

**Key reference database project**  
**ACC Internal Review**  
**Draft 20 March 2013**

**Causation of knee pathologies/injuries**  
**Part B**

Mechanisms of knee injury

Clinical presentation, diagnosis and imaging of some knee pathologies

Arthroscopic treatment of osteoarthritis

**Peter Larking**  
**Research**  
**ACC**

# Contents

<b>SUMMARY</b>	<b>3</b>
1. What are the typical mechanisms of injury and mechanisms of accident in: meniscal, anterior cruciate ligament, and chondral injuries/osteochondral injuries?	3
2. Are ACL disruptions always due to single episode trauma?	3
3. What is the prevalence of ACL disruption in asymptomatic individuals?	3
4. What investigations are useful in assessing a soft tissue injury of the knee e.g. indications to perform an MRI, distinction between the utility of weight-bearing versus non-weight-bearing radiograph in assessing osteoarthritis?	4
5. Define and describe the grading systems/scales for chondral thinning/articular cartilage change/OA such as the Kellgren-Lawrence, Outerbridge, ICRS (International Cartilage Repair Society) Scales. What is mild/moderate/severe OA?	4
6. What are the tests for meniscal pathology? What is the sensitivity/specificity/reliability of meniscal testing (e.g. McMurray's test)? What is the relevance of joint line tenderness?	4
7. What are the typical symptoms of meniscal pathology/OA/ACL injury? What are the features (clinical examination and radiological findings) of meniscal; anterior cruciate ligament; OA; and chondral injuries?	4
8. What are the radiological findings in patellofemoral instability? What are the measurements? What do they mean? What are normal/abnormal values?	5
9. What are the common MRI findings after a lateral patella dislocation/ subluxation episode in a patient with underlying patellofemoral alignment abnormalities (patella alta and trochlear dysplasia) versus a client without these underlying factors?	6
10. What is a normal tibial tubercle trochlear groove (TT-TG) distance in a knee (patellofemoral joint) and what is the relationship between this distance and patella instability?	6
11. What is the relationship between lateral patella dislocation and TT-TG, patella alta and trochlear dysplasia?	6
12. What is the relevance of subchondral/subcortical cystic change/sclerosis to meniscal/chondral pathology/OA in the knee?	7
13. Treating osteoarthritis of the knee with arthroscopy, is this an appropriate treatment in terms of efficacy?	7
 <b>CAUSATION OF KNEE PATHOLOGIES/INJURIES - PART B</b>	 <b>9</b>
<b>Mechanisms of knee injury, clinical presentation, diagnosis and imaging of knee pathology</b>	<b>9</b>
Common knee injuries, mechanism of injury, soft tissues structures involved and contusion patterns	9
General - Imaging of knee injuries	10
General – note on mechanical laxity	11
General - Grading of ligament injuries	11
ACL injuries	11
Meniscal injuries	15
Chondral injuries	18
 <b>Patellafemoral instability, subluxation and dislocation</b>	 <b>21</b>
 <b>Subchondral cysts</b>	 <b>24</b>
 <b>Arthroscopic treatment of OA</b>	 <b>25</b>
 <b>References</b>	 <b>27</b>

## Summary

1. What are the typical mechanisms of injury and mechanisms of accident in: meniscal, anterior cruciate ligament (ACL) and chondral /osteochondral injuries?

Key reference - Sanders<sup>1</sup>. This paper describes the common mechanisms of knee injuries with figures, MRI interpretation and bone edema pattern.

Sanders et al have described five key patterns of knee injury (Table 1) together with descriptions of the pattern of bone edema and the soft tissues involved. The five patterns described are: hyperextension, pivot shift, dashboard, clip injury and patellar dislocation. These authors consider that the pattern of bone contusion is indicative of the mechanism of injury. More detailed descriptions of typical mechanisms of injury are also included, using other references.

2. Are ACL disruptions always due to single episode trauma?

It is increasingly being recognised that the ACL is subject to degeneration with use, injury, disease and aging, in a manner analogous to that of the meniscus. The authors of a histological study in cadavers<sup>2</sup> found that mucoid degeneration of the ACL was present in 62% of subjects examined and cystic changes in 37%. The sum of all histological changes recorded increased with age.

The concept of partial tearing of the ACL is also recognised though there is debate about how it is defined, diagnosed and treated<sup>3</sup>. Reliable epidemiological studies on the prevalence of partial tears do not seem to be available, but they may represent 10 to 35% of ACL lesions<sup>4,6</sup>. Partial tears may follow a benign course, but some will progress to complete ACL deficiency<sup>3</sup>.

It seems likely then that ACL tears need not always be due to a single case of trauma and may be preceded by partial tearing and/or degenerative processes.

3. What is the prevalence of ACL disruption in asymptomatic individuals?

In a Framingham study of community living subjects (mean age 62; age range 51 to 89), free of radiographic evidence of osteoarthritis (OA), the prevalence of ligamentous lesions, described as complete tears of the ACL or posterior cruciate ligament (PCL), was 9% in total (8% of women and 11% of men). Partial tears were not counted. The presence of ACL tears was not significantly associated with pain<sup>7</sup>.

In a cross-sectional study of community-living patients with painful OA of the knee, about 23% had a complete ACL tear, of which about half had no prior history of ligament injury. In the control group with no knee pain, prevalence of complete ACL tears was 2.7%<sup>8</sup>.

4. What investigations are useful in assessing a soft tissue injury of the knee, e.g. indications to perform an MRI, distinction between the utility of weight-bearing versus non-weight-bearing radiographs in assessing osteoarthritis?
5. Define and describe the grading systems/scales for chondral thinning/articular cartilage change/OA such as the Kellgren-Lawrence, Outerbridge, ICRS (International Cartilage Repair Society) scales. What is mild/ moderate/severe OA?
6. What are the tests for meniscal pathology? What is the sensitivity/specificity/reliability of meniscal testing (e.g. McMurray's test)? What is the relevance of joint line tenderness?
7. What are the typical symptoms of meniscal pathology/OA/ACL injury? What are the features (on clinical and radiological examination) of meniscal, anterior cruciate ligament and chondral injuries, and of OA?

Key references – ACC MRI guideline 2010<sup>9</sup>, ACC Diagnosis and management of soft tissue knee injuries guideline 2003<sup>10</sup> (contains a diagnostic algorithm,pg. 12)

NB: definitions, sensitivity and specificity of tests are given in the main text.

#### ACL tears

Key reference -Guenoun<sup>5</sup> - MR imaging, anatomy, partial tears

A typical history<sup>10</sup> may include an audible 'pop', a sense of disruption, or that the knee 'came apart', and significant swelling occurring within a few hours. This presentation alone has been reported as being 70% accurate for the diagnosis of an ACL rupture. It should be noted that it is important to exclude other injuries associated with the development of early swelling, for example osteochondral fractures and patellar dislocations. The ability to continue the activity or walk off the field does not exclude an ACL injury.

A clinical examination for ACL tear performed by an orthopaedic surgeon is highly predictive of ACL tearing and an MRI has no additional value when the physical examination has shown anterior-posterior or rotational instability of the knee<sup>11</sup>. MRI has good sensitivity and specificity to detect full ACL tears but not partial tears<sup>5</sup> and it is useful to establish other intraarticular lesions. Performance of a complete physical exam (Lachman, pivot shift, anterior drawer test) has a higher sensitivity and specificity than a partial examination<sup>11</sup>.

#### Meniscal tears

Key reference - Alatakis<sup>12</sup> - this reference describes types of tears and also cartilage injuries

Typically, individuals who sustain a meniscal tear report a history of hearing a 'pop' while suddenly changing direction with or without contact<sup>13</sup>. No or minimal force may be a sufficient cause of injury in a degenerative meniscus. Patients may describe locking (on flexion) or catching, but these symptoms are not specific to meniscal injuries.

A history of a squatting or twisting injury associated with joint line tenderness, an effusion and loss of extension are strongly suggestive of a meniscal tear<sup>10</sup>.

Physical tests include joint line tenderness, the McMurray test and the Thessaly test, all of which have variable sensitivity and specificity.

MRI is sensitive and specific for the diagnosis of meniscal tears<sup>12 14</sup>.

#### Chondral damage

Mechanical symptoms of catching, locking, crepitus and instability may be in the history, but signs may be minimal with pure chondral injuries; pain may feature when the underlying subchondral bone is involved<sup>15</sup>.

The extent of cartilage injury may be assessed at arthroscopy using the Outerbridge scale or the International Cartilage Repair Society (ICRS) gradings<sup>16</sup> (Table 5).

The Kellgren Lawrence classification for OA is the most widely used radiological classification to identify and grade OA but it is widely recognised to have limitations. The definition of radiographic OA relies on the presence of definite osteophytes on the anteroposterior weight bearing x-ray. Grade 2 is usually used as the threshold as assessed on anteroposterior weight-bearing x-rays. There are a number of versions of the Kellgren Lawrence classification in use; the original version is given in Table 6.

Assessment of OA radiographically requires a bilateral anteroposterior image be taken while the patient is weight-bearing. Originally this was done with both knees in full extension, but it is now known that the knee should be in 30° or 45° flexion as these positions allow for more sensitive detection of joint space narrowing<sup>17 18</sup>. The ACC guidelines require that the radiographic image be taken 'weight-bearing' with the knee slightly flexed, and that weight-bearing x-rays accompany any referral for a specialist opinion<sup>9 10</sup>.

Cartilage injury can be assessed by MRI with an accuracy >90% compared to arthroscopy<sup>16</sup>. For MRI the International Cartilage Repair Society scale is often used over that of the Outerbridge scale (Table 5).

