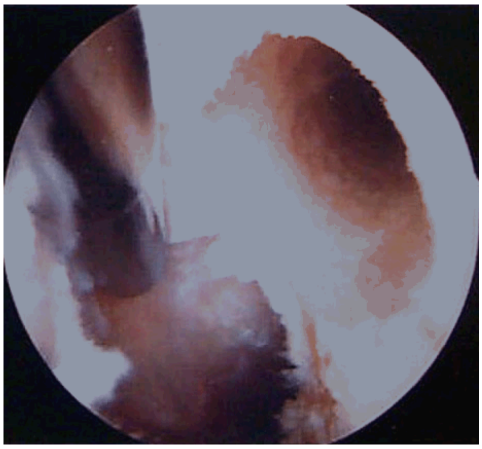
Placement in posterior half of a.-p. diameter and the center of the m.-l. diameter of the ACL footprint

Landmarks: anterior horn of the lateral meniscus
the tibial spines
the anterior border of the PCL
the intercondylar roof

Tibial tunnel drillling

Tibial tunnel aimer through the anteromedial portal set at 55°
(angle can be adjusted from 30 to 60° and matched with the length of the graft)
Entrance of guide wire medial on tibia!
Harvest bone graft from the tunnel to fill patellar donor site defect

Femoral tunnel placement

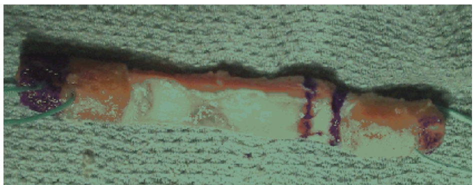


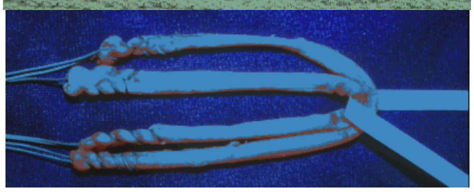
Identify posterior cortical margin of the lateral femoral condyle and probe the over-the-top position.
Avoid anterior femoral tunnel placement by mistaking the "resident's ridge" for the over-the-top position
7 mm-offset guides for anatomical tunnel position
O'clock position is 10:30 to 11:00 o'clock position for a right knee

Femoral tunnel drilling

Knee flexed at 90°
Drill in trans-tibial fashion
Length of the femoral tunnel should be the length of the femoral portion of the graft construct (drill extra 10 mm if tibial tunnel-plug mismatch is suspected).

Graft preparation





Graft preparation is completed at the back table in the meantime
Includes a mark with a sterile marking pen for visualization during arthroscopic graft insertion
Graft is inserted from the tibia using the beath pin and no. 5 Ethibond sutures

Graft fixation (B-PT-B)

Pull the construct into the femoral tunnel until femoral bone block is flush with the articular margin
Fix femoral bone block with a metal interference screw

Final fixation of the graft with the knee close to full extension, a manually applied posterior drawer, and tension on the graft [4]
Tibial fixation also with a metal interference screw.
A flexion-extension test should be performed to assure impingement-free range of motion as well as a Lachman test to assure successful fixation of the ACL replacement graft!

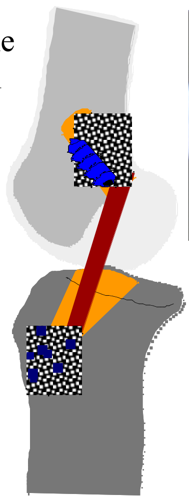
<u>Graft fixation (hamstring)</u>

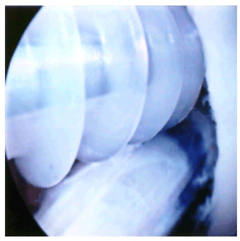
Pull construct into femoral tunnel until the mark for the femoral tendon portion is flush with the articular margin
Femoral fixation with:
- bio-absorbable interference screw
- endobutton (the graft needs to be pulled an additional 8 mm into the femoral tunnel in order to rotate the endobutton)
- Cross-pins (inserted from outside-to-in)

Tibial fixation with:
- Screw and washer
- Staple
- cross-pins
- bio-absorbable interference screws.

COMPLICATIONS AND PITFALLS

Preparation of the over-the-top position is critical
Failure to do so can result in anterior placement of the femoral socket and failure of the ACL reconstruction





If the femoral socket is placed too posterior, the back wall can be perforated, resulting in a femoral socket “blowout”

If poor fixation with an interference screw is encountered, different fixation techniques should be available

The no. 5 Ethibond sutures from the femoral bone block can be tied to a suture post that is placed in the lateral femoral cortex or an endobutton can be used.

Bio-absorbable interference screws can fail [5]

Graft tunnel mismatch in a B-PT-B graft (have different technical options available!)
The femoral bone block can remain flush with the articular margin, fix with metal interference screw and fix the tibial bone block outside the tunnel with a screw and washer or a staple

Or the femoral bone block will be pushed further up into the femoral tunnel, fix with a bio-absorbable interference screw, fix the tibial bone block with a metal interference screw inside the tunnel.

REFERENCES

1. Corry, I.S., J.M. Webb, A.J. Clingeleffer, and L.A. Pinczewski, Am J Sports Med, 1999. 27(4): p. 444-54.
2. Fu, F. and V. Musahl, J Orthop Surgery, 2001. 9: p. 77-80.
3. Harner, C.D., F. Fu, J.J. Irrgang, and T.M. Vogrin, Knee Surgery, Sports Traumatology, Arthroscopy, 2001. 9(6): p. 330-336.
4. Höher, J., A. Kanamori, J. Zeminski, F.H. Fu, and S.L.-Y. Woo, American Journal of Sports Medicine, 2000.
5. Martinek, V., R. Seil, C. Lattermann, S.C. Watkins, and F.H. Fu, Arthroscopy, 2001. 17(1): p. 73-6.
6. Otero, A.L. and L. Hutcheson, Arthroscopy, 1993. 9(2): p. 143-8.

Symposium: Patello-Femoral Instability
Tuesday, March 11, 2003 • Aotea Centre, Kupe/Hauraki Room

Chairman: Werner Mueller, Switzerland
Faculty: Donald C. Fithian, MD, USA, Alberto Pienovi, MD, Argentina, Scott F. Dye, MD, USA and Pieter Erasmus, MB CABM Med, South Africa

Müller W, Wirz D, Friederich NF

Dept. Orthopaedic Surgery & Traumatology

Kantonsspital, CH-4101 Bruderholz/Basel, Switzerland

Anatomy, Biomechanics and Dynamics of the Patellofemoral Joint

General remarks to biomechanics

The patellofemoral joint differs from other joints that are passively guided by the shape of the surfaces of congruent joint bodies such as a head in an acetabulum or by the motion guided by strong ligaments such as the cruciate and collateral ligaments in the knee [3 ,4].
The patellofemoral joint consists merely of a very mobile sesamoid bone (patella) within the quadriceps decelerator-extensor mechanism articulating on a jointlike surface of the femoral trochlea. This articulating contact surface is relatively small and changes considerably on both the patellar and the femoral side when the knee is flexed or extended. The passively guided ligamentous structures such as the medial and the lateral transverse patellar retinacula are weak in relation to the sum of the acting forces. The two longitudinal parapatellar retinacula as tendinous parts of the quadriceps decelerator and extensor mechanism also have no substantial lever arm for the passive guidance of the patella. These active steering forces of the quadriceps muscle, the iliotibial tract with tensor fasciae latae and the gluteus maximus, the hamstrings with biceps, semimembranosus, and the three pes anserinus muscles, sartorius, gracilis and semitendinosus rotate the knee and therefore move the tuberosity of the tibia to internal rotation and external rotation. The degree of this rotation varies individually from about 20° to 50°. This rotation leads to an important change in the force direction in the patellar tendon–quadriceps mechanism, a mainly dynamic, active process of centering or “subluxing” the sesamoid patella. [3 ,4].

Embryology and development

In the flexed knee of a human embryo (20 weeks) the shape of the patella differs greatly from that in an adult. It is almost quadrangular and sits deep between the condyles in the intercondylar space (transverse cross-section of the knee). During life, with the knee in extension, the patella then articulates with the trochlear surface, flattens out, and assumes its very individual shape, which depends on the trochlear shape on which it acts. Too often these individual adaptations are declared as “dysplasic patella,”without considering the dynamically changing conditions under which this sesamoid bone must apply on a corresponding femur.

moves up and become steadily broader with increasing flexion of the knee joint. The anatomical "technical" structure shows two strong bone layers: the posterior pressure side with the subchondral cortical layer and the anterior side which is the tension side, reinforced by the tension resisting galea aponeurotica (tension band function). Between these two hard cortical layers there is a thicker and more elastic layer which allows plastic deformation and therefore some adaptation to the contact surface of the trochlea. The patella becomes bent over the lateral condyle. We recently measured this deformation (elongation) in the anterior cortical tension layer in vitro on fresh frozen cadaverous knee joints. Even at a flexion angle of 90° and a quadriceps–tendon force of only 800 N the relative motion between the two points on the patella is in the order of 50 um [1 ,5]. The deformation is combined in longitudinal elongation and twist in transverse (Fig. 2). These three layers are also evident on normal radiography, especially in tangential views. Lateral magnetic resonance imaging also shows the thickest layer of patellar cartilage having contact with the trochlea in the 30° knee flexion position for the heel strike. Each patellar implant of a total knee rep lacement (TKR) changes this finely tuned structural system of the patella. This may be the reason why so many problems occur with the patella in TKR. These problems include limitation of motion (patella too thick), fractures, fatigue fractures, chronic subluxation, dislocation, sometimes early loosening, and pain.

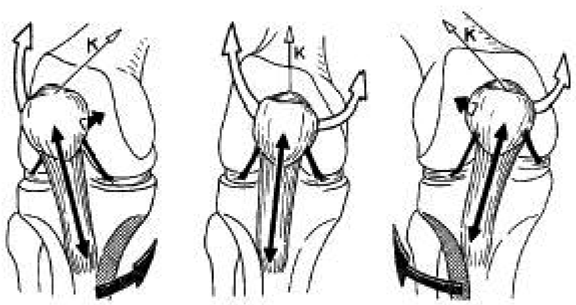


Fig. 1 Rotation of the tibia changes the force direction in the patellar tendon–quadriceps mechanism, principally a dynamic, active process of centering or "subluxing" the sesamoid patella. (From [3], with permission)

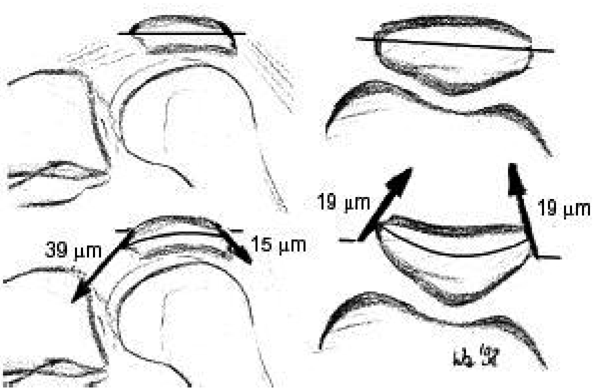


Fig. 2 Vectors of patella deformation measured at 60° of knee flexion with no load

Literature

1. Arnold MP, Friederich NF, Widmer H, Muller W (1998). Patellar substitution in total knee prosthesis--is it important? Orthopäde 27(9):637-41.
2. Goodfellow DS, Hungerford S, Zindel M (1976) Patello-femoral mechanics and pathology. 1. Functional anatomy of the patello-femoral joint. J Bone Joint Surg Br 58:287–290 .
3. Müller W (1982) Das Knie. Form, Funktion und ligamentäre Wiederherstellungschirurgie. Springer, Berlin Heidelberg New York .
4. Müller W, Wirz D (2000) Anatomie, Biomechanik und Dynamik des Patellofemoralgelenkes. In: Wirth CJ, Rudert M (ed): Das patellofemorale Schmerzsyndrom. Steinkopf-Verlag, Darmstadt.
5 . Müller W, Wirz D: The patella in total knee replacement: Does it matter? 750 LCS total knee replacements without sesurfacing of the patella (2001). Knee Surg. Sports Traumatol. Arthrosc. 9 (Suppl 1): S24-26

ISAKOS SYMPOSIUM

Patellofemoral Instability
Auckland, New Zealand
March 11, 2003
Chairman: Werner Müller, MD

What is the Source of Pain in Patellofemoral Instability?

Scott F. Dye, M.D.
Associate Clinical Professor
of Orthopaedic Surgery
University of California, San Francisco

NOTES

The knee is one of the most complicated systems in the known universe.

Patients with patellofemoral problems represent the most controversial group of orthopaedic patients.

The kinematics of the patellofemoral system results from both dynamic (muscular) and static (retinacular and ligamentous) restraints.

The knee functions as a type of living biological transmission whose purpose is to accept, transfer, and dissipate loads between components, and yet still maintain tissue homeostasis.

The patellofemoral system can be viewed as a slide bearing within this living transmission. The muscles act as living engines in concentric contraction and as dampening and shock absorbing systems in eccentric contraction (absorbing greater than 3 times the energy that is generated in motive forces). The highest loads in the entire musculoskeletal system are generated, transferred, and dissipated within the patellofemoral system, often nearing or exceeding biologic tissue load acceptance and transference capacity.

