

KINEMATICS AND DEGENERATIVE CHANGE IN LIGAMENT-INJURED KNEES

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ABSTRACT

The aim of the work presented in this thesis was to examine the associations between the kinematics of the knee characterised by the tibiofemoral contact pattern, and degenerative change, in the context of anterior cruciate ligament (ACL) injury. While the natural history of degenerative change following knee injury is well understood, the role of kinematics in these changes is unclear. Kinematics of the knee has been described in a variety of ways, most commonly by describing motion according to the six degrees of freedom of the knee. The advantage of mapping the tibiofemoral contact pattern is that it describes events at the articular surface, important to degenerative change. It was hypothesised that the tibiofemoral contact pattern would be affected by injury to the knee. A model of ACL injury was chosen because the kinematics of the knee have been shown to be affected by ACL injury, and because the majority of chronic ACL-deficient knees develop osteoarthritis, the associations between kinematics and degenerative change could be explored.

A technique of tibiofemoral contact pattern mapping was established using MRI, as a quantifiable measure of knee kinematics. The tibiofemoral contact pattern was recorded from 0° to 90° knee flexion while subjects performed a leg-press against a 150N load, using sagittal magnetic resonance imaging (MRI) scans. The technique was tested and found to be reliable, allowing a description of the tibiofemoral contact pattern in 12 healthy subjects.

The tibiofemoral contact patterns of knee pathology were then examined in a series of studies of subjects at a variety of stages of chronicity of ligament injury and osteoarthritis. Twenty subjects with recent ACL injury, 23 subjects with chronic ACL deficiency of at

least 10 years standing, and 14 subjects with established osteoarthritis of the knee were recruited. The 20 subjects with recent ACL injury were examined again at 12 weeks and 2 years following knee reconstruction.

The tibiofemoral contact patterns were examined for each group of subjects and the associations between changes in the contact patterns and evidence of joint damage explored. Evidence of joint damage and severity of osteoarthritis were recorded from x-rays, diagnostic MRI, operation reports and bone densitometry at the tibial and femoral condyles of the knee.

Each of the three groups with knee pathology exhibited different characteristics in the tibiofemoral contact pattern, and these differences were associated with severity of joint damage and osteoarthritis. The recently ACL-injured knees demonstrated a tibiofemoral contact pattern that was posterior on the tibial plateau, particularly in the lateral compartment. Those with chronic ACL deficiency demonstrated differences in the contact pattern in the medial compartment, associated with severity of damage to the knee joint. Osteoarthritic knees showed reduced femoral roll back and longitudinal rotation that normally occur during knee flexion. Two years following knee reconstruction there was no difference between the contact pattern of the reconstructed and healthy contralateral knees.

This technique of tibiofemoral contact pattern mapping is sensitive to the abnormal characteristics of kinematics in ligament injury and osteoarthritis. This is the first time the tibiofemoral contact characteristics of chronic ACL-deficient and osteoarthritis knees have been described and links examined between tibiofemoral contact patterns and degenerative change.

PUBLICATIONS

The work arising from these PhD studies has resulted in the following refereed publications and manuscripts. Each was based on a separate chapter from the thesis.

1. Smith PN, Refshauge KM, Scarvell JM (2003): Development of concepts of kinematics of the normal knee. *Archives of Physical Medicine and Rehabilitation* 84 (12): 1895-1902
2. Scarvell JM, Smith PN, Refshauge KM, Galloway HR, Woods KR (2003): Evaluation of a method to map the tibiofemoral contact points in the healthy knee. *Journal of Orthopaedic Research* (in press).
3. Scarvell JM, Smith PN, Refshauge KM, Galloway HR, Woods KR (2003): Comparison of two methods to analyse knee kinematics using MRI: tibiofemoral contact mapping, and mapping the position of the femoral condylar centre. *Journal of Orthopaedic Research* 2003 (in press).
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Scarvell JM, Smith PN, Galloway H, Refshauge KM and Woods K (2002): Kinematics and bone density changes in the ACL-injured knee. In: *Proceedings of the Vllth International Physiotherapy Congress of the Australian Physiotherapy Association*, Sydney

Scarvell JM, Smith PN, Galloway H, Refshauge KM and Woods K (2002): Critical bone mineral density loss in the anterior cruciate ligament injured knee. In: *Proceedings of the 62nd Australian Orthopaedic Association Annual Scientific Meeting*, Melbourne.