According to Recht<sup>19</sup> MRI is often capable of distinguishing between the traumatic and degenerative origins of chondral injuries<sup>19</sup>. Early degenerative changes may be shown by fibrillation and surface irregularity, changes in cartilage thickness and intrachondral alterations in signal intensity. Advanced changes are reflected in multiple areas of cartilage thinning of varying depth and size, edema, cysts and subchondral fibrosis or trabecular sclerosis. Traumatic chondral lesions appear as solitary focal cartilage defects with acutely angled margins often associated with bone bruising/edema or subchondral fracture<sup>19</sup>.

Further means of distinguishing acute posttraumatic chondral injuries from chronic are also discussed<sup>20</sup>.

#### **8. What are the radiological findings in patellofemoral instability? What are the measurements? What do they mean? What are normal/abnormal values?**

Key reference – Redziniak<sup>21</sup> - a straightforward review of patellar instability

The patellar height can be assessed on a lateral radiograph. Indices used include the Blackburne-Peel index, which relates the length of the articular surface of the patella to the distance of its inferior margin from the tibial plateau. It ranges from 0.85-1.09 in men and 0.79-1.09 in women. The Insall-Salvati index is an alternative index which compares the diagonal length of the patella with the length of the patellar tendon. It ranges from 0.9–1.1 in men and 0.94–1.18 in women. This index has the limitation that the length of the patella is not always an indication of the length of its articular surface<sup>21</sup>.

Other measures are also discussed in this report. It has been concluded that radiological measurements of patellar instability have low intra and inter-rater reliability<sup>22</sup>.

**9. What are the common MRI findings after a lateral patellar dislocation/ subluxation episode in a patient with underlying patellofemoral alignment abnormalities (patella alta and trochlear dysplasia) versus a client without these underlying factors?**

An American study<sup>23</sup> has reported that the common MRI findings in subjects with lateral patellar dislocation were joint effusion, contusion of the lateral femoral condyle or medial patella, osteochondral fragments and injury to the medial stabilisers, the medial retinaculum and the medial patellofemoral ligament (MPFL). The MPFL was injured in 98.6% of patients. See also questions, 8, 10, and 11.

**10. What is a normal tibial tubercle trochlear groove (TT-TG) distance in a knee (patellofemoral joint) and what is the relationship between this distance and patellar instability?**

A relatively large American study<sup>24</sup> has compared TT-TG distances in patients (5–42 years) with lateral patellar instability to those in controls. It reported that the mean TT-TG width was 14.6 +/- 4.6mm in patients compared to 10.6 +/- 4.0mm in controls, with 11% of patients having a TT-TG width >20mm (controls 2.2%) and 35% a distance >15mm (controls 10.3%). TT-TG distance was found to be independent of age.

Pandit et al<sup>25</sup> in a New Zealand based study have reported reference values for 100 males and females to be 9.91mm (95% CI 8.9-10.8) for males and 10.04mm (8.9-11.1) for females. They summarised their data by concluding that the reference values for TT-TG were 10 +/- 1mm.

In an English study<sup>26</sup> a cut-off of 14.5mm for TT-TG had a sensitivity of 80% and a specificity of 70% for detecting radiological instability.

**11. What is the relationship between lateral patellar dislocation and TT-TG, patella alta and trochlear dysplasia?**

It has been observed that patellofemoral instability has a spectrum of severity ranging from mildly abnormal patellofemoral tracking to complete dislocation where no part of the patella remains in the trochlear groove<sup>26</sup>. Subluxation exists when the apex of the patella is not sliding centrally down the trochlear groove. Variables able to discriminate between grades of patellar subluxation (mild, moderate, severe) have been determined as:

- Patellar engagement (% overlap of patella in trochlear sulcus)
- TT-TG (distance between tibial tuberosity and apex of trochlear notch)
- VMOP (distance of vastus medialis obliquus from patella)
- Patella alta (patellar length /tendon length)
- Sulcus angle (proximal- bone, see paper<sup>26</sup> for definition)
- Sulcus angle (proximal – cartilage, see paper for definition).

Of these six measures, patellar engagement had the strongest association with subluxation. No patient with an engagement of <30% appeared in the mild or no subluxation groups. The best cut-off point for predicting radiological instability was a patellar engagement of <=30%. Patella alta had less discriminatory power than patellar engagement. TT-TG increased from a mean of 12.0mm (SD 4.1) for mild or no

subluxation to a mean of 20.7mm (SD 4.3) for severe subluxation. The researchers found the best cut-off point for TT-TG to be 14.5mm for detecting radiological instability. VMOP ranged from 3.3mm (SD 2.9) for mild or no subluxation to 8.2 (SD 2.8) for the severe group.

## 12. What is the relevance of subchondral/subcortical cystic change/sclerosis to meniscal/chondral pathology/OA in the knee?

Key reference - Crema<sup>27 28</sup>

Subchondral cysts contain small fragments of necrotic bone etc. and are surrounded by a layer of fibrous connective tissue without any epithelial component. They may or may not communicate with the joint cavity<sup>27</sup>. It has been suggested that the term cyst is inappropriate and a better term would be 'subchondral cyst-like bone marrow lesions'<sup>27</sup>.

They appear in regions without full-thickness cartilage defects in around 50% of cases and are strongly associated with bone marrow lesions in the same subregion<sup>18</sup>. One larger cohort study of those at risk of developing OA or with OA has reported that 91.2% of the cysts were associated with bone marrow lesions and located in the lesion or adjacent to it<sup>27</sup>. In addition, prevalent bone marrow lesions showed a strong and significant association with incident subchondral cysts in the same subregion of the knee<sup>28</sup>.

Subchondral cysts in addition to having strong associations with bone marrow lesions are also -

- Commonly found in older subjects free of radiographic OA - in a Framingham study of community-living subjects free of radiographic evidence of OA (age range 51 to 89) the prevalence of subchondral cysts was 25% and prevalence increased with age<sup>7</sup>.
- Strongly associated with Kellgren Lawrence score and advanced OA<sup>29</sup>.

It is concluded that subchondral cysts have significant associations with bone marrow lesions, age and advancing OA.

## 13. Treating osteoarthritis of the knee with arthroscopy: is this an appropriate treatment in terms of efficacy?

Key reference – AAOS guideline<sup>30</sup>

There have been two randomised controlled trials that have investigated the use of arthroscopic lavage and removal of unstable meniscal tears etc. in those with OA. In both trials it was concluded after two years of follow-up that arthroscopic treatment was not superior to conservative treatment.

The American Association of Orthopaedic Surgeons (AAOS)<sup>30</sup> recommend against performing arthroscopy with debridement or lavage in patients with a primary diagnosis of symptomatic OA of the knee. They also recommend that arthroscopic partial meniscectomy or loose body removal is an option in patients with symptomatic OA of the knee who also have primary signs and symptoms of a torn meniscus and/or a loose body.

Further a recent large multicentre American RCT has found that in those over 45 years of age with mild to moderate OA and evidence of a meniscal tear, surgery for the tear

plus post operative physical therapy was of no greater benefit at 6 months and 12 months compared to physical therapy \*please see endnote on page 31.

There is therefore a significant body of evidence that in those of middle to older age with mild to moderate OA, surgery in the knee for relief of OA and/or repair or resection of partial meniscal tears of either traumatic or degenerative origin is in the great majority of cases no better than conservative treatment.



## Causation of knee pathologies/injuries - Part B

**Mechanisms of knee injury, clinical presentation, diagnosis and imaging of knee pathology**

**Common knee injuries, mechanism of injury, soft tissue structures involved and contusion patterns**

Key reference - Sanders<sup>1</sup>. This paper describes the common mechanisms of knee injuries with figures, MRI interpretation and bone edema pattern.

Sanders et al have described five key patterns of injury (Table 1) together with descriptions of the pattern of bone edema and the soft tissues involved. These authors consider that the pattern of bone contusion is indicative of the mechanism of injury. For further notes on bone edema pattern, analysed by knee structure involved, see below - 'Chondral Injuries - Distinguishing acute, post traumatic chondral injuries from chronic'.

Table 1. Sander's analysis of common knee injuries, soft tissues involved and locations of bone edema<sup>1</sup>

Authors' name for the injury	Description	Soft tissue structures affected	Contusion location (for further notes on bone contusion/ edema see page 19 of this report and page 21 of Part A)
Hyperextension	Knee bent back as after landing wrongly from a jump. In severe cases the injury can result in impaction of the anterior aspect of the femoral condyle against the anterior aspect of the tibial plateau. If a valgus force is applied as well the lesions will be located medially.	ACL, PCL and meniscal tears.	Kissing contusion pattern involving the anterior aspect of the proximal tibia and distal femur <sup>1</sup> .
Pivot shift injury	Valgus force /flexion/external rotation of the tibia or internal rotation of the femur.	ACL tear, and occasionally tears of the posterior capsule and arcuate ligament, the posterior horn of the lateral and medial meniscus and the medial collateral ligament.	Posterolateral tibial plateau and the midportion of the lateral femoral condyle.

Dashboard injury	Force applied to the anterior aspect of the proximal tibia while the knee is flexed.	Rupture of the PCL, and the posterior joint capsule.	Anterior aspect of the proximal tibia and occasionally on the posterior surface of the patella.
Clip injury	A pure valgus force applied while the knee is flexed.	Varying degrees of sprain or disruption to the medial collateral ligament (MCL).	A prominent area of edema at the lateral femoral condyle and a smaller area of edema involving the medial femoral condyle.
Lateral patellar dislocation	Common in younger subjects involved in athletic activities. The femur rotates internally on a fixed tibia while the knee is flexed; contraction of the quadriceps occurs resulting in lateral dislocation of the patella.	Sprain or disruption of the medial soft-tissue restraints.	Edema involves the inferomedial patella and the anterior aspect of the lateral femoral condyle.