The etiology of anterior knee pain in patients with patellofemoral instability probably results from a mosaic of pathophysiologic processes (loss of tissue homeostasis) of *innervated structures*. A neurosensory map is shown in Figure 1. These structures include synovium, retinaculum, and the intraosseous environment of the patella and trochlear bone. Any combination of these tissues can be traumatized and irritated leading to the perception of pain with each instability episode. With multiple recurrent instability episodes, these tissues can be chronically perturbed leading to the development of persistent anterior knee pain.

NOTES

Many patients with intermittent patellofemoral dislocations, however, are pain-free between episodes.

One should also keep in mind that recurrent synovial/fat pad impingement without malalignment or instability can cause sharp severe pain leading to reflex inhibition of muscles and giving way of the knee, thus simulating a subluxation/instability event. (When, in fact, instability is *not present*.) This possibility has obvious therapeutic implications regarding appropriate physical therapy and surgical recommendations.

The capacity of the patellofemoral components (or any musculoskeletal system) to accept, transfer, and dissipate loads and yet still maintain tissue homeostasis can be represented by the concept of the Envelope of Function. In its simplest form, the Envelope of Function is a load/frequency distribution that delineates a range of safe loading for a given musculoskeletal system over a given period of time.

Within the Envelope of Function is the region termed the Zone of Homeostasis. Loads that exceed the Envelope of Function but are insufficient to cause a macrostructural failure can be termed the Zone of Supraphysiologic Overload. Stress fractures and strains, etc, are example of injuries secondary to loading within this region. If sufficiently high forces are placed across the patellofemoral system, macrostructural failure can occur. If loads are diminished over prolonged periods of time, one can have loss of tissue homeostasis, as well, manifested by muscle atrophy and mineral loss from bone, that is defined by a lower threshold limit and is called the Zone of Subphysiologic Underload.

The goal of therapy for patellofemoral (and all orthopaedic) patients should be to help restore the Envelope of Function as safely and predictably as possible. In my experience, the vast majority of patients with patellofemoral pain respond well to a conservative program that emphasizes rigorous protection of the patellofemoral joint (i.e., keeping such a joint within its Envelope of Function) long enough for tissue healing to occur. The process can be helped by the addition of gentle rehabilitation and anti-inflammatory treatment to include intermittent tissue cooling and non-steroidal anti-inflammatory medication. One thus wishes to create the optimal internal environment, biomechanically, biochemically, and biophysically, to allow tissue homeostasis to be restored as safely as possible.

NOTES

The concept of chondromalacia and/or malalignment as the cause of most anterior knee pain is now being questioned and has failed to explain the vast majority of patients with patellofemoral pain symptoms.

The smaller subset of patients with overt patellofemoral instability, but without significant pain, may be understood and treated successfully from a straight-forward biomechanical perspective.

The primary pathophysiologic events leading to the persistence of patellofemoral symptoms often is the diminishment of the Envelope of Function following a supraphysiologic overloading event or events, such that many activities of daily living then become recurrent traumatic triggers subverting normal healing properties (certain activities of daily living become loads within the Zone of Supraphysiologic Overload).

Figure 1

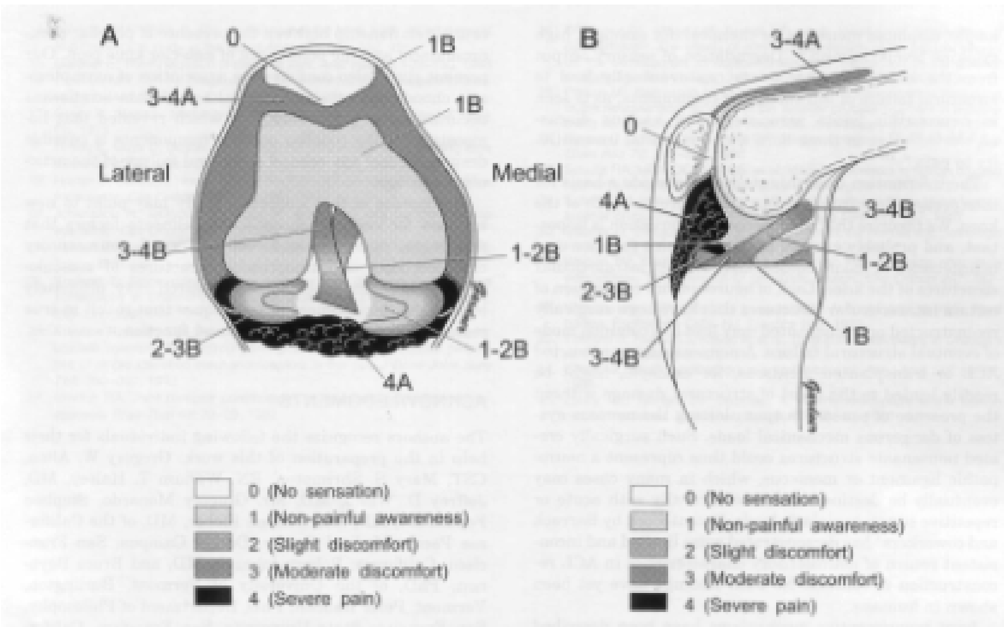
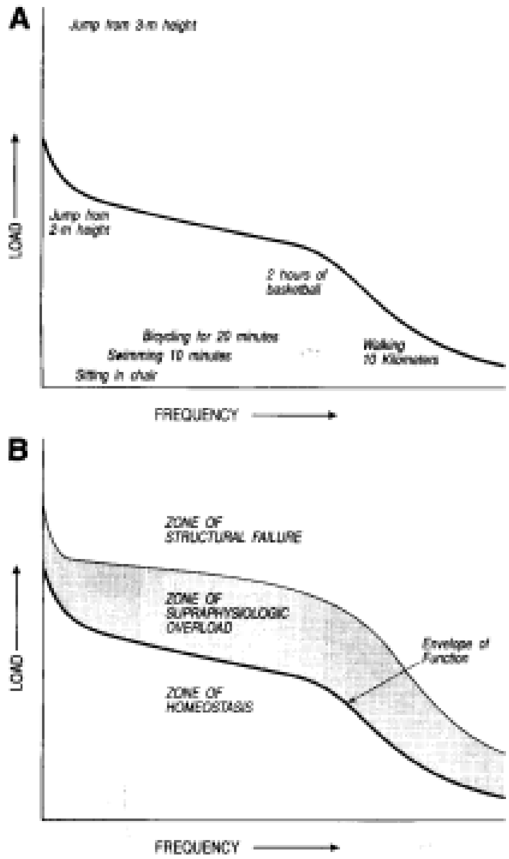


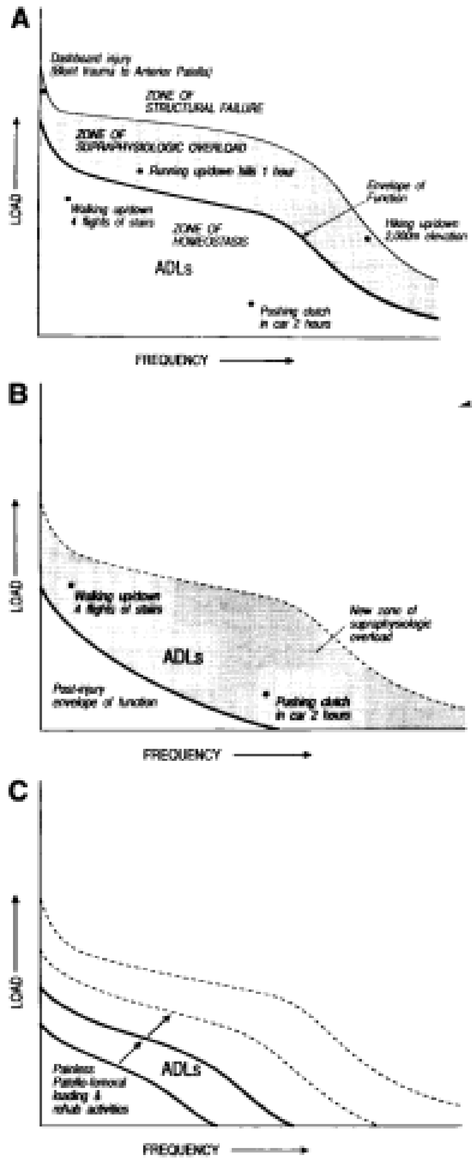
Figure 1. Coronal (A) and sagittal (B) schematic representations of the conscious neurosensory findings of the intraarticular structures of the knee. The right knee is illustrated. A, accurate spatial localization; B, poorly localized sensation.

Figure 2



(A) Graph representing the "envelope of function" for an athletically active young adult. All loading examples, except the jump from a 3m height, are within the envelope for this particular knee. The shape of the envelope of function represented here is an idealized theoretical model. The actual loads transmitted across an individual knee under these different conditions are variable because of multiple complex factors, including dynamic center of gravity, the rate of load application, and the angles of flexion and rotation. The limits of the envelope of function for the joint of an actual patient are probably more complex. (B) Graph showing the three different zones of loading across a joint. The area within the envelope of function is the zone of homeostasis. The region of loading greater than that within the envelope of function but insufficient to cause macrostructural damage is the zone of supraphysiologic overload. The region of loading great enough to cause macrostructural damage is the zone of structural failure. (Reprinted with permission.[10])

Figure 3



Changing envelopes of function in patients with patellofemoral trauma. (A) supraphysiologic loads outside the envelope: a dashboard injury, running up hill one hour, and hiking downhill 2000 meters. (B) Diminished envelope of function after supraphysiologic patellofemoral loading showing that activities of daily living and activities such as climbing four flights of stairs and pushing a clutch in a vehicle for 2 hours have become supraphysiologic loads, leading to recurrent loss of tissue homeostasis and continuance of peripatellar symptoms. (C) Incremental expansion of the diminished envelope of function by restricting patellofemoral loading to within the envelope.

REFERENCES:

1. Dye SF, Boll DH, Dunigan PE, et al: An Analysis of Objective Measurements Including Radionuclide Imaging in Young Patients with Patellofemoral Pain. *Am J Sports Med* 13:432, 1985.
2. Dye SF, Boll DA: Radionuclide Imaging of the Patellofemoral Joint in Young Adults with Anterior Knee Pain. *Ortho Clin North Am* 17:249-262, 1986.
3. Dye SF, Daniel JS, Fry PF, et al: Correlation of Increased Scintigraphic Activity and Patellar Osseous Pathology in Young Adults with Patellofemoral Pain. *Ortho Transact* 10:479, 1986.
4. Dye SF, Peartree PK: Sequential Radionuclide Imaging of the Patellofemoral Joint in Symptomatic Young Adults. *Am J Sports Med* 17:727, 1989.
5. Rothschild PA, Domesek JM, Dye SF, et al: MR Imaging of the Knee with a 0.064-T Permanent Magnet. *Radiology* 175:775-778, 1990.
6. Dye SF: Radionuclide Imaging of the Knee. In: Knee Surgery, Current Practice, ed. by Aichroth P, Cannon WD. London: Martin Dunnitz, pp 38-44, 1992.
7. Dye SF: Patellofemoral Anatomy. In: The Patellofemoral Joint. ed. by Fox JM, Del Pizzo W. New York:: McGraw Hill. 1993, 1-11.
8. Dye SF, Chew M, McBride JT, Sostre G: Restoration of Osseous Homeostasis of the Knee Following Meniscal Surgery. *Ortho Transact* 16:725, 1992.
9. Dye SF, Chew MH: Restoration of osseous homeostasis after anterior cruciate ligament reconstruction. *Am J Sports Med* 1993, 21:748-750.
10. Dye SF: Functional Anatomy and Biomechanics of the Patellofemoral Joint. In: The Knee. ed. by WN Scott, St. Louis: Mosby. 1994, 381-389.
11. Dye SF: The knee as a biologic transmission with an envelope of function. *Clin Orthop* 1996, 325:10-18.
12. Dye SF, Vaupel GL, Dye CC: Conscious Neurosensory Mapping of the Internal Structures of the Human Knee without Intra-articular Anesthesia. *Am J Sports Med* 1998, 26:773-777.
13. Dye SF, Wojtys EM, Fu FH, Fithian DC, Gillquist J: Factors Contributing to Function of the Knee Joint after Injury or Reconstruction of the Anterior Cruciate Ligament. *J. Bone Joint Surg* 1998, 80A:1380-1393.
14. Dye SF, Stäubli HU, Beidert RM, Vaupel GL: The mosaic of pathophysiology causing patellofemoral pain: Therapeutic implications. *Op Tech Sports* 1999, 7:46-54.
15. Dye SF, Merchant A: Magnetic resonance imaging of articular cartilage in the knee. An evaluation with use of fast-spin-echo imaging. *J Bone Joint Surg.* 81(9):1349-50, 1999.
16. Dye SF: The use of technetium scintigraphy to evaluate early arthrosis of the knee. The Arthritic Knee CD-ROM. Brian Cole, MD, (ed), Rosemont, IL, AAOS Multimedia Productions, 2000.
17. Dye SF, Campagna-Pinto D, Dye CC, et al: Soft Tissue Anatomy Anterior to the Human Patella. JBJS (Am) in press 2003.
18. Fulkerson JP: Disorders of the patellofemoral joint, 3rd Ed., Baltimore, Williams & Wilkins, 1997.
19. Grelsamer RP, McConnell J: The Patella. A Team Approach. Gaithersburg, MD, Aspen Publishers, 1998.