Scarvell JM, Smith PN, Galloway H, Refshauge KM and Woods K (2002): MRI analysis

of kinematics of the ACL-injured knee. In: *Proceedings of the 62nd Australian Orthopaedic Association Annual Scientific Meeting*, Melbourne.

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Scarvell JM, Smith PN, Galloway H, Refshauge KM and Woods K (2004): Kinematic consequences of chronic ACL injury. In: *Proceedings of the American Academy of Orthopaedic Surgeons Annual Meeting*, San Francisco.

Smith PN, Scarvell JM, Galloway H, Refshauge KM and Woods K (2004): MRI analysis of kinematics of the ACL-injured knee. In: *Proceedings of the American Academy of Orthopaedic Surgeons Annual Meeting*, San Francisco.

LIST OF ABBREVIATIONS

ACL	Anterior cruciate ligament
PCL	Posterior cruciate ligament
MCL	Medial collateral ligament
LCL	Lateral collateral ligament
MRI	Magnetic Resonance Imaging
RSA	Roentgen Stereophotogrammetric Analysis
EMG	Electromyography
BMD	Bone mineral density
DEXA	Dual energy x-ray absorptiometry
CT	computerized tomography
TF	tibiofemoral
FFC	flexion facet centre
FE	Flexion/extension
ANOVA	Analysis of variance
SE	Standard error
SD	Standard deviation
ICC	Intraclass Correlation

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CHAPTER 1

KNEE KINEMATICS AND DEGENERATIVE CHANGE

Relationship between ACL injury and osteoarthritis of the knee

Epidemiology of ACL injury

Development of osteoarthritis in ACL-injured knees

Epidemiology and pathology of osteoarthritis in the knee

Characteristics of osteoarthritis in the knee

Changes to articular cartilage

Changes to bony structures

Changes to periarticular soft tissues

Significance of the thesis

Aim of the thesis

KNEE KINEMATICS AND DEGENERATIVE CHANGE

The relationship between kinematics and degenerative change in the knee is not well understood, despite general recognition that injuries that alter the kinematic pattern are likely to result in degeneration (Frankel et al. 1971). Tear of the anterior cruciate ligament (ACL) is one such injury. Instability caused by the ligament injury permits greater anterior tibial translation and knee internal rotation during daily activities than healthy knees (Karrholm et al. 1988; Beard et al. 1996; Vergis et al. 1998; Brandsson et al. 2001). People with chronic ACL deficiency are known to have a 50-90% incidence of late degeneration in the injured knee (Clatworthy et al. 1999; Gillquist et al. 1999). While several studies have examined the kinematics of ACL-injured knees (Vergis et al. 1997; Brandsson et al. 2001; Georgoulis et al. 2003), none has linked the aberrant kinematics to degeneration.

Animal models have been used to study the process of degeneration resulting from ACL injury, as these studies are not possible in humans (Marshall et al. 1971; Lukoschek et al. 1988; Lopez et al. 2003a; Lopez et al. 2003b). Indeed, ACL sectioning has been used in experimental designs to produce an animal model for the development of osteoarthritis (Hulth et al. 1970; Marshall and Olsson 1971). Meniscectomy and impact trauma to the articular cartilage also provide models for the development of osteoarthritis (Moskowitz et al. 1987; Calvo et al. 1999; Messner et al. 2000; Calvo et al. 2001), but it is the kinematic pattern in ACL deficiency, rather than the trauma to the articular cartilage, that makes the ACL-section model relevant to this study. ACL-sectioned knees of dogs, rabbits and sheep produce a characteristic pattern of osteophytosis. This osteophytosis precedes the articular cartilage

degeneration, chronic meniscal damage and flattening of the femoral condyles and tibial plateau which signify osteoarthritic change (Marshall and Olsson 1971; Lukoschek et al. 1988; Lopez et al. 2003a; Lopez and Markel 2003b). These changes are seen on radiographs of human ACL-deficient knees occurring in the same manner (Kannus et al. 1989; Dejour et al. 1994; Gillquist and Messner 1999). Unusual shear stresses at the osteochondral junction stimulate the development of osteophytes early in ACL deficiency (Moskowitz and Goldberg 1987). These osteophytes indicate the role of aberrant knee kinematics in the process of degenerative change.

While ACL sectioning has been used as a model for degenerative change, the role of altered kinematics in degenerative change has not been examined in animal studies. Studies in dogs have shown the process of degeneration occurring in the ACL-sectioned knee over time (Marshall and Olsson 1971; Lopez and Markel 2003b). Studies have also looked at the laxity of knees of ACL-sectioned dogs and the ground reaction forces produced by the dogs running (Lopez et al. 2003a), but the links between knee kinematic changes over time and degeneration have not been examined.