## General - Imaging of knee injuries

Key reference – ACC Knee MRI guideline<sup>9</sup>

Key reference – ACC Diagnosis and management of soft tissue knee injuries guideline<sup>10</sup> (contains a diagnostic algorithm, pg 12)

ACC has recently published MRI guidelines for diagnosis of soft tissue knee injuries. They state that MRI is indicated for assessment of ACL injuries and meniscal injuries but is not always necessary when the diagnosis is clear. This guideline also requires that weight bearing x-rays accompany any referral for a specialist opinion<sup>9</sup>.

The Ottawa Knee rules are widely accepted as an important standard for investigation of acute knee injuries. Patients who do not meet the rules are highly unlikely to have clinically significant fractures and can have knee radiographs safely deferred.

The Ottawa Knee rules indicating need for x-rays after acute knee injury are –

- aged 55 years or over
- tenderness at the head of the fibula
- isolated tenderness of the patella
- inability to flex knee greater than 90 degrees
- inability to bear weight (defined as an inability to take four steps, i.e. two steps on each leg, regardless of limping) immediately and at presentation

## **General – note on mechanical laxity**

Mechanical laxity<sup>10</sup> refers to an excess in the range of movement in the joint due to loss of integrity of the ligaments and other soft tissues which contribute to joint stability.

Functional instability<sup>10</sup> refers to a 'sense of instability' or 'giving out' of the joint experienced by the person in the course of their usual activities. It may or may not be associated with mechanical laxity.

## **General - grading of ligament injuries**

Ligament injuries are commonly graded according to the severity of the injury<sup>10</sup>.

Grade I: a grade I ligament injury has no increased laxity and there is a firm end-point in testing. There is pain and tenderness over the course of the ligament, but no actual disruption of fibres has occurred.

Grade II: grade II ligament injuries involve some disruption of the fibres. There is some increased laxity compared with the other knee, but still a firm end-point on testing. There is usually pain and tenderness over the course of the ligament.

Grade III: grade III ligament injuries involve a complete rupture of the ligament with gross laxity and a 'mushy' end feel, or no end feel on testing. There may not be any tenderness over the ligament because there are no 'intact' fibres to stress while testing.

## **ACL injuries**

Key references: mechanism of ACL injury - Boden<sup>31</sup>; MR imaging, anatomy, partial tears - Guenoun<sup>5</sup>

### **Epidemiology**

Based on sport exposure measures, women have higher ACL injury rates than men<sup>32</sup>. In basketball players the female:male injury rate was 3:1. American studies suggest that reconstruction rates are highest overall in 18-34 year old males, and in females rates are highest in the 14-17 year age group followed by the 18-21 year old group<sup>32</sup>.

In a Framingham study of community living subjects (mean age 62; age range 51 to 89) free of radiographic evidence of OA, the prevalence of ligamentous lesions, described as complete tears of the ACL or PCL, was 9% in total (8% of women and 11% of men). Partial tears were not counted. Their presence was not significantly associated with pain<sup>7</sup>.

In a cross-sectional study of patients with painful OA of the knee (community and Veterans Administration cohorts of similar ages [age means varied from 68 to 65 depending on the group]) about 23% had a complete ACL tear, of which about half had no prior history of ligament injury. In the control group with no knee pain, prevalence of complete ACL tears was 2.7%<sup>8</sup>.

It does not appear to be known whether or not OA can result in ACL tears but it is well known that ACL tears will lead to OA in many subjects<sup>8</sup>.

### **Clinical presentation and symptoms**

A typical history<sup>10</sup> may include an audible 'pop', a sense of disruption, or that the knee 'came apart', with significant swelling occurring within a few hours. This presentation alone has been reported as being 70% accurate for the diagnosis of an ACL rupture. It should be noted that it is important to exclude other injuries associated with the development of early swelling, for

example, osteochondral fractures and patellar dislocations. The ability to continue the activity or walk off the field does not exclude an ACL injury.

### Mechanism of ACL injury

A French study<sup>5</sup> suggests that there are three main mechanisms of ACL injury –

- Valgus force/flexion/external rotation responsible for damage to the medial collateral ligament (MCL), the posteromedial corner, the ACL and the lateral meniscus. The external rotation results in the medial plateau advancing. The MCL may be stretched and the ACL and meniscus torn.
- Varus force/flexion/internal rotation which damages the ACL, the posterolateral corner, the lateral collateral ligament and the lateral meniscus and causes a Segond fracture. Internal rotation causes the lateral tibial plateau to move forward. The movement is opposed by the ACL. The ACL tear may be isolated.
- A hyperextension accident in which the ACL is injured against the intercondylar fossa.

Somewhat contrary to the above are the results of studies investigating non-contact mechanisms of ACL ruptures which account for about 72% of ACL ruptures<sup>32</sup>. It seems clear that the primary mechanism of non-contact ACL injury is increased tibia anterior translation<sup>31 32</sup>. Boden<sup>31</sup> describes the force leading to non-contact ACL rupture as ‘a compressive force acting on the posterior tibial slope which results in posterior displacement of the femoral condyle on the tibial plateau’. The compressive forces translated through the knee result mainly from inadequate absorption of the landing forces, though quadriceps and hamstring contraction can contribute<sup>31</sup>.

The authors considered that the mechanism described above was consistent with the pattern of bone bruising seen with non-contact ACL ruptures – i.e. medial and lateral femoral condyle bone bruises symmetric with respect to severity and depth<sup>32</sup>.

It was considered that knee valgus moment and internal rotation moments are unlikely on their own to rupture the ACL in non-contact situations but may contribute to the rupture when associated with an anterior shear force applied at the proximal end of the tibia<sup>32</sup>.

### Diagnosis

A clinical examination for ACL tear performed by an orthopaedic surgeon is highly predictive and an MRI has no additional value when the physical examination has shown anterior-posterior or rotational instability of the knee<sup>11</sup>. MRI is useful to establish other intraarticular lesions. Performance of a complete physical exam (Lachman, pivot shift, anterior drawer test, see Table 2) has a higher sensitivity and specificity than a partial examination<sup>11</sup>.

Table 2. Tests for diagnosis of ACL injuries<sup>33</sup>

Test	Description	Sensitivity %	Specificity %
Anterior drawer	The subject is supine, hip flexed to 45° and knee flexed to 90°. Examiner places his hands behind the proximal tibia and thumbs on the tibial plateau. Anterior force is applied to the proximal tibia. Hamstring tendons palpated with index fingers to ensure relaxation.	55	92

	Increased tibial displacement compared with the opposite side is indicative of an ACL tear.		
Lachman	Patient lies supine. Knee held between full extension and 15° of flexion. Femur is stabilized with one hand while firm pressure is applied to the posterior aspect of the proximal tibia in an attempt to translate it anteriorly. Test is positive (indicating ACL rupture) when there is anterior translation of the tibia with “soft” endpoint.	85	94
Pivot shift	The knee is fully extended and the foot internally rotated. A valgus stress is applied while progressively flexing the knee and watching and feeling for translation of the tibia on the femur.	24	98

## Imaging

### Key reference - Guenoun<sup>5</sup>

Guenoun et al<sup>5</sup> have described the MR imaging of knee ACL injuries. Sensitivity of diagnosing an ACL tear is 92-96% and specificity is 92-98%. These authors suggest that MRI is particularly useful when clinical examination is impossible or doubtful, and for detecting associated lesions.

The two direct specific signs of an ACL tear are –

- Partial or total discontinuity of the ACL in at least one reading plane
- Horizontalisation of the distal fragment of the ACL.

Other less specific signs and indirect signs that support diagnosis of an ACL tear have also been identified (see Guenoun<sup>5</sup>; for example, anterior subluxation of the lateral tibial plateau relative to the femur).

It is difficult to detect partial tears of the ACL by MRI<sup>5</sup>.

### Bilateral ACL injuries

Small numbers of subjects do experience ACL injuries in both knees on different occasions. Two cases series found frequencies of bilateral injuries of 4.8% and 7.7%. In one case series such subjects were all young women with a mean interval between the two occurrences of 2.4 years. All injuries were incurred during sports activities. Such subjects are reported to have higher knee laxity scores than those with unilateral injuries<sup>34</sup>.

### Risk factors for ACL injury

Risk factors for ACL injuries have been systematically reviewed<sup>35 36</sup>. The authors concluded that female sex, specific measures of bony geometry of the knee joint (e.g. decreased intercondylar femoral notch width, increased slope of the tibial plateaus), prior reconstruction of the ACL and

familial disposition were important factors. The authors considered that many factors are likely to act in combination to effect likelihood of injury.

Cadaveric studies<sup>37</sup> suggest that some of the sex differences in rates of ACL injury reflect ACL size and structural differences between males and females. The study also showed that gender, age, body mass and height were relevant variables affecting risk.

#### Partial tearing of the ACL

The concept of partial tearing of the ACL is recognised, though there is debate about how it is defined, diagnosed and treated<sup>3</sup>. The clinical diagnosis of a partial tear is made by assessing the degree of laxity at clinical examination; it may be difficult to diagnose partial tears from an intact ligament. MRI is not definitive<sup>5</sup>. Partial tears may follow a benign course but some will progress to complete ACL deficiency<sup>3</sup>. Reliable epidemiological studies on prevalence of partial ACL tears do not seem to be available, but they may represent 10 to 35% of ACL lesions<sup>4,6</sup>.

It seems likely then that ACL tears need not always be due to a single case of trauma.