Scott F. Dye, M.D.
45 Castro Street, Suite 117
San Francisco, CA 94114
(415) 861-9966 - Voice / (415) 861-0174 - Fax
e-mail - SFDyeMD@AOL.com

How I treat patellofemoral instabilities

Symposium S7
P.J.Erasmus MD
South Africa

Aim:

Stabilize the patella without causing late stage patellofemoral degeneration

Principle:

"Form follows function"; this also applies to the patella and the trochlea. Surgery that leads to marked changes in this relationship might lead to late stage patellofemoral degeneration. The aim of surgery should be to be to stabilize the patella without radically changing the position of the patella in relation to the trochlea.

Procedures:

1. Reconstruction of the patellofemoral ligaments: Usually the procedure of choice in posttraumatic instabilities with relative normal patella height and alignment.
2. Tibial tubercle osteotomies: Procedure indicated in cases of high riding maligned patella.
3. Trocleaplasty: Rarely indicated as it might lead to late patellofemoral degeneration
4. Combined procedures: a combination of more than one procedure might be necessary in some cases

December 2002

Lecture: Biology of Extra-articular Tendon-bone Healing
Wednesday, March 12, 2003 • Aotea Centre, Kupe/Hauraki Room

W.R. Walsh, Ph.D.
Orthopaedic Research Laboratories, Division of Surgery, University of New South Wales, Prince of Wales Hospital
Graduate School for Biomedical Engineering
University of New South Wales
Sydney Children's Hospital, Division of Surgery
Sydney, NSW Australia, W.Walsh@unsw.edu.au

An improved understanding of the processes governing tendon incorporation into bone has applications in tendon repair and attachment to underlying bone, selection and securing of tendon grafts and in post-operative rehabilitation. The ultra-structural features of the healing tendon-bone interface involve the organisation of the initial cellular vascular infiltrate to an extra-cellular collagen rich matrix. This ultimately progresses to a continuum between the graft and the adjacent bone, which can withstand applied loads. A number of recent studies have reported tendon-bone healing gross morphology in a variety of models. The molecular events (protein expression) at the healing tendon-bone interface have yet to be fully characterised. In addition, reports on the long-term healing properties of extraarticular tendon-bone healing are limited.

This presentation will examine extraarticular tendon-bone healing using a well-established ovine patellar tendon model. The temporal expression of growth factors and BMPs and the Smad family during normal repair and the long-term biomechanical properties of this interface will be explored. Understanding BMP/TGF-β signal transduction pathways during tendon-bone healing represents an important step in the search for therapeutic strategies designed to augment or control this process. Finally, methods of non-invasive stimulation to augment tendon-bone healing will be discussed.

Symposium: Practical Tips for Hand and Wrist Arthroscopy
Wednesday, March 12, 2003 • Kupe/Hauraki Room

Chairman: Anastasios Georgoulis, Greece
Faculty: Pak Cheong Ho, MD, Gregory Bain, FRACS, Australia and Gary Poehling, MD, USA

Technical tips during the learning curve of endoscopic carpal tunnel release (ECTR)

Anastasios Georgoulis MD, Associate Professor,
Head of Arthroscopy and Sports Medicine,
Dept of Orthopaedics,
University of Ioannina, Greece
e-mail: ageorg@cc.uoi.gr   Fax: 0030 26510 74 770,
Address: P.O.Box 1012, Ioannina 45 110, Greece

Endoscopic carpal tunnel release (ECTR) provides early post-op comfort and faster recovery of strength compared to the open release but the method is not widely used because of criticism from hand surgeons that severe complications may occur during the learning curve. ECTR has the same rate of complications as open CTR.

To assist colleagues who wish to begin performing ECTR, we would like to underline the following points that are the result of our experience in undertaking ECTR and in teaching the method in cadaveric courses or to young residents.

The most severe complications are:

Injury to the **ulnar nerve**
median nerve
cutaneous palmaris branch
finger nerve
palmar arc

Division of one of the **flexor tendons**

Related anatomy of the wrist

The surgeon must be very conversant with the anatomy of the region. There are some landmarks that can be very helpful in our orientation. They are:

Flexor carpi ulnaris – os pisiforme

Flexor palmaris longus

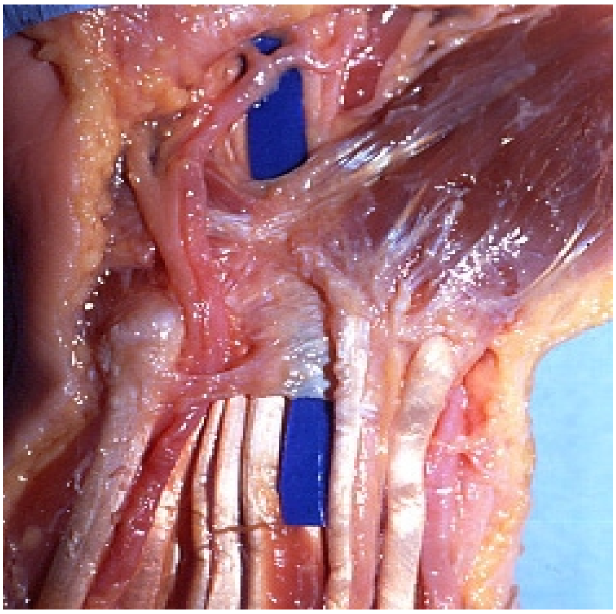
Flexor carpi radialis

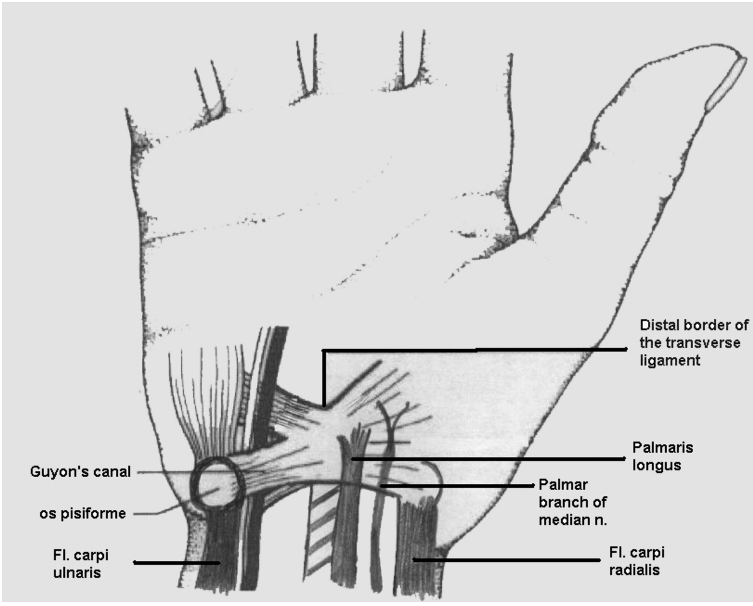
Proximal crease of the palmar side of the wrist

Distal crease of the palmar side of the wrist
Distal margin of the thenar .

–

1. The ulnar nerve and artery are located parallel and close to the fl. carpi ulnaris. We have to work at least 2 cm radially to the fl. carpi ulnaris – os pisiforme to avoid introduction in the Guyon's canal where the ulnar nerve and artery are located.





2. The palmar branch of the median nerve is located parallel to and between fl. Carpi radialis and fl. palmaris longus. We have to introduce our instruments ulnarly to the fl. palmaris longus to avoid laceration of this nerve.

3. The median nerve is located under both the palmaris longus and the fascia. This means that when we open the fascia immediately below the palmaris longus we can identify and protect the median nerve.

4. The palmar arc is located about 1.5-2cm distal to the distal border of the carpal ligament. It must be identified with palpation and visualization and we must avoid working distally to it

5. The fibres of the carpal ligament are transverse but often covered by synovial tissue and so not easily identifiable. Before cutting anything, we have to palpate and visualize the transverse fibres. The synovial tissue that covers these transverse fibres has to be removed.

6. The carpal ligament extends from the distal crease of the palmar surface of the wrist to the distal margin of the thenar. The best entry point is the proximal crease of the wrist. Here the fascia is identifiable but has not yet become a thick ligament. Furthermore we can work more easily here as there is not a lot of subcutaneous fat as is found in the distal crease of the wrist.

Surgical technique

The most useful methods are:

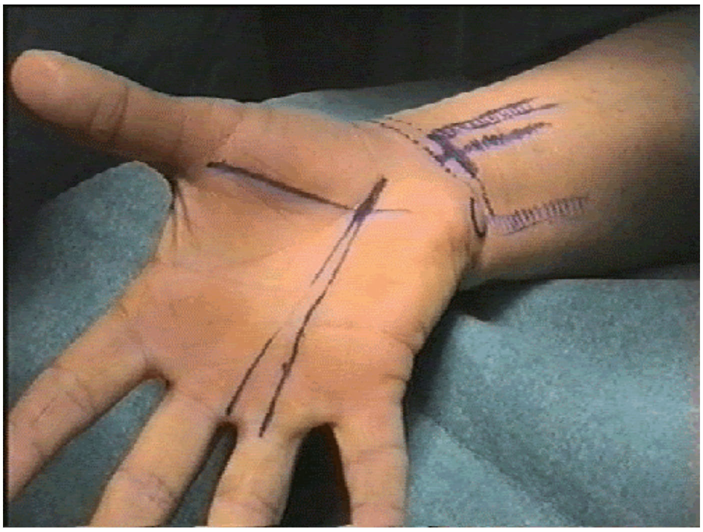
1. The two incision technique of Chow

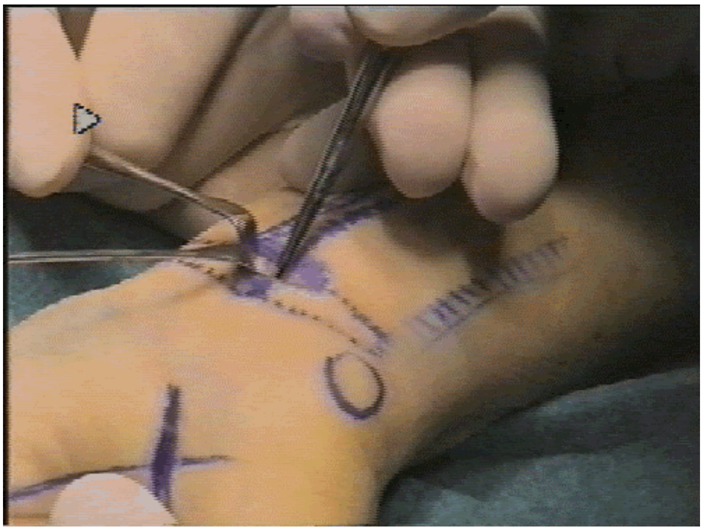
2. The one incision technique of Agee

Both techniques are very effective and if one follows the correct steps the result will be successful and we can avoid the complications recognized as occurring during the learning curve.

It is recommended that ECTR be performed under local anaesthesia and using a tourniquet. It is very important to inject small amounts of anaesthetic only into the skin to avoid oedema and limitation of visualization during the procedure.

For both techniques it is essential to recognize the fl. palmaris longus and to make the incision ulnarly to it. The use of magnifying lenses to identify all the anatomic features is of great value as is experience in performing open CTS.





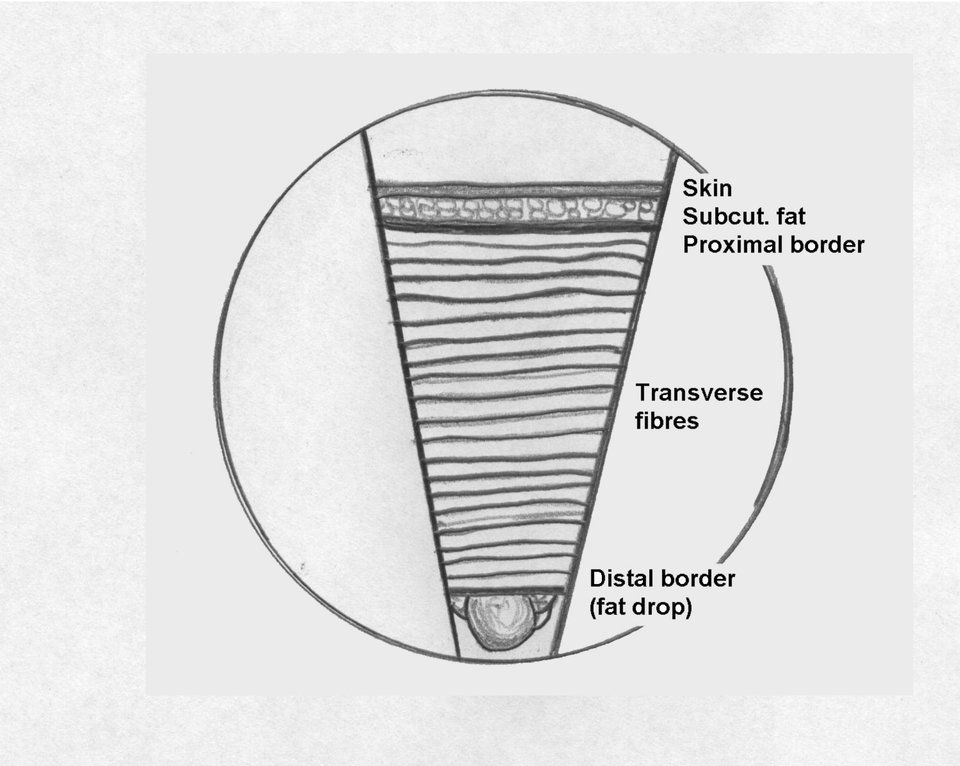
At first we try to find a path under the carpal ligament using a curved elevator,. With this curved elevator we can always palpate the transverse fibres of the ligament and also the distal border of the ligament.