#### ACL degeneration

In advanced OA, histological changes in the ACL are very common and include cystic changes, disorientation of collagen fibres and mucoid degeneration<sup>2</sup>. Mucoid degeneration of the ACL is composed of glycosaminoglycan deposits around the collagen bundles causing hypertrophy.

In one case series referred for MRI, mucoid lesions of the ACL were found in 1.8% of cases; 24% had mucoid degeneration and 76% mucoid cysts<sup>38</sup>. The ratio of occurrence between the sexes was about 1:1 and the mean age of those with mucoid degeneration was 42 years.

Posterior pain of the knee is the most common symptom of ACL degeneration<sup>38</sup>. Mucoid degeneration is said to be diagnosable by MRI<sup>38</sup> but prevalence as determined by MRI appears to be much lower than that determined histologically (see next paragraph).

A histological study in cadavers<sup>2</sup> (120 knee joints in 65 subjects, mean age 66.1 years, age range 23 – 92) has shown that –

- The rate of ACL rupture was around 10% in the sample.
- In those with severe OA the rate of ACL rupture was 24.1%.
- The total ACL histological score, being the sum of all histological changes, increased with age.
- There were no normal ACLs in those with grade 2 to 4 cartilage changes (Outerbridge/ICRS scores), suggesting that ACL changes accompany cartilage degeneration.
- Mucoid degeneration was present in 62% of subjects in contrast to much lower levels detected by MRI and is therefore likely to be a very common occurrence.
- Mucoid degeneration was present in 4 of the 9 subjects who were less than 45 years of age and without cartilage degeneration.
- Cystic changes were seen in 37% of subjects. The authors considered that cystic changes were probably a relatively late event in ACL degeneration.

- Mucoïd degeneration and disorientation of the collagen fibres were the earliest detectable age-related changes in the ACL and these changes could occur in the absence of cartilage degeneration.
- The authors concluded that ACL degeneration is highly prevalent in knees with cartilage defects and may precede cartilage changes; ACL deficiencies may not only be important in post traumatic OA but may also be a feature associated with knee OA in general.

Surgical reconstruction v. conservative rehabilitation

#### Key reference - Frobell<sup>39</sup>

A randomised controlled trial (RCT) has compared structured rehabilitation plus early ACL reconstruction with structured rehabilitation plus the option of delayed reconstruction in adults 18 – 35 years of age. At two years there was no difference in pain, symptoms, sports function or knee-related quality of life<sup>40</sup>. At five years similar results were reported. Over the 5 years, only 50% of those who initially opted for structured rehabilitation with the option of delayed reconstruction subsequently elected to have surgery, making a significant impact on surgical intervention rates<sup>39</sup>.

### **Meniscal injuries**

Clinical presentation and symptoms

Typically individuals who sustain a meniscal tear report a history of hearing a ‘pop’ while suddenly changing direction with or without contact<sup>13</sup>. No or minimal force may be sufficient cause of tearing in a degenerative meniscus. Patients may describe locking (on flexion) or catching, but these symptoms are not specific to meniscal injuries.

A history of a squatting or twisting injury associated with joint line tenderness, an effusion and loss of extension are strongly suggestive of a meniscal tear<sup>10</sup>.

Mechanism of tearing: degeneration or trauma

Meniscal injuries<sup>10</sup> result from either ‘acceleration or deceleration typically associated with flexion, tibial rotation and compression components causing a shear stress across the meniscus’. For younger people, the types of movement typically associated with meniscal injuries include twisting, squatting or cutting manoeuvres. For middle-aged and older people, injury can occur with more trivial movements. Critical aspects of the history of meniscal injuries include the mechanism of injury, catching, locking, pain and swelling. Locking is commonly associated with a meniscal tear and has been defined as ‘an acute block to extension with some flexion possible’. Locking however can also be associated with anterior cruciate ligament tears, loose bodies, degenerative changes and muscle spasm<sup>10</sup>.

Note that rotation of the knee joint does not occur in full extension and it is very uncommon for both menisci to be torn in a single event<sup>41</sup>.

Classification of tears according to traumatic or degenerative origin is common in the literature but is not absolute. Degenerative tears are very common in those over 50 years of age and may occur in the absence of acute injury (see part A of this report).

Drosos et al<sup>42</sup> have described a case series of 392 subjects with normal x-rays (average age 39, age range 18-60) who had meniscal tears verified at arthroscopy (exclusions were those with OA, rheumatoid or other inflammatory diseases or previous knee injuries or surgery). Their findings are summarised in Table 3. Drosos et al reported that very large numbers of subjects,

though relatively young, had tears of degenerative origin and the injury mechanism was for many either minor or there was no injury history at all. In this case series then, the number who had a non-degenerative type of tear and who had also experienced a significant trauma event was quite small.

Table 3. Analysis of a case series<sup>42</sup> of subjects with meniscal tears: contribution of significant trauma and tear type to the total case load.

Injury type	% of case series	% injured by a significant trauma	% with non-degenerative tear	Average age (years)	Notes
Sports injury	32.4	100	55.9	33	59.1% of injuries occurred in contact sports. 10.2% occurred while jogging <u>and in the absence of a twisting injury.</u>
Non sport injury	38.8	28.1	38.2	41	Of the 72% who were injured by minor trauma, 51% were injured in the process of crouching or when getting up from a squatting position.
No identifiable injury	28.8	0	18.6	43	

## Diagnosis

The American Physical Therapy Guideline<sup>13</sup> claims that meniscal tears may be diagnosed with a fair level of certainty when the patient presents with the following clinical findings –

- Twisting injury
- Tearing sensation at the time of injury
- Delayed effusion (6-24 hours post injury)
- History of catching or locking
- Pain with forced hyperextension
- Pain with maximum flexion
- Pain or audible click with McMurray test
- Joint line tenderness
- Discomfort or sense of locking/catching over the medial or lateral joint line during the Thessaly test when performed at 5° or 20° flexion.

Some studies suggest that clinical examination by an experienced examiner using multiple meniscus tests is sufficient for diagnosis of a meniscal tear<sup>43</sup>.



The characteristics of physical tests for meniscal tears are listed in Table 4. The sensitivity and specificity of these tests is variable.

Table 4. Characteristics of tests for meniscal tears<sup>13</sup>.

Test	Description	Sensitivity Medial %	Sensitivity Lateral %	Specificity Medial %	Specificity Lateral %
Joint line tenderness	Amount of tenderness along medial and lateral joint line	83	68	76	97
McMurray Test	The patient is supine. The examiner grasps the ankle of the tested limb with one hand. The opposite hand is placed on the tested knee with the thumb over the lateral joint line and the middle finger over the medial joint line. The knee is maximally flexed, externally rotated and then slowly extended to assess the medial meniscus. The knee is maximally flexed, internally rotated and then slowly extended to evaluate the lateral meniscus. A palpable or audible click is positive.	50	21	77	94
Thessaly Test	The patient is standing. The patient is instructed to stand on the tested limb. The patient can use upper extremity support by holding the clinician's hands during the test. The patient rotates his knee and body internally and externally 3 times with the knee in 5° and 20° of flexion. Discomfort or sense of locking over the joint line is positive.	41-66	16-81	68-86	89-91

## Imaging of meniscal tears

Key reference - Alatakis<sup>12</sup> - this reference describes types of tears and also cartilage injuries

MRI is considered to be an accurate diagnostic tool for the detection of meniscal pathology. For medial tears sensitivity is 93.3% and specificity 88.4%; the corresponding figures for lateral tears are 79.3% and 95.7%<sup>44</sup>.

A 2012 study<sup>14</sup> has investigated the accuracy of MRI compared to arthroscopy for the diagnosis of meniscal tears in older patients. The study showed that specificity fell off markedly when equivocal assessments of tearing were included in the analysis; this emphasises the fact that MRI readings are not always absolute.

In advanced OA, meniscal tears are very common and MRI is not required to prove their presence in this case<sup>45 46</sup>. This is true even when the OA is not symptomatic.

## Arthroscopic treatment of degenerative meniscal tears

### Key reference - Herrlin<sup>47 48</sup>

Arthroscopic partial meniscectomy for degenerative medial meniscal tears in subjects 45-65 years of age with constant non-traumatic knee pain, but no or low grades of OA, has been compared with a conservative programme of regular exercises in a randomised controlled trial<sup>47</sup>. Results were examined on both an 'intention to treat' basis and by 'per - protocol'. At five years of follow up<sup>48</sup>, arthroscopic surgery and exercise was not superior to exercise therapy alone. One third of patients from the exercise group who did not improve with exercise went on to have arthroscopic surgery. The authors considered that exercise therapy could be recommended as the initial treatment.

According to Englund<sup>49</sup>, even in painful knees partial meniscectomy is a questionable alternative in middle-aged to older subjects.

## Chondral injuries

### Clinical presentation and symptoms

Mechanical symptoms of catching, locking, crepitus and instability may be in the history, but signs may be minimal with pure chondral injuries. Pain may feature when the underlying subchondral bone is involved<sup>15</sup>.

### Mechanism of injury

32% to 58% of chondral injuries result from a traumatic, non-contact mechanism<sup>13</sup>. Established risk factors for chondral lesions associated with ACL injuries are increasing patient age and a longer time from ACL injury<sup>13</sup>.

### Diagnosis and classification

The extent of cartilage injury may be assessed at arthroscopy using the Outerbridge scale or the International Cartilage Repair Society (ICRS) grade<sup>16</sup> (Table 5).

Table 5. Outerbridge and ICRS grading of cartilage lesions<sup>16</sup>.