We always separate the synovium from the tranverse fibres to aid visualization. In the one portal technique we do this with a special instrument before introducing the optic. In the two incision technique this is done using a probe that is introduced from the distal incision to be performed immediately at the distal border of the tranverse ligament.

We cut the ligament after four conditions have been fulfilled:

1. Identification of the proximal border of the carpal ligament
2. Identification (fat drop) of the distal border of the carpal ligament
3. Visualisation of the transverse fibres of the carpal ligament
4. Palpation of the transverse fibres of the carpal ligament (wash board effect

We are sure that all the fibres are divided when fat drops into our view.



Bearing all of the above in mind we can treat a very common disease using a modern arthroscopic technique without severe complications and with minimal inconvenience to the patient.

References

Boeckstyns 1999 Does ECTR have a higher rate of complications than open CTR?.

Born 1995 Cutaneous distribution of the ulnar nerve in the palm after ECTR

Bozentka 1995. Complications of ECTR.

Chow1994. Ulnar nerve transection as a complication of two-portal ECTR.

De Smet 1995 Transection of the motor branch of the ulnar n. in two-portal ECTR

del Pinal 1997. Total ulnar nerve transection during ECTR.

Einhorn 1996 Pitfalls of ECTR.

Kropfl 1996. Technique, results and risks of ECTR.

Luallin1993. Incidental Guyon's canal release during attempted ECTR:

Muller 2000. ECTR: results with special consideration to possible complications.

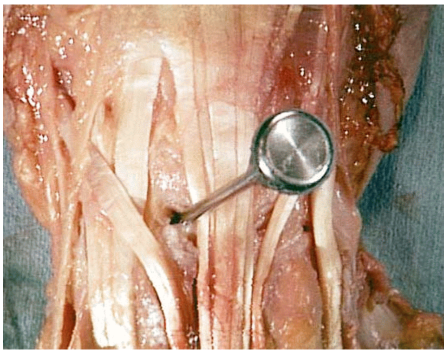
Murphy 1994. Major neurovascular complications of ECTR.

Nakamura 1999 Longitudinal Median Nerve Conduction Studies After ECTR.

Shinya 1995. Risk and complications in ECTR

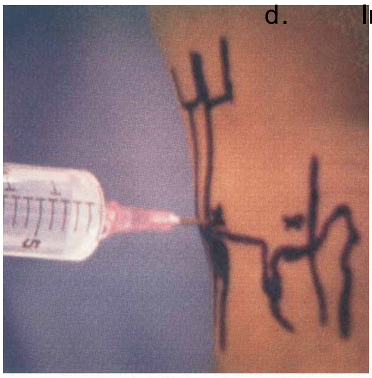
Stark 1994. Ulnar nerve transection as a complication of two-portal ECTR

III. Approach to the Radiocarpal Joint

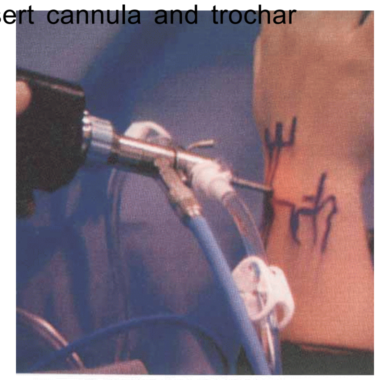


A. 3-4 portal

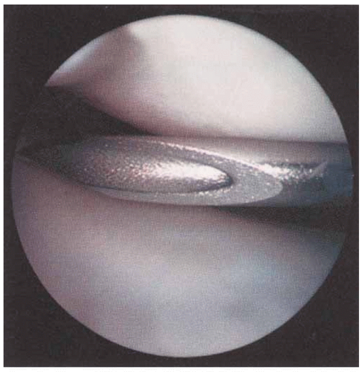
1. Anatomy
   a. Radial - EPL
   b. Ulnar - EDC
   c. Proximal - Radius
   d. Distal - Scapholunate ligament
2. Establish 3-4 portal
   a. Insert 18 gauge needle in 3-4 portal
   b. Distend joint: 5-10 cc
   c. Incise skin
   d. Insert cannula and trochar



Joint Distension



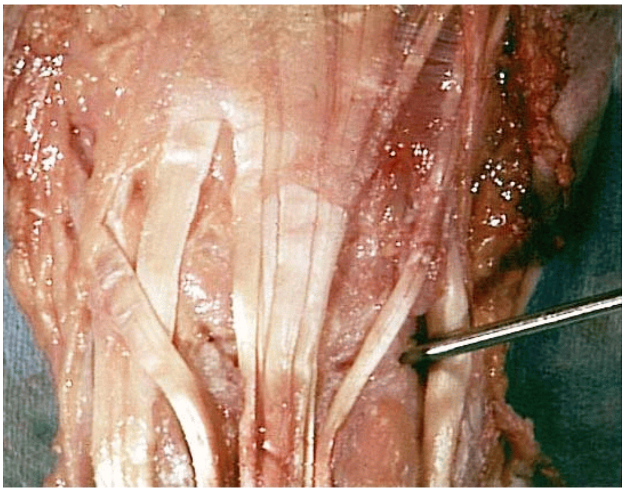
Scope in 3-4 Portal



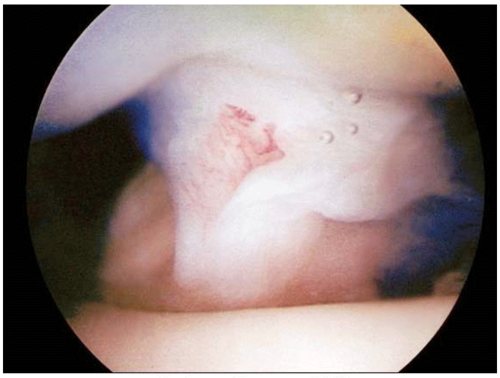
Needle in 6-R Portal

B. 6-R Portal

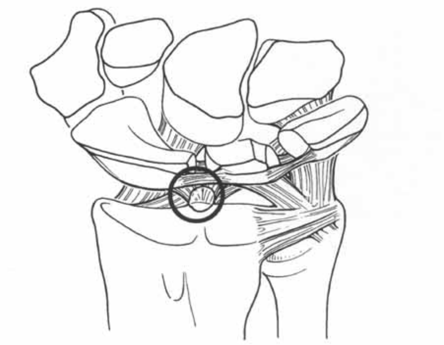
1. Anatomy
   a. Radial - EDQ
   b. Ulnar - ECU
   c. Proximal - Ulnar head
   d. Distal - Lunate
2. Establish portal 6-R
   a. Insert 18 gauge needle in 6-R portal
   b. Directly view needle insertion
   c. Adjust insertion site of the needle to center of the joint
   d. Incise skin
   e. Insert trochar



IV. Arthroscopic Anatomy

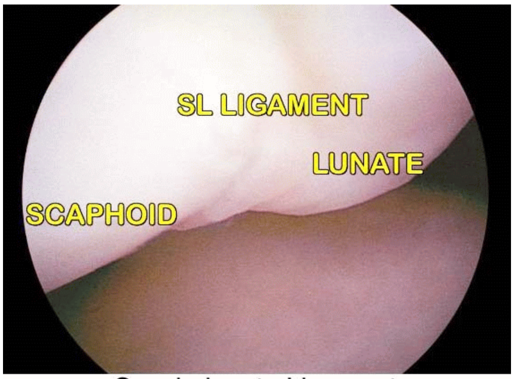


Fat pad over RSL



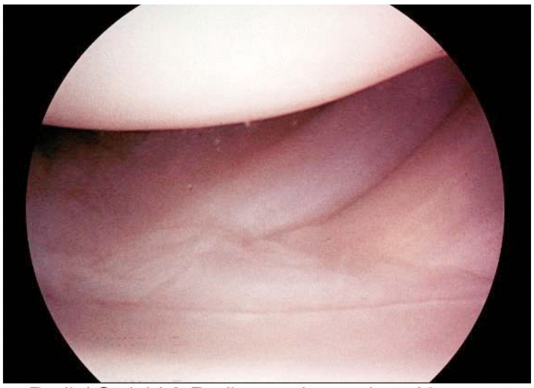
Visualize through either 3-4 or 6-R portal

The fat pad over the radio-scapho-lunate ligament is a convenient orientation point and usually the first thing seen at initial insertion in the 3-4 portal.

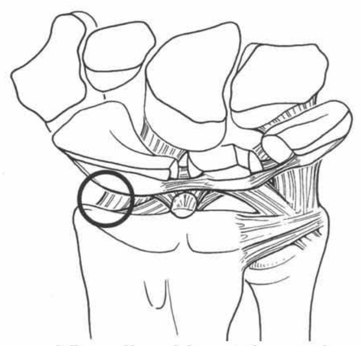


Scapholunate Ligament

The scapholunate ligament is a thin proximal ligament that is easily defined by palpation with a probe. It has three anatomic regions. The most visible is the proximal which is composed of fibrocartilage and has little strength. The stronger regions are the Dorsal and Palmar. They are made of collagen fibers. Degenerative tears most often occur in the Proximal portion.



Radial Styloid & Radio-scapho-capitate Ligament



Visualized best through 3-4 portal

This is the radial-most extent of the radiocarpal joint. The scaphoid is above, and the radial carpal ligaments are seen palmarly.

Arthroscopic Management of a Traumatised Wrist

Comparison of plain radiographs and arthroscopic findings for assessment of scapholunate instability

Gregory Bain, FRACS, Australia

75 patients with disorders of the wrist that had undergone arthroscopy were assessed for evidence of scapholunate instability.

These findings were compared to the measurements obtained from standardised radiographs which included specialising instability series such as clenched fist views and scapholunate angle views [1].

The findings included:

1. All patients who had a SL interval of > 4mm had a SL ligament tear.
2. 90% of patients with a SL interval of > 3mm had a SL instability.
3. All patients with a SL angle of > 80° had a SL ligament instability.
4. 90% of patients with a SL angle > 60° had instability. There was a difference > 0.5mm between the SL interval on the injured wrist compared to the opposite wrist, this was usually associated with instability.
5. The clenched fist view had the highest correlation with arthroscopic findings for assessment of SL instability.
6. Recommended plain radiographs included bilateral PA clenched fist, true lateral plain radiographs.

Arthroscopic assisted reduction and percutaneous fixation of acute scapholunate injuries.

Twenty patients were prospectively managed with arthroscopic assisted fixation of scapholunate interval.

The indications for inclusion in the study were patients who presented with an acute injury (<6 weeks) to the wrist, with localised tenderness over the scapholunate ligament, who had pain or instability with Watson's scapholunate instability test.

The instability of the scapholunate interval was graded using the Geissler classification.

The scapholunate interval was stabilised with two 1.6 mm non-threaded K-wires. The K-wires were advanced into the scaphoid just distal to the radial styloid under fluoroscopy control. The surgeon then manipulates the scapholunate interval. While viewed through the midcarpal joint the joy sticks were manipulated until an anatomic reduction was obtained and two pre-positioned K-wires in the scaphoid are advanced (Figure 1).

The patients were independently assessed and a standardised set of radiographs were performed.

The scapholunate interval was preoperative 3.3 mm, at follow-up with K-wires in-situ 3.0 mm, at final follow-up 3.2 mm and increased to 3.9 m with a clenched fist view. The contralateral was 2.7 mm.

Even in the acute setting of delay for less than 6 weeks, it is common to have a persistent diastasis of the SL interval. We no longer use this technique.

Management of distal radial fractures

Arthroscopy provides a magnified view to allow positioning of the articular fragments within the radiocarpal joint with precision. It is well known that steps of greater than 2mm are associated with 100% chance of degenerative arthritis [2]. Arthroscopy also allows assessment of the associated carpal injuries including triangular fibrocartilage tears, scapholunate ligament tears and osteochondral fractures.

Fluoroscopy is valuable as it allows assessment of the position of fixation devices such as K-wires; it allows assessment of the metaphyseal reduction and assessment of fracture and carpal stability.

Fracture fragments can be manipulated with the aid of the arthroscopic probe, K-wires, which are inserted into the fracture fragments can be used as joy sticks or by placing the K-wires below the major fragments and used as buttress pins as described by Kapandji. Under arthroscopic vision the position of the fracture fragments can be confirmed to be satisfactory prior to advancing the wire. Once a satisfactory reduction has been obtained the distal radius can then be gently mobilised under the fluoroscopic control to assess whether stability has been obtained. If stability has not obtained extra fixation is required either in the form of an external fixateur or further K-wires (Figure 2).

With the aid of fluoroscopy we were able to obtain a reduction with no discernible step on post operative radiographs in the majority of cases [3]. In the majority of the other patients there was a step of less than 1mm. Knirk and Jupiter had reported that the size of the step was correlated with degenerative arthritis as identified on post operative radiographs. We have identified the incidents of pain as identified on a visual analogue scale, is correlated to the size of the steps.

Arthroscopy for mid carpal instability

We have used the bipolar radiofrequency arthroscopic probe to perform capsular shrinkage on the ulnar limb of the arcuate ligament between the hamate and triquetrum. Those patients in which there was gross instability we also performed radiofrequency on the dorsal aspect of the radiocarpal joint. This did provide an improvement with regard to the mid carpal instability symptoms. One patient in the series did have a recurrence of their symptoms after a period of approximately 3 years.