Grade	Outerbridge	ICRS
0	Normal	Normal
1	Intact surface cartilage with softening and swelling	Superficial lesion; soft indentation and/or superficial fissures and cracks
2	A partial thickness defect with fissures on the surface that do not reach subchondral bone or exceed 1.3cm	Lesions extending down to <50% of cartilage depth
3	Fissuring to the level of the subchondral bone in an area with a diameter >1.3cm	Cartilage defects extending down >50% of the cartilage depth as well as down to calcified layer and down to but not through subchondral bone
4	Exposed subchondral bone	Complete loss of cartilage cover, bone only

The Kellgren Lawrence (KL) classification for OA is the most widely used radiological classification to identify and grade OA. The definition of radiographic OA relies on the presence of definite osteophytes on the anteroposterior weight bearing x-ray. Grade 2 is usually used as the threshold as assessed on anteroposterior weight-bearing x-ray. There are a number of versions used: the original version is given in Table 6.

The KL system is limited by the invalid assumption that radiographic changes appear in a linear manner and the fact that it is a composite measure of joint space width and osteophyte presence<sup>50</sup>. Despite these limitations, progression of OA over three years was associated with KL score at baseline: the higher the grade the more likely it was to progress<sup>51</sup>.

Table 6. Original descriptions of the Kellgren Lawrence classification of OA<sup>52</sup>

Grade	Description
0	No features of OA
1	Doubtful narrowing of joint space and possible osteophytic lipping
2	Definite osteophytes and possible narrowing of the joint space
3	Moderate multiple osteophytes, definite narrowing of the joint space and some sclerosis and possible deformity of bone ends
4	Large osteophytes, marked narrowing of the joint space, severe sclerosis and definite deformity of bone ends

## Imaging

Key reference - Recht<sup>19</sup>, especially for notes on distinguishing between chondral lesions of traumatic and degenerative origin (see page 903 of the paper)

Assessment of OA radiographically requires a bilateral anteroposterior image taken while the patient is weight-bearing. Originally this was done with both knees in full extension but it is now known that the knee should be in 30° or 45° flexion as these positions allow more sensitive detection of joint space narrowing<sup>17 18</sup>. The ACC guideline requires that the image be taken weight-bearing with the knee slightly flexed<sup>10</sup>.

Cartilage injury can be assessed by MRI with an accuracy >90% compared to arthroscopy<sup>16</sup>. The advantage of the ICRS system (above) is that it is more readily linked to the MRI image whereas the Outerbridge poses difficulties<sup>53</sup>. Other classification systems have been devised that are designed for use in association with MRI and have an emphasis on the whole knee joint<sup>18</sup> (e.g. the Whole Organ MRI Score, Knee Osteoarthritis Scoring System, Boston Leeds Osteoarthritis Knee Score).

According to Recht<sup>19</sup> MRI is often capable of distinguishing between the traumatic and degenerative origins of chondral injuries<sup>19</sup>. Early degenerative changes may be shown by fibrillation and surface irregularity, changes in cartilage thickness and intrachondral alterations in signal intensity. Advanced changes are reflected in multiple areas of cartilage thinning of varying depth and size, edema, cysts and subchondral fibrosis or trabecular sclerosis. Associated degenerative changes are also present - osteophytes, joint effusion and synovitis.

Traumatic chondral lesions appear as solitary focal cartilage defects with acutely angled margins, often associated with bone bruising/edema or subchondral fracture<sup>19</sup>.

Chondral Injuries - distinguishing acute, post traumatic chondral injuries from chronic

Key reference - Hempfling<sup>20</sup>

Hempfling et al<sup>20</sup> have attempted to establish a basis for distinguishing acute chondral injuries from chronic (they deal with the topic in general but include the knee). They suggest that –

- Evidence of pre-existing OA should be sought.
- An osteochondral fracture as detected by x-ray or a fracture involving the articular surface is evidence of acute trauma.
- A subchondral bone bruise adjacent to a cartilage lesion supports a diagnosis of an acute chondral lesion.
- A traumatic cartilage lesion is indicated if in the acute phase (0 to 6 weeks after the accident) subchondral bone edema without noticeable cartilage damage is detected and at the follow-up examination (not earlier than 3 months after the initial examination) new cartilage damage develops below or in the vicinity of the edema. The diagnosis is confirmed when additional concomitant injuries are present, such as cruciate ligament and collateral ligament lesions or traumatic meniscal damage.
- Edema of traumatic origin usually diminishes over time and very rarely persists. In the great majority of cases it will clear within a 3 week to 2 year time frame<sup>54</sup>. The exceptions are pre-existing osteoarthritic cartilage damage, secondary development of chondral damage as a result of a trauma, or pre-existing osteochondral injuries at the time of the accident. These exceptions can make evaluation difficult<sup>20</sup>.
- Traumatic bone edema in younger patients at the time of diagnosis is usually not associated with macroscopically (MRI or arthroscopy) discernible cartilage damage.
- In acute cartilage lesions the bone edema may occasionally be missing but the traumatic chondral lesions can be readily distinguished from degenerative lesions (see Recht above).
- Pre-existing edema resulting from degenerative changes may occur in parallel with traumatic edema. They can usually be differentiated as follows:
  - Traumatic (acute) edema (bruising) usually shows a distinct distribution pattern<sup>55</sup> -
    - After transient patellar dislocation, bone contusions are found at the medial patellar edge and at the lateral femoral condyle in combination with a tear of the medial patellofemoral ligament<sup>20</sup>.
    - After ACL tear, the classic location is the anterior or middle aspect of the lateral femoral condyle and the posterolateral tibial plateau (see Geeslin for further references<sup>55</sup>. In one case series one quarter of subjects also had bone contusions at the medial femoral condyle and/or the medial aspect of the tibial plateau<sup>56</sup> (for further notes on bone contusion/edema see Part A of this report, page 21).
    - After MCL tear, bruising is typically found at the lateral compartment.

- After PCL tears, the bone bruising is typically dispersed about the knee.
- After posterior lateral corner injuries bone bruises are typically present medially, most commonly at the anteromedial femoral condyle<sup>55</sup>.
- In the acute stage, traumatic edema is rarely accompanied by adjacent chondropathy, while this is always present in MRI-detectable osteoarthritis.
- Edema associated with osteoarthritis is always subchondral and almost always shows concomitant chondropathy, which may be located superficially directly adjacent to the edema, but also intrachondrally. In addition, degenerative meniscal changes are frequently found in the same compartment.
- OA lesions show a distinct regional pattern on MRI usually affecting the weight-bearing regions. Non-traumatic cartilage damage usually affects primarily the medial femorotibial joint or the posterior aspect of the patella. In advanced stages, concomitant osseous changes such as osteophytes, subchondral sclerosis and bone attrition are observed.
- Subchondral bone marrow lesions in osteoarthritis tend to progress with time.
- Note that bone marrow lesions are strongly associated with knee pain<sup>57</sup> and the presence of OA – in one community-living cohort, 71% of those with radiographic OA had bone marrow lesions<sup>58</sup>.

### **Patellofemoral instability, subluxation and dislocation**

Key references - Monk<sup>26</sup> (has definitions of variables used to assess risk factors for patellar dislocation), Balcarek<sup>23</sup>, Diederichs<sup>59</sup> - a good general review.

#### **Epidemiology**

An American prospective cohort study<sup>60</sup> that followed patients from 2 to 5 years found that females in the 10 to 17 year age group were at highest risk for acute and recurrent patellar dislocation. In this age group, risk of first time dislocation was about 33% higher for girls compared to boys and risk of a recurrent episode was 3 times higher for girls than boys. In the 18 to 29 year age group, risk of recurrent dislocation was the same for men and women. Factors measured indicating increased risk of subsequent dislocation were prior history of subluxation/dislocation and young age at first injury.

#### **Clinical presentation**

Subjects with patellar instability sometimes report anterior knee pain, but episodes of collapsing or shifting in the knee is more commonly reported as 'giving way' or 'going out'<sup>21</sup>. Suggestions have been described for a physical examination to assess patellar stability<sup>21</sup>.

#### **Imaging**

Instability can be assessed by radiography and MRI. The patellar height can be assessed on a lateral radiograph. Indices used include the Blackburne-Peel index which relates the length of the articular surface of the patella to the distance of its inferior margin from the tibial plateau. It ranges from 0.85-1.09 in men and 0.79-1.09 in women.

The Insall-Salvati index is an alternative index which compares the diagonal length of the patella with the length of the patellar tendon; it ranges from 0.9–1.1 in men and 0.94–1.18 in women. This index has the limitation that the length of the patella is not always an indication of

the length of its articular surface<sup>21</sup>. It has been concluded that radiological measurements of patellar instability have low intra and inter-rater reliability<sup>22</sup>.

Rupture of the medial patellofemoral ligament is detectable by MRI<sup>21</sup>. Patellar dislocation may not be suspected in as many as 50% of subjects undergoing MR imaging<sup>59</sup>.

#### Imaging - TT-TG distance

A relatively large American study<sup>24</sup> has compared TT-TG distance in patients aged 5 – 42 years with lateral patellar instability to that in controls. The study reported that the mean TT-TG width was 14.6 +/- 4.6mm in patients compared to 10.6 +/- 4.0mm in controls, with 11% of patients having a TT-TG width >20mm (controls 2.2%) and 35% a distance >15mm (controls 10.3%). TT-TG distance was found to be independent of age.

Pandit et al<sup>25</sup> in a New Zealand based study have reported reference values for 100 males and females to be 9.1mm (95% CI 8.9-10.8) for males and 10.04mm (8.9-11.1) for females. They summarised their data by concluding that the reference value for TT-TG was 10 +/- 1mm.

In an English study<sup>26</sup> a cut-off of 14.5mm for TT-TG had a sensitivity of 80% and a specificity of 70% for detecting radiological instability.

It is doubtful whether ACC should accept any measurement of TT-TG without the radiologist providing reference data.

#### Acute patellar dislocation - MRI findings

An American study<sup>23</sup> has reviewed patterns of medial patellofemoral ligament (MPFL) injury in 73 patients (mean age 23) after acute lateral patellar dislocation. The MPFL was injured in 98.6% of patients, a finding supported by other reports (see<sup>23</sup>). A complete tear was found in 51.4% of cases, most often at the femoral attachment site, and a partial tear in 48.6%. Injury to the femoral origin, to the midsubstance and to the patellar insertion of the MPFL was found in 50.0%, 13.9% and 13.9% respectively. More than one site of injury was found in 22.2%. A close correlation between predisposing anatomical factors and MPFL injury pattern was found.

As reviewed in the American study<sup>23</sup>, the common MRI findings in subjects with lateral patellar dislocation were joint effusion, contusion of the lateral femoral condyle or medial patella, osteochondral fragments and injury to the medial stabilisers, the medial retinaculum and the MPFL. Differences in anatomy between these cases and the controls were as follows (all significant):

Anatomic measure	Controls	Cases
TT-TG (mm)	10.66	12.75
Mean index of patellar height (Insall-Salvati index)	1.04	1.21
Trochlear asymmetry (see paper for further details)	60.7	35.50

In another case series study it was found that acute lateral patellar dislocations were associated with the following -

- 83% of the cases had an injury to the medial patellar bone, 77% with bone contusions and 6% with discrete fractures.
- 59% had patellar chondral injuries.
- 95% had contusion at the anterolateral aspect of the lateral femoral condyle.
- 91% had an injury to the MPFL<sup>61</sup>.

#### Mechanism of injury

Movement to flexion, mostly from a straight start, occurred in 84% of cases in one series investigating mechanism of primary patellar dislocation. The authors concluded that spontaneous patellar relocation is common in skeletally immature girls and locked dislocation is common in skeletally mature men<sup>62</sup>. Diederichs<sup>59</sup> claims that first-time patellar dislocation most commonly occurs when the knee is flexed with internal rotation on a planted foot with a valgus component. A traumatic component of an external force may also contribute and result in disruption of the medial ligaments.

#### Variables associated with patellar dislocation

It has been observed that patellofemoral instability has a spectrum of severity ranging from mildly abnormal patellofemoral tracking to complete dislocation where no part of the patella remains in the trochlear groove<sup>26</sup>. Subluxation exists when the apex of the patella is not sliding centrally down the trochlear groove. Variables able to discriminate between grades of patellar subluxation (mild, moderate, severe) have been determined as –

- Patellar engagement (% overlap of patella in trochlear sulcus)
- TT-TG (distance between tibial tuberosity and apex of trochlear notch)
- VMOP (distance of vastus medialis obliquus from patella)
- Patella alta (patellar length /tendon length)
- Sulcus angle (proximal- bone, see paper for definition)
- Sulcus angle (proximal – cartilage, see paper for definition).

Of these six measures, patellar engagement had the strongest association with subluxation. No patient with an engagement of <30% appeared in the mild or no subluxation groups. The best cut-off point for predicting radiological instability was <=30%.

Patella alta had less discriminatory power than patellar engagement.

TT-TG increased from a mean of 12.0mm (SD, 4.1) for mild or no subluxation to a mean of 20.7mm (SD, 4.3) for severe subluxation. The researchers found the best cut-off point for TT-TG to be 14.5mm for detecting radiological instability.

VMOP ranged from 3.3mm (SD 2.9) for mild or no subluxation to 8.2mm (SD 2.8) for the severe group.

The risk factors found in American adults with lateral patellar dislocation are similar to those found in children<sup>63</sup>. MPFL injury was found in 90.2% of children and 100% of adults and patterns of injury were similar. Trochlear groove anatomy and magnitude of trochlear dysplasia did not differ between adult and child cases.

Risk factors for lateral patellar dislocation may differ between men and women with lateral patellar instability: Balcarek<sup>64</sup> has reported that trochlear dysplasia and the TT-TG distance is more pronounced in women.

### Patellar dislocation and OA

Even at a mean age of 26 years, a case series study has found that both first time and recurrent dislocators had cartilage lesions mostly on the central patella and early OA; cartilage defect size and prevalence of OA were correlated with the number of dislocations<sup>65</sup>.

### Trochlear dysplasia

Key reference - Bollier<sup>66</sup> - general review of trochlear dysplasia in relation to patellofemoral instability. A classification of dysplasia and diagnosis using lateral x-rays is presented in the paper.

Trochlear dysplasia is characterised by a shallow flattened trochlear groove with inadequate bony resistance to lateral patellar translation<sup>66</sup>. The bony restraint is particularly important after the first 20° of knee flexion; the soft tissues act as restraints at lesser degrees of flexion. According to Bollier<sup>66</sup> it is unclear if dysplasia is congenital, develops as a result of lateral tracking and chronic instability, or is caused by a combination of factors. It occurs in less than 2% of the population but is present in up to 85% of patients with recurrent patellar instability.

### Treatment note

A systematic review has cautiously concluded, with several caveats, that operative management of patellar dislocation is associated with significantly higher risk of patellofemoral OA but significantly lower risk of subsequent patellar dislocation compared to non-surgical management<sup>67</sup>.

### Subchondral cysts

Subchondral cysts contain small fragments of necrotic bone etc. and are surrounded by a layer of fibrous connective tissue without any epithelial component<sup>27</sup>. They may or may not communicate with the joint cavity. It has been suggested that the term cyst is inappropriate and a better term would be 'subchondral cyst-like bone marrow lesions'<sup>27</sup>.

They are thought to either arise from synovial fluid intrusion as a consequence of elevated intra-articular pressure or from traumatic bone necrosis after impact of two opposing articular surfaces.

They appear in regions without full-thickness cartilage defects in around 50% of cases and are strongly associated with bone marrow lesions in the same subregion<sup>18</sup>. One large cohort study of those at risk of developing OA or with OA (400 subjects, one knee per subject, 5600 subregions studied) has reported that cysts were found in 4.6% of the subregions. 91.2% of the cysts were associated with bone marrow lesions and located in the lesion or adjacent to it<sup>27</sup>. In addition, prevalent bone marrow lesions showed a strong and significant association with incident subchondral cysts in the same subregion of the knee<sup>28</sup>.

Crema<sup>28</sup> suggests that evidence for the traumatic hypothesis includes the fact that subchondral cysts commonly occur in the same subregion where a bone marrow lesion is found and that larger bone marrow lesions are associated with increased risk for the subsequent development of subchondral cysts. Further, loss of cartilage is not a necessary prerequisite for cyst



development, which is contrary to what would be expected if entry of synovial fluid into the bone was required.

In addition to having strong associations with bone marrow lesions, subchondral cysts are also -

- Commonly found in older subjects free of radiographic OA - in a Framingham study of community-living subjects free of radiographic evidence of OA (age range 51 to 89), the prevalence of subchondral cysts was 25% and prevalence increased with age<sup>7</sup>.
- Strongly associated with Kellgren Lawrence score and advanced OA<sup>29</sup>.

It is concluded that subchondral cysts have significant associations with bone marrow lesions, age and advancing OA.

### Arthroscopic treatment of OA

Key references – Felson (for summary of trials)<sup>68</sup>, Kirkley (for RCT evidence)<sup>69</sup> and the RCT by Katz et al (\* please see endnote on page 31)

Arthroscopic surgery as a treatment for OA is not usually an appropriate intervention for the older subject with meniscal tearing and significant OA (much of this section is abstracted from the 2010 report<sup>70</sup>).

Felson<sup>68</sup> has reviewed two RCTs that have examined outcomes from arthroscopy for knee OA. The first by Moseley<sup>71</sup> compared outcomes from three groups: lavage (lavage and removal of unstable tears); debridement (shaving rough articular cartilage, removing loose debris and trimming torn or degenerated meniscal fragments); and placebo (sham surgery). No efficacy of arthroscopic debridement or lavage over sham surgery was seen at two years after surgery.

Moseley's trial was strongly criticised and led to a second, directed by Kirkley<sup>69</sup>, which examined outcomes in a broader sample of subjects. Debridement with lavage (including synovectomy, excision of degenerative tears in the meniscus and fragments of cartilage, and excision of osteophytes) was compared with no surgery, with all patients receiving optimized additional therapy including physical therapy. There was no difference in outcomes with respect to pain or mechanical symptoms at one or two years.

Felson drew attention to an editorial by Marx<sup>72</sup>, a prominent American orthopaedic surgeon whose editorial accompanied Kirkley's paper: Marx gave two scenarios and suggested that arthroscopy would be indicated in a patient with mild or no osteoarthritis, who had an acute injury involving twisting of the knee, whose symptoms date clearly to that injury and where an MRI demonstrated a remediable lesion likely due to the injury. Marx's other case concerned an older lady with advanced OA and co-existent meniscal tears in which case he stated that arthroscopy would be of no value.

Howell<sup>17</sup> who also supported the views of Marx above writes that there are three indications which may justify arthroscopy in a knee with mild to severe OA –

- A complaint of mechanical symptoms from a loose body which resides anteriorly in the suprapatellar pouch on the radiograph (a posterior loose body does not cause locking<sup>17</sup>).
- The removal of a meniscal tear which is causing mechanical symptoms with pain localized on the joint line (but a tear is rarely the primary cause of pain in a knee with advanced OA<sup>17</sup>).

- Excision of an anterior anvil osteophyte to improve extension in the knee with mild OA and a flexion contracture.