The present recommendation on the role of this technique that it should remain limited. We still give this option to the patient but do point out that in the intermediate or long term that they may still have further trouble and that it may be a better option to proceed with an immediate limited wrist fusion.

KUPE/HAURAKI

Arthroscopic capsular release

For those patients who have post traumatic stiffness of the wrist an arthroscopic capsular release and synovectomy can provide a significant improvement with regard to their range of motion and strength. This technique should be reserved for those patients in which the articular surfaces have minimal damage or incongruenty and where the principal restriction in the range of motion is within the joint capsule. An arthroscopic capsular release can be performed involving the volar radiocarpal ligaments. Care should be taken to preserve at least the radial aspect of the RSC ligament to prevent ulnar translocation of the carpus [4]. Release of the volar capsule will often provide a significant improvement of the wrist extension.

In those patients in which there is a restriction in the range of wrist flexion, a dorsal arthroscopic capsulotomy can be performed. A volar wrist portal enables the surgeon to have a direct visualisation of the dorsal capsule to assist with regard to the capsulotomy.

References

1. Moneim M. The tangential posteroanterior radiograph to demonstrate scapholunate dissociation. J Bone Joint Surg, 1981: 63A1324-1326.

2. Knirk JL, Jupiter JB. Intrarticular fractures of the distal end of the radius in young adults. Journal of Bone and Joint Surgery, 1986: 68A647-659.

3. Mehta JA, Bain GI, Heptinstall RJ. Anatomical reduction of intra-articular fractures of the distal radius. An arthroscopically-assisted approach. J Bone Joint Surg Br, 2000: 82(1): 79-86.

4. Verhellen R, Bain GI. Arthroscopic capsular release for contracture of the wrist: a new technique. Arthroscopy, 2000: 16(1): 106-110.

List of Figures

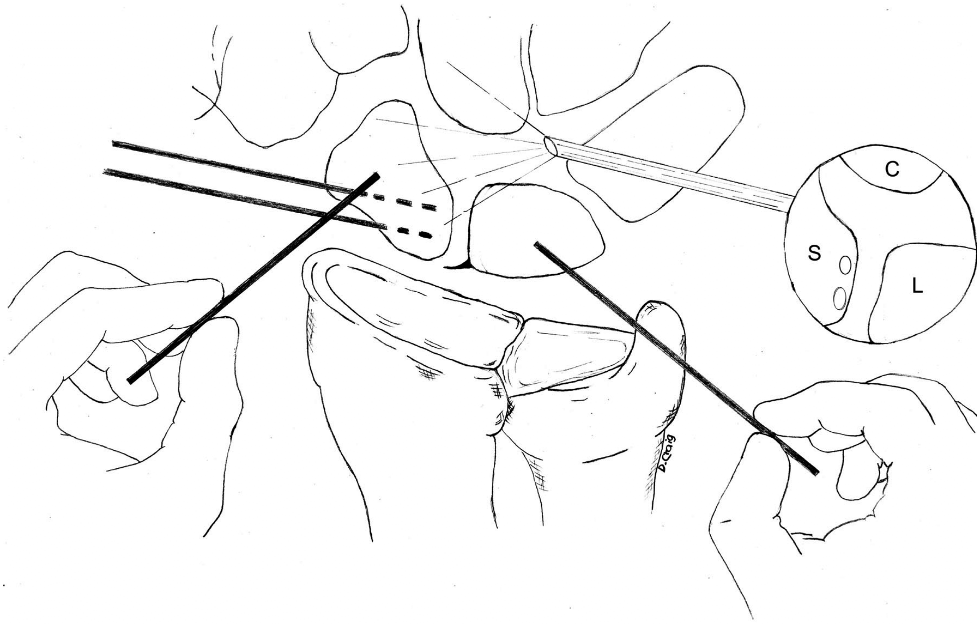


Figure 1

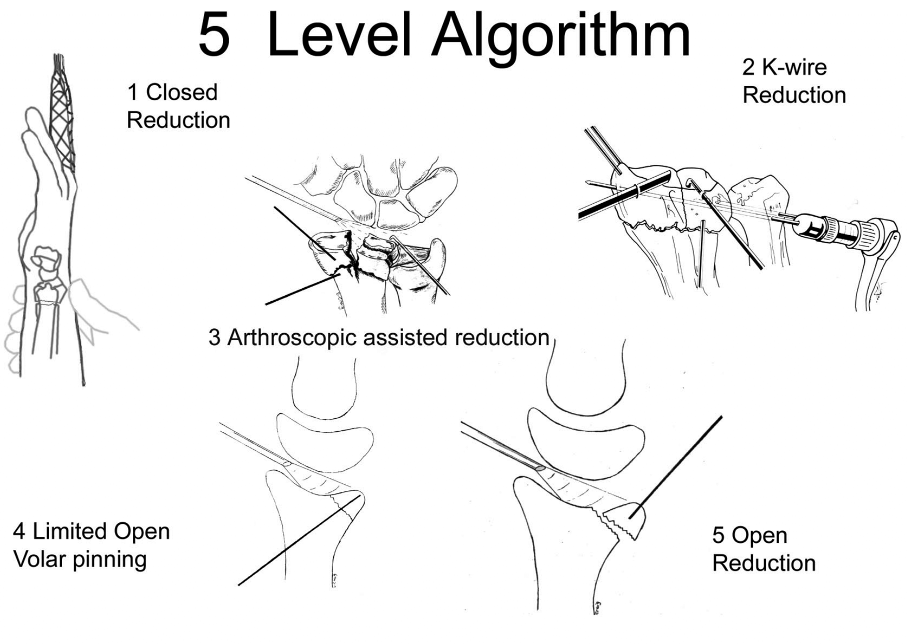


Figure 2

Symposium: Outcomes Research
Wednesday, March 12, 2003 • Aotea Centre, Kupe/Hauraki Room

Chairman: Robert G. Marx,,MD, MSc, FRCSC, USA
Faculty: Jon Karlsson, MD, PhD, Sweden and Nicola Maffulli, MD, MS, PhD, FRCS(Orth), England

I.	Marx:	Introduction to Clinical Research Methodology
II.	Karlsson:	Overview of Evidenced Based Medicine in Sports Medicine Applicability of Randomized Controlled Trials in Sports Medicine
III.	Maffulli:	Practical Aspects of Randomization
IV.	Marx:	Clinical Measurement and Sample Size in Randomized Clinical Trials

Symposium: Comparative Animal Models in Orthopaedic Research
Wednesday, March 12, 2003 • Aotea Centre, Kupe/Hauraki Room

William G. Rodkey, DVM, Diplomate, ACVS, Chairman
C. Wayne McIlwraith, DVM, PhD, Diplomate, ACVS, Co-Chairman

Introduction

This symposium will examine various knee and other joint problems commonly seen in domestic animals. The etiologies and pathologies will be compared to similar problems seen in human patients, especially active athletes. These discussions will include the natural history in both large (equine) and small (canine) animals which develop these conditions through routine or competitive athletic activities. Speakers will explain how various clinical problems are managed, and how their own research efforts have changed the standard of care for both animal and human athletes. Emphasis will be on ligaments, articular cartilage, and the meniscus.

Articular Cartilage

There is a remarkable similarity between problems facing the equine athlete and the human athlete. In the cyclic trauma of competitiveness, the complete spectrum of traumatic arthritis including synovitis, capsulitis, intraarticular fracture, osteochondritis dissecans and osteoarthritis occur. We present two different models and some associated research findings.

Osteochondral Fragment - Exercise Model For Osteoarthritis

Osteochondral chip fragments occur commonly in the carpus of the racehorse, and if these fragments are not removed promptly (arthroscopically), severe osteoarthritis ensues. We used this knowledge to develop a chip model of osteoarthritis in the horse. There are a number of advantages including 1) the chip fragment is made arthroscopically; 2) exercise can be controlled; 3) athletic exercise is introduced two weeks after chip fragment creation; and 4) the degree of lameness, synovitis and osteoarthritis is quite consistent among horses.

Briefly, the arthroscope is placed between the common and lateral digital extensor tendons to enter the intercarpal (midcarpal) joint. An instrument portal is then made over the surface of the third carpal bone which allows introduction of an 8 mm curved osteotome, and a fragment is made by driving the osteotome perpendicular to the articular surface of the radial carpal bone. A motorized burr is then used to burr back the defect to 15 mm in width and the debris is left in the joint. At two weeks, horses are put on the treadmill in a regimen of two minutes trot and three minutes gallop (25-28 mph) and two minutes trot instituted daily. All studies done in the past have used eight horses in the control group and eight horses in the treatment group. The defect is only induced in one side to eliminate systemic effects, even with intraarticular therapy.

Information from this model has been used to investigate three commonly used intra-articular corticosteroids as well as an intravenous hyaluronan product. Most recently we have used the model to validate a gene therapy technique using the equine IL-1 RA with an adenoviral vector. At the end of an eight week period, horses were euthanized and at gross examination had focal articular cartilage erosions. There were histologic changes consistent with OA and depletion of safranin-0 staining in the matrix. The gene therapy protocol eliminated gross erosions in the articular cartilage, restored safranin-0 staining and increased glycosaminoglycan synthesis as well as decreased severity of the histologic findings.

Equine Articular Cartilage Defect Model To Evaluate Cartilage Healing

An equine full thickness articular cartilage defect model has been used to evaluate potential therapies for articular cartilage resurfacing in the human knee. A 1 cm square defect was made in the central weightbearing portion of the medial condyle of the femur in the horse using arthroscopic technique. The principal challenge to creation of the model has been ensuring that all calcified cartilage is removed while the subchondral bone plate is retained. With sufficient cadaver work it has been established that the depth of the articular cartilage is consistently 2 mm $\pm$ 0.1 mm. With the use of careful arthroscopic examination and a depth gauge, calcified cartilage is consistently removed. Experimentation has shown marked difference in the normal healing of these defects, depending on whether the calcified cartilage is removed.

The model has been used to scientifically evaluate the effect of subchondral microfracture as well as newer tissue engineering techniques. In a long term study with subchondral microfracture, a significantly increased amount of repair tissue in the microfracture defects as well as significantly enhanced type II collagen content were demonstrated. In a second short term study, gene expression for type I and type II collagen as well as aggrecan were evaluated in early healing. This experiment demonstrated a gradual and significant increase in messenger RNA content (gene expression) for both type II collagen and aggrecan over an eight week period and also showed that at eight weeks the expression for type II collagen was significantly enhanced by subchondral microfracture.

Benefits that we consider to exist in this model include articular cartilage thickness close to human, an ability to create defects and treat them arthroscopically, an ability to have controlled exercise after surgery (however nonweightbearing cannot be done), as well as obviously realistic forces to the repair site. The technique of microfracture is now used routinely in clinical cases of chondral defects, especially in racehorses. Clinical outcomes have been encouraging.

Facilitation of cartilage repair falls into one of two categories: 1) repair by stimulation of pluripotential cells from the subarticular level (e.g. microfracture), or 2) transplantation of cartilage osteochondral grafts or chondrocytes from remote regions. Our research group has focused on manipulation of endogenous healing, and the next study will involve gene therapy with growth factors as a test for augmentation of healing a defect within the joint.

Anterior Cruciate Ligament

Rupture of the anterior cruciate ligament (ACL) is one of the most common knee injuries seen in dogs. While the techniques for surgical replacement used in animals have usually been adapted from those popularized in human clinical patients, several biological concepts, such as ACL repair in adolescents, the mechanisms of ACL graft remodeling, and the effect of exercise on ligaments and tendons have been investigated in animals. This presentation will examine how clinical knee problems in animals have been used to investigate, develop, and define some of the therapeutic principles used in the treatment of ACL injuries in humans.

ACL Healing Response Technique

The "healing response" procedure, a direct extension of the microfracture technique used to treat chondral defects, may be useful as an alternative to knee ligament reconstruction. Microfracture holes are made into the cortical bone at the origin of the disrupted ligament and into the ligament itself. The surgically-induced marrow clot captures the ends of the ligament and provides an enriched environment for tissue regeneration. The ligament ends reunite without other fixation. We evaluated this technique in a canine model to assess temporal morphologic and growth factor changes. Eighteen dogs underwent bilateral subtotal laceration of the posterior cruciate ligament at the origin, followed by this microfracture treatment. Distal femurs, surgically-induced clots and ligaments were harvested at 1, 4, or 7 days. Three dogs/6 joints were analyzed morphologically, and 3 dogs/6 joints were analyzed biochemically. Ten additional dogs underwent a similar procedure, but one knee received treatment, and the contralateral knee served as an untreated control. Joints were examined morphologically at 12 weeks. We observed progressive organization of the marrow clots with maturation and remodeling. Healing tissue had dense collagen bundles originating from microfracture holes which joined native ligament bundles. Untreated control tissue was disorganized fibrous scar. Transforming growth factor β1 (TGF β1) concentration (per weight of tissue) decreased significantly while insulin-like growth factor-1 (IGF-1) concentration increased significantly from Day 1 to Day 7. Reciprocal TGF β1 and IGF-1 changes probably contribute to cell recruitment and differentiation for ligament repair, and these temporal changes correlate directly with observed cellular responses and progressive tissue development. The gross and histologic changes observed in this study are very similar to those described for healing vascularized soft tissues. Thus, we conclude that the surgically-induced marrow clot resulting from the microfracture ("healing response") procedure provides an enriched intraarticular milieu that appears conducive to ligament healing that is similar to normal tissue repair in the extraarticular environment.