Felson himself concluded that the failure of arthroscopy to be effective suggested that all the elements of that treatment were ineffective, including the surgical treatment of stable degenerative tears that are commonly present. He concluded that arthroscopy for OA has now been convincingly demonstrated to be ineffective and should not be carried out. The only exception would be where there was evidence of recent trauma and a symptomatic meniscal tear.

The AAOS in their guideline<sup>30</sup> recommend against performing arthroscopy with debridement or lavage in patients with a primary diagnosis of symptomatic OA of the knee. They also recommend that arthroscopic partial meniscectomy or loose body removal is an option in patients with symptomatic OA of the knee who also have primary signs and symptoms of a torn meniscus and/or a loose body.

Englund<sup>49</sup> believes that while persons with meniscal damage without surgery are at high risk of developing knee OA, there is no evidence that current surgical techniques reduce this risk. He stated that even in painful knees, surgery is a questionable alternative in those aged over 50.

Further a recent large multicentre American RCT has found that in those over 45 years of age with mild to moderate OA and evidence of a meniscal tear, surgery for the tear (not the OA as above) plus post operative physical therapy was of no greater benefit at 6 months and 12 months (pain and physical function) compared to physical therapy alone (\* please see endnote on page 31). Inclusion criteria did not require that meniscal tears were of non-traumatic origin: the symptoms had to be consistent with a torn meniscus and include at least one of the following: clicking, catching, popping, giving way, pain with pivot or torque, pain that was episodic, pain that was acute and localized to one joint line. Crossover from the physical treatment group was allowed during the trial and 30% of subjects in this group did crossover during the trial: the patients that did crossover did not appear to be disadvantaged with respect to outcome compared to those in the two main trial arms. These findings are similar to the results of Herrlin (see page 18) who compared outcomes in groups who had physical therapy or partial meniscectomy plus physical therapy for degenerative, non-traumatic meniscal tears.

There is therefore a significant body of evidence that in those of middle to older age with mild to moderate OA, surgery in the knee for relief of OA and/or repair or resection of partial meniscal tears of either traumatic or degenerative origin is in the great majority of cases no better than conservative treatment.

## References

1. Sanders TG, Medynski MA, Feller JF, Lawhorn KW. Bone contusion patterns of the knee at MR imaging: footprint of the mechanism of injury. *Radiographics : a review publication of the Radiological Society of North America, Inc* 2000(pp).
2. Hasegawa A, Otsuki S, Pauli C, Miyaki S, Patil S, Steklov N, et al. Anterior cruciate ligament changes in the human knee joint in aging and osteoarthritis. *Arthritis Rheum* 2012;64(3):696-704.
3. DeFranco MJ, Bach BR, Jr. A comprehensive review of partial anterior cruciate ligament tears. *J Bone Joint Surg Am* 2009;91(1):198-208.
4. De Smet AA. How I diagnose meniscal tears on knee MRI. *American Journal of Roentgenology* 2012;199(3):181-499.
5. Guenoun D, Le Corroller T, Amous Z, Pauly V, Sbihi A, Champsaur P. The contribution of MRI to the diagnosis of traumatic tears of the anterior cruciate ligament. *Diagn Interv Imaging* 2012;93(5):331-41.
6. Van Dyck P, De Smet E, Veryser J, Lambrecht V, Gielen JL, Vanhoenacker FM, et al. Partial tear of the anterior cruciate ligament of the knee: injury patterns on MR imaging. *Knee Surg Sports Traumatol Arthrosc* 2012;20(2):256-61.
7. Guermazi A, Niu J, Hayashi D, Roemer FW, Englund M, Neogi T, et al. Prevalence of abnormalities in knees detected by MRI in adults without knee osteoarthritis: Population based observational study (Framingham Osteoarthritis Study). *Bmj* 2012;345(7874):2012.
8. Hill CL, Seo GS, Gale D, Totterman S, Gale ME, Felson DT. Cruciate ligament integrity in osteoarthritis of the knee. *Arthritis Rheum* 2005;52(3):794-9.
9. ACC. MRI guidelines for the diagnosis of soft tissue knee injuries: Internal derangements. [http://www.acc.co.nz/PRD\\_EXT\\_CSMP/groups/external\\_communications/documents/gui/de/wpc087399.pdf](http://www.acc.co.nz/PRD_EXT_CSMP/groups/external_communications/documents/gui/de/wpc087399.pdf). 2010.
10. ACC. The diagnosis and management of soft tissue injuries: internal derangements. Best practice evidence based guideline. 2003.
11. Meuffels DE, Poldervaart MT, Diercks RL, Fievez AW, Patt TW, Hart CP, et al. Guideline on anterior cruciate ligament injury. *Acta Orthop* 2012;83(4):379-86.
12. Alatakis S, Naidoo P, Alatakis S, Naidoo P. MR imaging of meniscal and cartilage injuries of the knee. *Magn Reson Imaging Clin N Am* 2009;17(4):741-56.
13. Logerstedt DS, Snyder-Mackler L, Ritter RC, Axe MJ, Orthopedic Section of the American Physical Therapy A. Knee pain and mobility impairments: meniscal and articular cartilage lesions. *J Orthop Sports Phys Ther* 2010;40(6):A1-A35.
14. Subhas N, Sakamoto FA, Mariscalco MW, Polster JM, Obuchowski NA, Jones MH. Accuracy of MRI in the diagnosis of meniscal tears in older patients. *AJR Am J Roentgenol* 2012;198(6):W575-80.
15. Kocheta A, Toms A. Isolated chondral injuries of the knee (diagnosis and treatment). *Current Orthopaedics* 2004;18(2):154-63.
16. Hughes RJ, Houlihan-Burne DG. Clinical and MRI considerations in sports-related knee joint cartilage injury and cartilage repair. *Semin Musculoskelet Radiol* 2011;15(1):69-88.
17. Howell SM. The role of arthroscopy in treating osteoarthritis of the knee in the older patient. *Orthopedics* 2010;33(9):652.
18. Braun HJ, Gold GE. Diagnosis of osteoarthritis: imaging. *Bone* 2012;51(2):278-88.
19. Recht MP, Goodwin DW, Winalski CS, White LM. MRI of articular cartilage: revisiting current status and future directions. *AJR Am J Roentgenol* 2005;185(4):899-914.

20. Hempfling H, Bohndorf K, Roemer F. [Acute, traumatic versus chronic cartilage lesions as terms of a medical expert's opinion]. *Zeitschrift fur Orthopadie & Unfallchirurgie* 2008;146(3):381-91.
21. Redziniak DE, Diduch DR, Mihalko WM, Fulkerson JP, Novicoff WM, Sheibani-Rad S, et al. Patellar instability. *J Bone Joint Surg Am* 2009;91(9):2264-75.
22. Smith TO, Cogan A, Patel S, Shakokani M, Toms AP, Donell ST. The intra- and inter-rater reliability of X-ray radiological measurements for patellar instability. *Knee* 2013;20(2):133-8.
23. Balcarek P, Ammon J, Frosch S, Walde TA, Schuttrumpf JP, Ferlemann KG, et al. Magnetic resonance imaging characteristics of the medial patellofemoral ligament lesion in acute lateral patellar dislocations considering trochlear dysplasia, patella alta, and tibial tuberosity-trochlear groove distance. *Arthroscopy* 2010;26(7):926-35.
24. Balcarek P, Jung K, Frosch KH, Sturmer KM. Value of the tibial tuberosity-trochlear groove distance in patellar instability in the young athlete. *Am J Sports Med* 2011;39(8):1756-61.
25. Pandit S, Frampton C, Stoddart J, Lynskey T. Magnetic resonance imaging assessment of tibial tuberosity-trochlear groove distance: Normal values for males and females. *International Orthopaedics* 2011;35 (12):1799-803.
26. Monk AP, Doll HA, Gibbons CL, Ostlere S, Beard DJ, Gill HL, et al. The patho-anatomy of patellofemoral subluxation. *J Bone Joint Surg Br* 2011;93(10):1341-7.
27. Crema MD, Roemer FW, Marra MD, Niu J, Lynch JA, Felson DT, et al. Contrast-enhanced MRI of subchondral cysts in patients with or at risk for knee osteoarthritis: the MOST study. *Eur J Radiol* 2010;75(1):e92-6.
28. Crema MD, Roemer FW, Zhu Y, Marra MD, Niu J, Zhang Y, et al. Subchondral cystlike lesions develop longitudinally in areas of bone marrow edema-like lesions in patients with or at risk for knee osteoarthritis: detection with MR imaging--the MOST study. *Radiology* 2010;256(3):855-62.
29. Link TM, Steinbach LS, Ghosh S, Ries M, Lu Y, Lane N, et al. Osteoarthritis: MR imaging findings in different stages of disease and correlation with clinical findings. *Radiology* 2003;226(2):373-81.
30. AAOS. American Academy of Orthopaedic Surgeons treatment of osteoarthritis of the knee (non-arthroplasty). <http://www.aaos.org/Research/guidelines/GuidelineOAKnee.asp> Accessed 30 Jan 2013. 2008.
31. Boden BP, Sheehan FT, Torg JS, Hewett TE. Noncontact anterior cruciate ligament injuries: Mechanisms and risk factors. *J Am Acad Orthop Surg* 2010;18 (9):520-27.
32. Dai B, Herman D, Liu H, Garrett WE, Yu B. Prevention of ACL injury, part I: injury characteristics, risk factors, and loading mechanism. *Res Sports Med* 2012;20(3-4):180-97.
33. Micheo W, Hernandez L, Seda C. Evaluation, management, rehabilitation, and prevention of anterior cruciate ligament injury: current concepts. *Physical Medicine and Rehabilitation* 2010;2(10):935-44.
34. Motohashi M. Profile of bilateral anterior cruciate ligament injuries: a retrospective follow-up study. *Journal of Orthopaedic Surgery* 2004;12(2):210-5.
35. Smith HC, Vacek P, Johnson RJ, Slauterbeck JR, Hashemi J, Shultz S, et al. Risk factors for anterior cruciate ligament injury: a review of the literature - part 1: neuromuscular and anatomic risk. *Sports health* 2012;4(1):69-78.
36. Smith HC, Vacek P, Johnson RJ, Slauterbeck JR, Hashemi J, Shultz S, et al. Risk factors for anterior cruciate ligament injury: a review of the literature-part 2: hormonal, genetic, cognitive function, previous injury, and extrinsic risk factors. *Sports health* 2012;4(2):155-61.