Implications in the Rehabilitation of ACL Reconstructions

(Information in this section courtesy of and provided by Dr. Steven P. Arnoczky)

Numerous experimental studies have examined the biological and biomechanical maturation of grafts used for ACL reconstructions. While most have demonstrated a progressive transition of the autologous patellar tendon into a functional "ligament-like" tissue by one year, this remodeling was also associated with a precipitous loss of mechanical strength in weeks following transplantation. This alteration in material properties was thought to be a natural sequelae of the remodeling process and most rehabilitation algorithms were designed to "protect" the graft during this early remodeling phase. However, in recent years the advent of the so-called "accelerated" rehabilitation programs have challenged that concept.

To examine the role of physical stress in the normal physiology of connective tissues we examined the mechanical response of connective tissues to various paradigms of tensile load in an *in vitro* model. We demonstrated that as little as two weeks of stress deprivation results in a 20% decrease in the tensile modulus of tendons. This progresses to almost 50% by six weeks and is accompanied by a marked decrease in cell number. These *in vitro* results are similar to *in vivo* observations over the same time frame. Using a custom designed loading apparatus we significantly ($p<0.05$) reduced the loss in tensile modulus over a 4 week period by applying low level cyclic strain (0.5% strain, @ 1 cycle/min) for 2 hours daily.

We hypothesized that the stress deprivation caused an up-regulation of collagenase activity in the tendon cells and that tensile loading (and the associated mechanotransduction of cell signals) has a stress-suppressible effect on collagenase expression. We used a monoclonal antibody to collagenase to demonstrate increased collagenase expression in tendon cells under stress deprived conditions. Conversely, cyclic tensile loading (in the algorithm described above) suppressed this expression. Thus, lack of tensile loading initiates a catabolic process in the tendon that can markedly alter the mechanical properties of this tissue. These results would suggest that autogenous patellar tendon grafts, since they are transplanted with viable cells, require immediate cyclic tensile load to minimize the catabolic effect associated with stress deprivation. While this finding has significant implications in the early rehabilitation of ACL reconstructions, the magnitude, frequency, and mode of application of these stresses have yet to be determined.

Meniscus

The canine meniscus has many similarities to the human meniscus. Dogs often tear their menisci during athletic activities, usually requiring surgical removal. As in humans, in the dog loss of the meniscus cartilage of the knee increases concentration of stresses and minimizes contact areas on articular surfaces of both the femur and tibia, often leading to osteoarthritis and irreversible joint damage.

Development of the Collagen Meniscus Implant

We used tissue engineering techniques to develop a resorbable collagen scaffold (the Collagen Meniscus Implant) that supports ingrowth of new tissue and eventual regeneration of the lost meniscus cartilage. In numerous laboratory and animal studies, we demonstrated that this implant supports ingrowth and maturation of meniscus fibrochondrocytes and development of mature and functional fibrocartilaginous matrix. We carried out most of the animal studies in dogs using either a subtotal meniscectomy model or a die punch model. In general, we observed significant tissue regeneration grossly in most joints. Histologic examination of the collagen meniscus implant regenerated tissue revealed fibrocartilage with dense well-organized collagen bundles similar to a normal meniscus. These and other findings supported use of this device in human clinical trials. In those trials, all patients improved clinically from preoperatively to 1 and 2 years postoperatively based on pain, Lysholm scores, Tegner activity scale, and self assessment. Relook arthroscopy revealed tissue regeneration with apparent preservation of the joint surfaces. Histologic analysis confirmed new fibrocartilage matrix formation. Further follow-up evaluation of these patients at an average 5.8 years after implant placement has confirmed the durability of the new tissue as well as its chondroprotective properties. The feasibility trials led us into multicenter clinical trials in the United States, Europe and Japan. Results of the clinical trials have been encouraging. Enrollment in the US multicenter trial is now complete, and the additional follow up required by the FDA is ongoing. Approval of the collagen meniscus implant for general use has been obtained in Europe, Australia, and Chile. Hence, we conclude that the collagen meniscus implant can be implanted arthroscopically, is biocompatible, is resorbable, and supports new tissue regeneration as it is resorbed. Importantly, this newly regenerated tissue seems to function similar to meniscus tissue by protecting the chondral surfaces.

Symposium: Patellar Tendinopathy Advances and Innovations
Thursday, March 13, 2003 • Aotea Centre, Kupe/Hauraki Room

Chairman: Annunziato Amendola, MD, USA
Faculty: Bent Wulff Jakobsen, MD, Denmark, Niek van Dijk, PhD, Netherlands and Hakan Alfredson, MD, PhD, Sweden

NOTES

Symposium: Unicompartmental Knee Replacement (Minimally Invasive)
Thursday, March 13, 2003 • Aotea Centre, Kupe/Hauraki Room

Chairman: David Murray, FRCS(Orth), United Kingdom
Faculty: Kenneth DeHaven, MD, USA, Greg Keene, MBBS, FRACS, Australia and Chris Dodd, United Kingdom

The speakers will present an overview of their experience of Minimally Invasive Unicompartmental Knee Replacement, There will then be a detailed discussion of four topics. Areas of agreement and disagreement will be highlighted:

A) Design philosophy (eg., pre-knee replacement, fixed bearing, mobile bearing, etc.)
B) Surgical principles (eg., burr, saw, mill, etc.)
C) Indications
D) Results based on these indications. This should include both short term functional results and, if available, long term results.

NOTES

Symposium: Total Knee Arthroplasty: Controversies in Revision
Thursday, March 13, 2003 • Aotea Centre, Kupe/Hauraki Room

Chairman: James Rand, MD, USA
Faculty: John Hart, MBBS, FRACS, Australia, Paolo Aglietti, MD, Italy
and Robert Trousdale, MD, USA

Metal Augment or Bone Graft for Management of Bone Defects at Revision Total Knee?
J.A. Rand ISAKOS 2003

I. Bone Loss in TKA
- A. Etiology
  1. Implant loosening
  2. Sepsis
  3. Osteolysis
- B. Classification (Engh)

Type	Metaphyseal Bone
I	Intact
II	Damaged
II	Deficient

- C. Management
  1. Type I Bone Loss
     a. Cement
     b. Particulate bone graft
  2. Type II Bone Loss
     a. Cement
     b. Modular augment
     c. Particulate bone graft
     d. Structural graft
     e. Extended stems
  3. Type III Bone Loss
     a. Tumor type TKA
     b. Structural graft with standard revision TKA
     c. Allograft prosthetic composite
- D. Location of Bone Loss
  1. Femur
     a. Distal
     b. Posterior

c. Combined
d. RESTORE JOINT LINE and balance flexion/extension spaces
2. Tibia
a. Central
b. Peripheral
c. Fill defect and provide support for implant on host bone when possible

II. Management of Bone Loss in TKA
A. Structural Bone Graft
1. Indications
a. Femoral head- single condyle
b. Proximal tibia or distal femur allograft for global metaphyseal defect
c. Particulate graft for contained defect
2. Bone Graft Technique
a. Rigid fixation
b. Avoid cement interposition
c. Stemmed implant
d. Cover graft with prosthesis
3. Histology of Structural Bone Graft (Engh)
a. Knees 7
b. Autopsy retrieval
c. F/U 20-60 mo.
d. Not remodeled
e. All united
f. No revascularization
g. No collapse

B. Particulate bone graft
1. Rationale
a. Easily performed
b. Rapid incorporation
c. Remodeled
d. Restores bone
2.Disadvantages
a. Does not provide structural support
b. Lack of instrumentation
3. Technique
a. Morselized cancellous bone

b. Vascular host bed
c. Tight packing of graft
d. Cement over graft
e. Stability of implant <u>not</u> dependent upon graft (i.e. long stem)

4. Biomechanics of Morselized Grafts (van Loon)
   a. Distal femora 10
   b. Uncontained unicondylar defect with primary TKA
   c. Cyclic load 750 N
   d. No failures

5. Biomechanics of Morselized Grafts (van Loon)
   a. In vivo equine study
   b. Uncontained unicondylar defect with primary TKA
   c. Cyclic load 750 N
   d. No differences from controls

6. Histology of Particulate Grafts (Whiteside)
   a. Biopsy 14 grafts
   b. F/U 1-37 months
   c. New bone formation
   d. Remodeling

7. Results

a. Cementless Reconstruction for Bone Loss in Revision TKA (Whiteside)
   1. Knees 63
   2. Morselized allograft
   3. Subsequent loosening 2

b. Revision TKA by Impaction Grafting (Bradley)
   1. Knees 19
   2. F/U 33 months
   3. Morselized graft
   4. Structural graft in 3
   5. Failure 1

c. Morselized Bone Grafting In Revision TKA (Benjamin)
   1. Knee 33
   2. F/U 2 years

3. No failures
4. Lucent lines 35%

d. Instrumented Impaction Grafting in Revision TKA(Heyligers)

1. Knees 11
2. F/U 4 years
3. Primary implants
4. No failures

C. Modular Bone Augments

1. Advantages
    a. Simple
    b. Rapid
    c. No healing
2. Disadvantages
    a. No restoration of bone
    b. Fretting
    c. Corrosion
    d. Disassembly
3. Indications
    a. Femur
        1. Joint line restoration
        2. Flexion/extension space balancing
    b. Tibia
        1. Single condyle defect
    c. Types
        1. Blocks
            a. Rectangular defect
            b. Angled defect >20 degrees
            c. Minimizes shear stress
        2. Wedges
            a. Angled defects < 20 degrees

III. Conclusions

A. Modular augments for small defects, soft tissue balancing, and joint line restoration
B. Allografts for extensive bone loss
C. Tumor hinge for massive bone loss and low demand patient
D. Long stems for fixation in intact host bone

KUPE/HAURAKI

References

1. Benjamin, J.; Engh, G.; Parsley, B; Donaldson, T.; Coon, T. Morselized Bone Grafting of Defects in Revision Total Knee Arthroplasty Clin. Orthop. 392:62-67, 2001.
2. Bradley, G.W. Revision Total Knee Arthoplasty by Impaction Bone Grafting Clin. Orthop. 371:113-118, 2000.
3. Heyligers, I.C.;van Haaren, E.H.; and Wuisman, P.I.J.M. Revision Knee Arthroplasty Using Impaction Grafting and Primary Implants. J. Arthroplasty 16:533-537, 2001.
4. Van Loon, C.J.M.; de Waal Malefijt, M.C.; Verdonschot, N.; Burma, P.;van der Aa, A.J.A.M.; and Huiskes, R. Morselized Bone Grafting Compensates for Femoral Bone Loss in Revision Total Knee Arthroplasty. Biomaterials 20:85-89, 1999.
5. Van Loon, C.J.M.; de Waal Malefijt, M.C.; Burma, P.; Stolk, P.W.Th.; Verdonschot, N.; Tromp, A.M.; Huiskes, R.; and Barneveld, A. Morselized Bone Grafting Restores Uncontained Femoral Bone Defects in Knee Arthroplasty J Bone Joint Surg. 82B:435-444, 2000.
6. Whiteside, L.A. Cementless Reconstruction of Massive Tibial Bone Loss in Revision Total Knee Arthroplasty. Clin. Orthop. 248:80-86, 1989.
7. Whiteside, L.A.; and Bicalho, P.S. Radiologic and Histologic Analysis of Morselized Allograft in Revision Total Knee Replacement. Clin. Orthop. 357:149-156, 1998.
8. Lonner, J.H.; Lotke, P.A.; Kim, J.; and Nelson, C. Impaction Grafting and Wire Mesh for Uncontained Defects in Revision Knee Arthroplasty. Clin. Orthop. 404:145-151, 2002.
9. Gofton W.T.; Tsigaras, H.; Butler, R.A.; Patterson, J.J.; Barrack, R.L.; and Rorabeck, C.H. Revision Total Knee Arthroplasty. Clin. Orthop. 357;158-168, 2002.
10. Laskin, R.S. Joint Line Position Restoration During Revision Total Knee Replacement. Clin. Orthop. 404:169-171, 2002.
11. Nazarian, D.G.; Mehta, S.; and Booth, R.E. A Comparison of Stemmed and Unstemmed Components in Revision Knee Arthroplasty. Clin. Orthop. 357:256-262, 2002.
12. Bourne, R.; Finlay, J. The Influence of Tibial Component Intramedullary Stems and Implant – Cortex Contact on the Strain Distribution of the Proximal Tibia Following Total Knee Arthroplasty. Clin. Orthop. 208:95-99, 1986.
13. Brand, M.; Daley, R.; Frederick, C.; Ewald, F.; Scott, R. Tibial Tray Augmentation With Modular Metal Wedges for Tibial Bone Stock Deficiency. Clin. Orthop. 248:71-79, 1989.
14. Brooks, P.J.; Walker, P.S.; and Scott, R.D. Tibial Component Fixation in Deficient Tibial Bone Stock. Clin. Orthop. 184:302-308, 1984.
15. Chen, F.; and Krackow, K.A. Management of Tibial Defects in Total Knee Arthoscopy. Clin. Orthop. 305:249-257, 1994.

16. Dorr, L.; Ranawat, C.; Sculco, T.; McKaskill, B.; and Orisek, B. Bone Graft for Tibial Defects in Total Knee Arthoplasty. Clin. Orthop. 205:153-165, 1986.
17. Engh, G.; Herzwurm, P.; and Parks, N. Treatment of Major Defects of Bone with Bulk Allografts and Stemmed Components during Total Knee Arthoplasty. J Bone Joint Surg. 79A(7):1030-1039, 1997.
18. Fehring, T.K.; Peindl, R.D.; Humble, R.S.; Harrow, M.E.; and Frick, S.L. Modular Tibial Augmentations in Total Knee Arthroplasty. Clin. Orthop. 327:207-217, 1996.
19. Garg, A.; and Walker, P. The Effects of the Interface on the Bone Stresses Beneath Tibial Components. J. Biomechanics. 19(12):957-967, 1986.
20. Ghazavi, M.T.; Stockley, I.; Yee, G.; Davis, A.; and Gross, A.E. Reconstruction of Massive Bone Defects with Allograft in Revision Total Knee Arthroplasty. J Bone Joint Surg. 79A(1):17-25, 1997.
21. Haas, S.; Insall, J.; Montgomery, W.; and Windsor, R. Revision Total Knee Arthoplasty with Use of Modular Components with Stems Inserted Without Cement. J Bone Joint Surg. 77A(11):1700-1707, 1995.
22. Harris, A.I.; Poddar, S.; Gitelis, S.; Sheinkop, M.D.; and Rosenberg, A.G. Arthroplasty with a Composite of an Allograft and a Prosthesis for Knees with Severe Deficiency of Bone. J Bone Joint Surg. 77A(3):373-386, 1995.
23. Hartford, J.; Goodman, S.; Schurman, D.; and Knoblick, G. Complex Primary and Revision Total Knee Arthroplasty Using the Condylar Constrained Prosthesis. J. Arthroplasty. 13(4):380-387, 1998.
24. Kim, Y.; Oh, J.; and Oh, S. Osteolysis Around Cementless Porous-Coated Anatomic Knee Prostheses. J Bone Joint Surg. 77B:236-241, 1996.
25. Laskin, R. Total Knee Arthoplasty in the Presence of Large BonyDefects of the Tibia and Marked Knee Instability. Clin. Orthop. 248:66-70, 1989.
26. Lotke, P.; Wong, R.; and Ecker, M. The Use of Methylmethacrylate in Primary Total Knee Replacements with Large Tibial Defects. Clin. Orthop. 270:288-294, 1991.
27. Lindstrand, A.; Hansson, U.; Toksvig-Larsen, S.; and Ryd, L. Major Bone Transplantation in Total Knee Arthoplasty: A 2- to 9-Year Radiostereometric Analysis of Tibial Implant Stability. J. Arthroplasty. 14(2):144-148, 1999.
28. Martin, S.; Haas, S.; and Insall J. Primary Total Knee Arthroplasty after Patellectomy. J Bone Joint Surg. 77A:1323-1327, 1995.
29. Mnaymneh, W.; Emerson, R.H.; Borja, F.; Head, W.C.; and Malinin T.I. Massive Allografts in Salvage Revisions of Failed Total Knee Arthroplasties. Clin. Orthop. 260:144-153, 1990.
30. Mow, C.S.; and Wiedel, J.D. Structural Allografting in Revision Total Knee Arthroplasty. J. Arthroplasty. 11(3):235-241, 1996.
31. Murray, P; Rand, J.; and Hanssen, A. Cemented Long-Stem Revision Total Knee Arthroplasty. Clin. Orthop. 309:116-123, 1994.
32. Pagnano, M.; Trousdale, R.; and Rand, J. Tibial Wedge Augmentation for Bone Density in Total Knee Arthroplasty. Clin. Orthop. 321:151-155, 1995.

33. Parks, N.; and Engh, G. Histology of Nine Structural Bone Grafts Used in Total Knee Arthroplasty. Clin. Orthop. 345:17-23, 1997.
34. Parthington, P.; Sawhney, J.; Rorabeck, C.; Barrack, R.; and Moore, J. Joint Line Restoration After Revision Total Knee Arthroplasty: Clinical Significance and Accuracy presented at The Knee Society, February 7, 1999, Anaheim, California.
35. Peters, C.; Hennessey, R.; Barden, R.; Galante, J.; and Rosenberg, A. Revision Total Knee Arthroplasty With a Cemented Posterior-Stabilized or Constrained Condylar Prosthesis. J. Arthroplasty. 12(8):896-903, 1997.
36. Peters, P.; Engh, G.; Dwyer, K.; and Vinh, T. Osteolysis after Total Knee Arthroplasty Without Cement. J Bone Joint Surg. 74A:864-876, 1991.
37. Rand, J.; and Franco, M. Revision Considerations for Fracture About the Knee in Controversies of Total Knee Arthroplasty, ed. Goldberg V. Raven Press, P. 2335-248, 1991.
38. Springer, B.D.; Hanssen, A.D.; Sim, F.H.; and Lewallen, D.G. The Kinematic Rotating Hinge Prosthesis for Complex Knee Arthroplasty. Clin. Orthop. 392:283-291, 2001.
39. Rand, J; and Stauffer, R. Kinematic Rotating Hinge Total Knee Arthoplasty. J Bone Joint Surg. 69A:489-497, 1987.
40. Rand, J. Augmentation of Total Knee Arthroplasty with Modular Metal Wedge. J Bone Joint Surg. 77A:266-268, 1995.
41. Rand, J. Bone Deficiency in Total Knee Arthoplasty: Use of Metal Wedge Augmentation. Clin. Orthop. 271:63-71, 1991.
42. Rand, J. Modularity in Total Knee Arthroplasty. Acta Orthop Belgica. 62(1), 1996.
43. Rand, J.A. Planning for Revision Total Knee Arthroplasty. Instructional Course Lectures. 48:161-166, 1999.
44. Rand, J. Preoperative Planning and Templating in Total Knee Arthroplasty, ed. Rand, J. Raven Press, New York, p. 91-114, 1993.
45. Ritter, M.; Keating, M.; and Faris, P. Screw and Cement Fixation of Large Defects in Total Knee Arthroplasty. J Arthrop. 8(1):63-65, 1993.
46. Ritter, M; Montgomery, T.; Zhou, H.; Keating, M.; Faris, P.; and Meding, J. The Clinical Significance of Proximal Tibial Resection Level in Total Knee Arthroplasty. Clin. Orthop. 360:174-181, 1999.
47. Stockley, I.; McAuley, J.P.; and Gross, A.E. Allograft Reconstruction in Total Knee Arthoplasty. J Bone Joint Surg. 74B(3):393-397, 1992.
48. Tsahakis, P.J.; Beaver, W.B.; and Brick, G.W. Technique and Results of Allograft Reconstruction Revision Total Knee Arthroplasty. Clin. Orthop. 303:86-94, 1994.
49. Takahashi, Y.; and Gustilo, R. Nonconstrained Implants in Revision Total Knee Arthroplasty. Clin. Orthop. 309:156-162, 1994.
50. Vince, K.; and Long, W. Revision Knee Arthroplasty: The Limited Press Fit Medullary Fixation. Clin. Orthop. 317:172-177, 1995.
51. Whtieside, L.; and Bicalho, P. Radiologic and Histologic Analysis of Morselized Allograft in Revision Total Knee Replacement. Clin. Orthop. 357:149-156, 1998.

52. Whiteside, L.A. Cementless Reconstruction of Massive Tibial Bone Loss in Revision Total Knee Arthoplasty. Clin. Orthop. 248:80-86, 1989.
53. Wilde, A.H.; Schickendantz, M.S.; Stulberg, B.N.; and Go, R.T. The Incorporation of Tibial Allografts in Total Knee Arthroplasty. J Bone Joing Surg. 72A(6):815-824, 1990.

THE PATELLA IN REVISION TOTAL KNEE ARTHOPLASTY(TKA)

John A. L Hart
Monash University, The Alfred
Melbourne , Australia
ISAKOS, Auckland, N.Z. March, 2003

The patellofemoral joint (PFJ) is the most common cause of reoperation after TKA.[1,2.] Patellar revision may be indicated as an isolated procedure or as part of a global revision. Resurfacing may be required for anterior knee pain (AKP) following non resurfacing at a primary TKA. Wear and loosening are responsible for 75% of reoperations[1]

	INDICATIONS YES + NO -	
PROBLEM	**ISOLATED**	**GLOBAL**
Infection	**+**	**+**
Loosening	**+**	**+/-**
Wear metal backed	**+[3,4,5,6]**	**+/-**
allpoly	**+**	**-**
Osteolysis	**+**	**+/-**
Implant fracture	**+**	**-**
P/E metal disassociation	**+[7]**	**+(if femur damaged)**
Instability/ maltracking acute	**- (dehissence)**	**-**
chronic	**+/- (with realignment)**	**+ (if malpositioned)**
Malposition	**+**	**-**
Patella baja	**-**	**+ (if joint line proximal)[8]**
Avascular necrosis	**+**	**-**
Patellar fracture minor	**-**	**-**
major	**+[9]**	**+ malalignment**
Overstuffing	**-**	**+ downsize femur**
Non resurfacing	**+**	**-**

The principles of management are to correct the underlying cause and to revise the patella.

	PRINCIPLES		
	PATELLA		KNEE
PROBLEM	REVISION	CORR.	
Infection	Remove	-	Remove. Antibiotics.
Loosening	Remove/ Exchange	+/-	+/- correct malalignment
Wear metal backed	Remove/ Exchange	+/-	+/- remove if damaged
		+/-	+/- correct malalignment
allpoly	Remove/ Exchange	+/-	+/- correct malalignment
Osteolysis	Remove/ Exchange	+/-	+/- correct malalignment
Implant fracture	Remove/ Exchange	+/-	-
P/E metal disassociation	Remove/ Exchange	-	+/- remove if damaged
Instability/ maltracking acute	+/-	+	-
chronic	+/-	+	+ if malpositioned
Malposition	+	-	-
Patella baja	-	+	+ if joint line proximal
Avascular necrosis	Remove	-	-
Patellar fracture minor	-	-	-
major	-/ remove	+/-	+ if malaligned
Overstuffing	-	-	+ downsize femur
Nonresurfacing	+	-	-

Specific Problems

Exposure

- **Adequate medial release[8]**
- **Pin patellar tendon**
- **Resect fat pad**
- **Divide patellofemoral ligament**
- **Subcutaneous pouch for obese[10]**
- **Rectus snip[11]**
- **V-Y turndown[12]**
- **Tibial osteotomy[10]**
-

Patellar management

Major problems are bone loss and high complication rate[1,2]

Berry and Rand[1]: isolated resurfacing, HSS 71-81; 34% complications

Options:

Better results with replacement implant than removal and bone shell[13]

However depends on bone stock

>11 mm. cortical ring intact: resurface bone graft and onlay prosthesis

biconvex disc(BCD) (varying thickness)

superior results with BCD[8]

<11mm. cortical ring deficient: salvage bone shell, KSS 46-69, AKP 29%[14]

gull wing osteotomy

button replacement; poor results[8]

patellectomy variable results

satisfactory[15,17] small series

unsatisfactory[1,13]

bone graft in soft tissue pouch[17]

KSS improved, patellar bone stock increased

Realignment

Lateral release

Medial plication

Distal transfer

Patellar Fracture

Modified Goldberg classification[8]

Undisplaced ,intact extensors Types 1,11B, 111B: conservative

Displaced, prothesis disturbed, deficient extensors Types 11A, 111A: simple repair/patellectomy

Fracture dislocations Type 1V: patellectomy

References

1. Berry,D and Rand, J: Clin Orthop 1993;286:110
2. Brick ,G and Scott,R: Clin Orthop 1988;231:163
3. Bayley, J et al : J Bone Joint Surg 1988;70A:668
4. Stulberg, S et al Clin Orthop 1988;236:88
5. Rosenberg, A et al Clin Orthop 1988;236:106
6. Dennis, D: J Arthroplasty 1992;7:217
7. Lombardi, D et al: J Bone Joint Surg 1988;70A:675
8. Laskin, R: Orth Clin North Amer 1998;29:355
9. Goldberg, V et al: Clin Orthop 1988;236:115
10. Whiteside, L: Clin Orthop 1997;321;32
11. Insall, J: in Insall J et al: Surgery of the Knee. New York, Churchill Livingstone, 1993
12. Trousdale,R et al: Clin Orthop 1993;286:48
13. Barrack, R et al: Clin Orthop 1998;356:139
14. Pagnano, M et al: Clin Orthop 1988;356:134
15. Drakeford, M et al: Orthop Trans 1993;17:992
16. Seel M et al: Orthop Trans 1995;19:338
17. Hanssen,A: J Bone Joint Surg 2001;83A:171

REOPERATIONS AFTER 3200 REVISION TOTAL KNEE REPLACEMENTS: RATE, ETIOLOGY AND LESSONS LEARNED

Robert T. Trousdale, M.D.; Rafael J. Sierra, M.D.; Mark W. Pagnano, M.D.

Introduction: The mechanism by which primary total knee arthroplasties fail are fairly well established. The purpose of this study was to evaluate the rate, etiology, and evolution over time of reoperations done after revision total knee replacement (RTKA).

Methods: From 1970-2000, 3251 total knee revisions were done at our institution. Those included 574 RTKA done in the 1970s, 1010 done in the 1980s and 1667 done in the 1990s. 695 knees (26%) subsequently have been re-operated on 1 or more times. Age at RTKA averaged 62 years ± 12.5. 365 of the knees were in males and 330 in females. Left knee reoperations outnumbered the right knee by a 5:3 margin.