37. Hashemi J, Mansouri H, Chandrashekar N, Slauterbeck JR, Hardy DM, Beynnon BD. Age, sex, body anthropometry, and ACL size predict the structural properties of the human anterior cruciate ligament. *J Orthop Res* 2011;29(7):993-1001.
38. Lintz F, Pujol N, Boisrenoult P, Bargoin K, Beaufile P, Dejour D. Anterior cruciate ligament mucoid degeneration: a review of the literature and management guidelines. *Knee Surg Sports Traumatol Arthrosc* 2011;19(8):1326-33.
39. Frobell RB, Roos HP, Roos EM, Roemer FW, Ranstam J, Lohmander LS. Treatment for acute anterior cruciate ligament tear: five year outcome of randomised trial. *Bmj* 2013;346:f232.
40. Frobell RB, Roos EM, Roos HP, Ranstam J, Lohmander LS. A randomized trial of treatment for acute anterior cruciate ligament tears. [Erratum appears in *N Engl J Med*. 2010 Aug 26;363(9):893]. *N Engl J Med* 2010;363(4):331-42.
41. Donohoe M, Aslanian H, Solomon K. Types of knee injuries & how they occur. *Forensic Examiner* 2005;14(1).
42. Drosos GI, Pozo JL. The causes and mechanisms of meniscal injuries in the sporting and non-sporting environment in an unselected population. *Knee* 2004;11(2):143-9.
43. Ercin E, Kaya I, Sungur I, Demirbas E, Ugras AA, Cetinus EM. History, clinical findings, magnetic resonance imaging, and arthroscopic correlation in meniscal lesions. *Knee Surg Sports Traumatol Arthrosc* 2012;20(5):851-6.
44. Oei EH, Ginai AZ, Hunink MG. MRI for traumatic knee injury: a review. *Semin Ultrasound CT MR* 2007;28(2):141-57.
45. Bhattacharyya T, Gale D, Dewire P, Totterman S, Gale ME, McLaughlin S, et al. The clinical importance of meniscal tears demonstrated by magnetic resonance imaging in osteoarthritis of the knee. *J Bone Joint Surg Am* 2003;85-A(1):4-9.
46. Kemp MA, Lang K, Dahill M, Williams JL. Investigating meniscal symptoms in patients with knee osteoarthritis-Is MRI an unnecessary investigation? *Knee* 2011;18(4):252-53.
47. Herrlin S, Hallander M, Wange P, Weidenhielm L, Werner S. Arthroscopic or conservative treatment of degenerative medial meniscal tears: a prospective randomised trial. *Knee Surg Sports Traumatol Arthrosc* 2007;15(4):393-401.
48. Herrlin SV, Wange PO, Lapidus G, Hallander M, Werner S, Weidenhielm L. Is arthroscopic surgery beneficial in treating non-traumatic, degenerative medial meniscal tears? A five year follow-up. *Knee Surg Sports Traumatol Arthrosc* 2013;21(2):358-64.
49. Englund M. Meniscal tear -- a common finding with often troublesome consequences. *J Rheumatol* 2009;36(7):1362-4.
50. Roemer FW, Crema MD, Trattnig S, Guermazi A, Roemer FW, Crema MD, et al. Advances in imaging of osteoarthritis and cartilage. *Radiology* 2011;260(2):332-54.
51. Cibere J, Sayre EC, Guermazi A, Nicolaou S, Kopeck JA, Esdaile JM, et al. Natural history of cartilage damage and osteoarthritis progression on magnetic resonance imaging in a population-based cohort with knee pain. *Osteoarthritis Cartilage* 2011;19(6):683-8.
52. Schiphof D, Boers M, Bierma-Zeinstra SM. Differences in descriptions of Kellgren and Lawrence grades of knee osteoarthritis. *Ann Rheum Dis* 2008;67(7):1034-6.
53. Brittberg M, Winalski CS. Evaluation of cartilage injuries and repair. *J Bone Joint Surg Am* 2003;85-A Suppl 2:58-69.
54. Mandalia V, Henson JH. Traumatic bone bruising--a review article. *Eur J Radiol* 2008;67(1):54-61.
55. Geeslin AG, LaPrade RF. Location of bone bruises and other osseous injuries associated with acute grade III isolated and combined posterolateral knee injuries. *Am J Sports Med* 2010;38(12):2502-8.

56. Yoon KH, Yoo JH, Kim KI. Bone contusion and associated meniscal and medial collateral ligament injury in patients with anterior cruciate ligament rupture. *J Bone Joint Surg Am* 2011;93(16):1510-8.
57. Zhang Y, Nevitt M, Niu J, Lewis C, Torner J, Guermazi A, et al. Fluctuation of knee pain and changes in bone marrow lesions, effusions, and synovitis on magnetic resonance imaging. *Arthritis Rheum* 2011;63(3):691-9.
58. Ip S, Sayre EC, Guermazi A, Nicolaou S, Wong H, Thorne A, et al. Frequency of bone marrow lesions and association with pain severity: results from a population-based symptomatic knee cohort. *J Rheumatol* 2011;38(6):1079-85.
59. Diederichs G, Issever AS, Scheffler S. MR imaging of patellar instability: Injury patterns and assessment of risk factors. *Radiographics* 2010;30(4):961-81.
60. Fithian DC, Paxton EW, Stone ML, Silva P, Davis DK, Elias DA, et al. Epidemiology and natural history of acute patellar dislocation. *Am J Sports Med* 2004;32(5):1114-21.
61. Menon S, Rapaport H, Al-Tawil O, Math K, Scuderi G, Katz D. Acute lateral patellar dislocation: Incidence and patterns of osseous, cartilaginous, and medial patellofemoral ligament injury in 150 patients on MRI. *Emergency Radiology Conference: ASER* 2011;18(6).
62. Nikku R, Nietosvaara Y, Aalto K, Kallio PE. The mechanism of primary patellar dislocation: trauma history of 126 patients. *Acta Orthop* 2009;80(4):432-4.
63. Balcarek P, Walde TA, Frosch S, Schuttrumpf JP, Wachowski MM, Sturmer KM, et al. Patellar dislocations in children, adolescents and adults: a comparative MRI study of medial patellofemoral ligament injury patterns and trochlear groove anatomy. *Eur J Radiol* 2011;79(3):415-20.
64. Balcarek P, Jung K, Ammon J, Walde TA, Frosch S, Schuttrumpf JP, et al. Anatomy of lateral patellar instability: trochlear dysplasia and tibial tubercle-trochlear groove distance is more pronounced in women who dislocate the patella. *Am J Sports Med* 2010;38(11):2320-7.
65. Vollnberg B, Koehlit T, Jung T, Scheffler S, Hoburg A, Khandker D, et al. Prevalence of cartilage lesions and early osteoarthritis in patients with patellar dislocation. *European radiology* 2012;22(11):2347-56.
66. Bollier M, Fulkerson JP. The role of trochlear dysplasia in patellofemoral instability. *J Am Acad Orthop Surg* 2011;19(1):8-16.
67. Smith TO, Song F, Donell ST, Hing CB. Operative versus non-operative management of patellar dislocation. A meta-analysis. *Knee Surg Sports Traumatol Arthrosc* 2011;19(6):988-98.
68. Felson DT, Felson DT. Arthroscopy as a treatment for knee osteoarthritis. *Baillieres Best Pract Res Clin Rheumatol* 2010;24(1):47-50.
69. Kirkley A, Birmingham TB, Litchfield RB, Giffin JR, Willits KR, Wong CJ, et al. A randomized trial of arthroscopic surgery for osteoarthritis of the knee. [Erratum appears in *N Engl J Med*. 2009 Nov 12;361(20):2004]. *N Engl J Med* 2008;359(11):1097-107.
70. Larking P. Causation Review - Meniscal tears. ACC Report. 2010.
71. Moseley JB, O'Malley K, Petersen NJ, Menke TJ, Brody BA, Kuykendall DH, et al. A controlled trial of arthroscopic surgery for osteoarthritis of the knee. [Summary for patients in *J Fam Pract*. 2002 Oct;51(10):813; PMID: 12401143]. *N Engl J Med* 2002;347(2):81-8.
72. Marx RG. Arthroscopic surgery for osteoarthritis of the knee?. [Erratum appears in *N Engl J Med*. 2008 Oct 9;359(15):1637]. *N Engl J Med* 2008;359(11):1169-70.

---

\* The papers by Buchbinder (2013) and Katz et al (2013) were obtained too late to be included in the list of references. The details of these papers are as follows:

Buchbinder, R. (2013). "Meniscectomy in Patients with Knee Osteoarthritis and a Meniscal Tear?" New England Journal of Medicine **368**(18): 1740-1741.

Katz, J. N., R. H. Brophy, et al. (2013). "Surgery versus Physical Therapy for a Meniscal Tear and Osteoarthritis." New England Journal of Medicine **368**(18): 1675-1684.