Results: Follow-up averaged 15 years ± 8.2. The average time from RTKA to the first reoperation was 3.8 years. The total number of reoperations was 1051. 107 knees were reoperated on twice, 55 knees 3 times, 25 knees 4 times, 19 knees 5 times, 7 knees 6 times, 5 knees 7 times and 1 knee 8 times. Reoperations included revision of components in 258 for septic loosening, debridement for infection in 128, and resection of components without reinsertion in 120 knees. From the 1970s to the 1990s, removal of all components after RTKA decreased by 400% while joint debridements increased 250%.

Discussion: The rate of reoperation in this large series of RTKA was surprisingly high. Despite substantial progress over the past three decades in component design, surgical technique and the prevention of infection, patients who require revision TKA are at substantial risk of developing one or more subsequent problems that require a reoperation.

Symposium: Arthroscopic Treatment of Shoulder Stiffness
Friday, March 14, 2003 • Aotea Centre, Kupe/Hauraki Room

Chairman : Philippe Beaufils, MD, France
Faculty: Daniel Mole, MD, France, Stephen Burkhart, MD, USA, Gregory Bain, Australia

Introduction

Ph Beaufils

Versailles France

pbeufils@ch-versailles.fr

Shoulder stiffness is defined as a restriction of the passive range of motion of the shoulder. This excludes shoulder motion limitation due to pain. Shoulder stiffness can be caused by glenohumeral, subacromial, and/or periarticular abnormalities. Terms used today or in the past to designate the stiff shoulder include capsular retraction (Duplay), frozen shoulder (Codmann , De Palma , Ozaki , Zulkermann), adhesive capsulitis (Neviaser, Murnogan, Hanaffin), and shoulder stiffness. The considerable heterogeneity in the conditions designated by these terms makes it difficult to interpret the literature on the diagnosis and management of shoulder stiffness. The cause of the loss of motion, which is sometimes difficult to identify, can be contraction of the capsule, capsular adhesions or coalescence (Neviaser), capsular scars produced after an injury or surgical procedure,

Shoulder stiffness can be due either to extracapsular lesions (bursa, long head of the biceps) or to capsular abnormalities, including capsular contraction and scar tissue formation. Capsular contraction (as opposed to adhesions) occurring alone is called primary frozen capsulitis, whereas capsular contraction with subacromial impingement is called bipolar shoulder stiffness. Scar tissue formation characterizes shoulder stiffness secondary to an injury or surgical procedure.

This confusion has resulted in poor standardization of the therapeutic strategy for stiff shoulder. In addition to conservative treatments, joint distention (Ekelund, Hsu , mobilization with or without arthroscopy (Hsu, Pollock, Uitvlugt), and surgical release have been proposed, with differences in indications according to the cause and to the time since the first treatment. More recently arthroscopic release has been proposed with promising results in selective indications .

Arthroscopic Treatment of Shoulder Stiffness: Anatomical Considerations

Gregory Bain
North Adelaide
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The axillary nerve is a terminal branch of the posterior cord of the brachial plexus, which passes inferior to the lower border of the subscapularis muscle. At that point it passes within some loose areolar tissue adjacent to the capsule before passing posterior to exit through the quadrilateral space. The axillary nerve is particularly at risk during arthroscopic procedures such as a capsulotomy and radio-frequency. [1, 2]

We performed a cadaveric study to assess the proximity of the axillary nerve to the inferior capsule of the glenohumeral joint in a cadaveric model. [3]

Via a delto-pectoral approach the axillary nerve was identified and needles were placed through the nerve and through the adjacent capsule into the glenohumeral joint.

From an arthroscopic perspective it was possible to determine that the nerve was at risk between the 5 and 7 o'clock position. The nerve ran from anterior medial through to the posterior aspect of the capsule before passing between the teres minor and teres major muscle.

Various positions of the shoulder do change the relationship between the axillary nerve and the capsule.

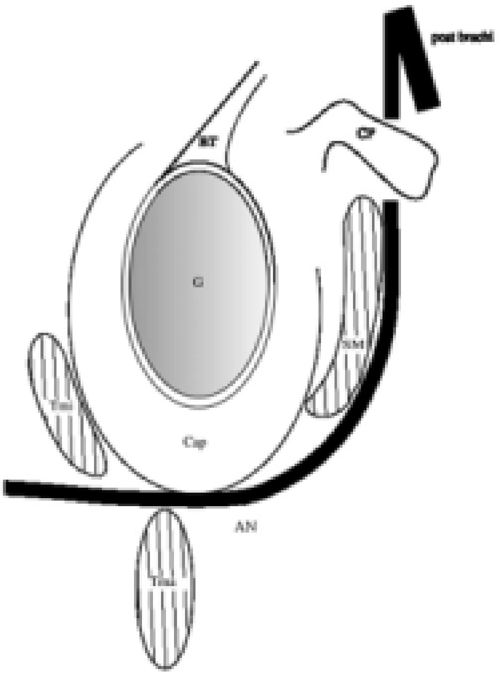
Patients with tears of the posterior superior labrum can develop a sizeable ganglion, which can cause encroachment upon the suprascapular nerve. These ganglion are likely to be similar to the lateral meniscal cysts which occur with a flap valve tear of the lateral meniscus causing fluid to be pumped into the cysts. Arthroscopic debridement of the tear and having it open disarms the pump. Therefore the difficult open exposure of the suprascapular nerve and the cysts are avoided.

References

1. Duval MJ, Parker AW, Drez D, Jr., Hinton MA. The anterior humeral circumflex vessels and the axillary nerve. An anatomic study. Orthop Rev, 1993: 22(9): 1023-1026.

2. Segmuller HE, Taylar DE, Hogan CS, Saies AD, Hayes MG. Arthroscopic treatment of adhesive capsulitis. Journal of Shoulder and Elbow Surgery, 1995: 4403-408.

3. Uno A, Bain GI, Mehta JA. Arthroscopic relationship of the axillary nerve to the shoulder joint capsule: an anatomic study. Journal of Shoulder and Elbow Surgery, 1999: 8(3): 226-230.



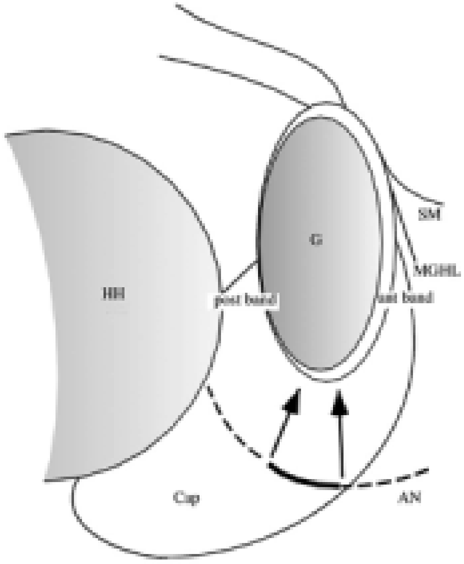


fig 2

fig 1

Arthroscopic Release of Shoulder Stiffness

Technique

Ph Beaufils
Versailles - France
pbeaufils@ch-versailles.fr

Which Structure is Contracted ? : Cadaveric Study

1- sequential capsular shrinkage (bipolar radio frequency) followed by sequential capsular release of 12 fresh cadaveric shoulders (Ph Boisrenoult, P Gaudin, Ph Beaufils)
2- Coraco-humeral ligament and SGHL, then MGHL, then IGHL, then section of the subscapularis tendon
3- Coraco-Hum ligament + SGHL
    a. Shrinkage : loss of 17° external rotation
    b. Release : gain of 40° external rotation

4- IGHL
    a. Shrinkage : loss of elevation 20°
    b. Release : gain of elevation 33°

5- Synergy between release of superior and inferior structures of the capsule in terms of external rotation and elevation
6- Subscapularis section : gain of ER = 14°. One subsequent anterior dislocation

These study suggests that the key points for an effective release are the coraco-humeral ligament and the IGHL. It is not necessary to cut the subscap tendon (risk of dislocation). The posterior capsule has not been assessed in this study.

Technique

1- Position : beach chair position or decubitus lateral can be used. Whith the beach chair position, the mobilization of the shoulder is easier
2- Anaesthesia :General anesthesia can be used (more comfortable for the patient and the surgeon), but it is essential to combine a scalene block with catheter which allows a good control of the post op pain.
3- Assessment of ROM
4- The arthroscope is inserted via the posterior standard portal. Despite the motion restriction, and the tight joint, this introduction is always possible.
5- Exploration. Hyperemia, fibrosis with white thick capsule evoke a primary frozen shoulder. Bursoscopy shows a normal appearance in these cases +++. Synovitis and fibrosis of the glenohumeral joint, with abnormalities of the bursa (impingement, rotator cuff tear…) evoke a bipolar stiffness. Shoulders in the post-injury/surgery group demonstrate scar tissue with or without adhesions
6- Capsular Release : is carried out by anterior standard portals, using mechanical instruments or laser, or bipolar radiofrequency. It is essential to release the rotator interval (coraco-humeral ligament), by cutting the contracted capsule at the front of the long head of the biceps. Section of the anterior and inferior capsule is done from above to below at 5 mm from the glenoid rim. It should include MGHL and the two bundles (ant and posterior) of the IGHL. It is not necessary to cut the tendon of the subscapularis muscle. In case of restriction of the internal rotation, section of the posterior capsule can be carried out, inverting the scope and the instruments. Additional procedures are sometimes required (acromioplasty, section of the long head of the biceps, synovectomy, debridement of the subacomial bursa, …) These procedures must be avoided in case of primary frozen shoulder.

7- Gentle mobilization of the shoulder is done at the end of the arthroscopy procedure.
8- Post op analgesia is a key point, using scalenic block thrue a catheter , and morphine
9- Immediate post op continuous passive motion is required.

Adhesive Capsulitis ; Treatment and outcome
A retrospective multicentric study of 158 cases

D. Molé, G. Daubinet, M. Allard and the French Society of Arthroscopy (S.F.A.)
Nancy (France**)**
d.mole@wanadoo.fr

The treatment of idopathic adhesive capsulitis of the shoulder remains a challenge ; functional management and arthroscopic capsular release can both be proposed. This study is, to our knowledge, the first attempt to compare the results of these two procedures.

- **Patients and method**

158 patients have been reviewed, from 11 Shoulders Centers. The main criteria of inclusion was a true idiopathic adhesive capsulitis (forward passive elevation < 90°, external rotation 0°, internal rotation < 90°). The patients with an history of trauma as well as the patients with a rotator cuff lesion, were excluded. All the patients have been reviewed with an average follow-up of 4,3 years (more than two years).
There were 73 % females ; Average age was 48,7 years (27 to 68). The controlateral shoulder was involved in 26 % of the cases. 13 % of the patients related a diabetic disease. 8 % had a Dupuytren disease. In 17 % of the cases, the symptoms involved the whole upper limb with painfull stiffness of the hand

and the fingers, allowing to consider the diagnosis of sympathic neuro dystrophy (SND).

126 patients were treated conservatively; 32 had an arthroscopic release after a mean delay of symptoms of 25 months.

- **Results**

The Average duration of the disease was 24 months. The mean Constant Score at revision was 74 points. The passive range of motion was normal in only 65 % of the cases ; the shlouder was totally painfree in 49 %. The results are not worse in diabetic patients but the length of the evolution is longer (37 months). SND patients had a worse (Constant Score 71) and longer (31 months) outcome.

An arthroscopic release has been performed in 32 cases. In these patients, where functional treatment had failed, it allowed the same result than the whole series. The results of arthroscopy compared to functional treatment seem even superior in male, younger, SND patients or in patients also complaining of a Dupuytren disease. On a technical point of view, the passive range of motion obtained at the end of operation remained stable at review ; no progress can be expected from functional postoperative treatment. The release must concern the superior, anterior and inferior capsule, without any need of a subscapularis tendon release, a posterior capsular release, or a subacromial procedure. The length of evolution after arthroscopy was 7 months.

- **Discussion-Conclusion**

Adhesive capsulitis of the shoulder is far from being a self healing pathology ; half of the patients still complain of symptoms after more than two years. Treatment must be medical and functional ; arthroscopic release is indicated when conservative management fails and could be considered more often in young patients, in males, when a Dupuytren disease is associated, or in cases of true sympathic neuro dystrophy.

Post-Traumatic and Post-Surgical Stiffness of the Shoulder

Stephen S. Burkhart, M.D.
San Antonio, Texas, USA
ssburkhart@msn.com

I. Etiology

A. Capsular contracture
B. Subacromial adhesions ("captured shoulder" described by Gross et al).
C. Combination of capsular contracture and subacromial adhesions

II Examination: differentiating capsular contracture from extra-capsular etiology.

A. Capsular contracture causes loss of external rotation and forward elevation.
B. Extra-capsular (subacromial) adhesions cause loss of forward elevation with preservation of normal external rotation.

III. Arthroscopic findings

A. Capsular contracture
   1. Very thick capsule, often > 7 mm.
   2. Very tight joint space.
   3. Normal subacromial space
B. Subacromial adhesions
   1. Obliteration of subacromial space with dense adhesions
   2. Normal intra-articular findings
C. May have combined pathology with features of capsular contracture and subacromial adhesions

IV. Treatment

A. Capsular release (rotator interval, coracohumeral ligament, posterior capsule, anterior and anterior-inferior capsule).

B. Excision of subacromial adhesions

C. Injection of corticosteroid

D. Post-op stretching by P.T. (no CPM) daily for three weeks

V. Potential complications

A. Recurrent stiffness

B. Instability

C. Subscap weakness (DO NOT RELEASE SUBSCAP!)

D. Nerve injury

1. Axillary

2. Musculocutaneous

NOTES