In humans, kinematics of acute ACL-injured knees have been studied in some detail, but not chronic ACL-deficient knees (Jonsson et al. 1989; Yack et al. 1994; Beard et al. 1996; Dennis et al. 1996; Vergis and Gillquist 1998; Brandsson et al. 2001). The kinematics of chronic ACL-deficient knees are not necessarily the same as acutely injured knees, because of adaptation of tissues under altered loads, degenerative wear of soft tissues and an altered neuromuscular environment (Steele et al. 1995; Wexler et al. 1998; Ferber et al. 2002). To study the relationship between kinematics of chronic ACL-deficient knees and degenerative

change, the kinematics of chronic ACL-deficient knees need to be accurately mapped and the differences in kinematic characteristics compared to the pattern of degeneration.

The end stage of the degenerative process is knee osteoarthritis (Buckwalter et al. 1997), which is a major problem in our aging community (Segal et al. 2002), yet there are few studies of the kinematics in osteoarthritic knees (Nagao et al. 1998). Gait analysis, using multiplanar video recordings with skin markers has been used to calculate kinetic forces and kinematics (Kaufman et al. 2001; Al-Zahrani et al. 2002; Gok et al. 2002). Assessment of knee kinematics by gait analysis has mainly been limited to sagittal plane motion (Kaufman et al. 2001) though frontal plane rotations have been reported (Hurwitz et al. 1998). Coronal plane rotation during active knee extension was shown to be reduced in osteoarthritic knees in a study using ultrasound (Nagao et al. 1998). There are still many unanswered questions regarding knee kinematics in knee osteoarthritis, such as the role of ligament injury and attrition.

Movement is comprised of kinematics and kinetics. The term kinematics is used to describe the three rotational motions of the tibia with respect to the femur (flexion-extension, adduction-abduction, internal-external rotation) and the three translational motions of the tibia with respect to the femur without reference to the forces or moments that cause the motion. Kinetics is used to describe the joint forces and moments associated with motion (Papadonikolakis et al. 2003). It is kinematics that has been studied in this thesis.

Methods used to examine kinematics of ACL-injured knees have included techniques which enable visualisation of the bony anatomy such as roentgen stereophotogrammetric analysis

(RSA) (Karrholm et al. 1988; Brandsson et al. 2001) and magnetic resonance imaging (MRI), indirect measurement of bony anatomy such as video analysis of jumping in subjects with implanted intracortical pins (Ramsey et al. 2001) and electrogoniometry (Vergis et al. 1997; Kvist et al. 2001; Hollman et al. 2002; Georgoulis et al. 2003); and measurement from skin markers such as 3D video analysis (Hollman et al. 2002; Georgoulis et al. 2003). MRI has the advantages of direct measurement of bony structures, including accuracy and direct visualisation of the tibiofemoral contact areas of the knee, without the radiation dosage or complications of surgical procedures associated with RSA or intracortical pins (Thompson et al. 1991; Todo et al. 1999). In an open-field MRI machine it is possible to perform everyday activities, such as a squat, step up or lunge (Vedi et al. 1999; Hill et al. 2000; Nakagawa et al. 2000). In a closed field MRI these activities can be simulated in supine, by controlling the knee position in a loaded situation (Smith et al. 1999). Currently MRI gives the best image resolution for accurate visualisation of knee anatomy when performed as a series of still images, at intervals through range of motion. Cine MRI is a technology still under development (Niitsu et al. 1990; Niitsu 2001). For the assessment of knee kinematics in vivo, current MRI technology provides the best direct visualisation method, with least risk to the patient.

This thesis has investigated the ACL-deficient human model, both recently injured knees, chronic ACL-deficient knees and knees with established osteoarthritis in order to explore the relationship between movement at the tibiofemoral interface and degenerative change.

Relationship between ACL injury and osteoarthritis of the knee

Epidemiology of ACL injury

Injuries to the ACL are common among active people. In the United States of America the incidence of ACL injury is estimated at 30 per 100 000 population per year (Miyasaka et al. 1991). This represents 80 000 ACL injuries annually in the USA (Griffin et al. 2000). ACL injuries account for 20% of knee injuries presenting at general hospitals (Daniel et al. 1994) and 60% of those occurring on the ski fields (Feagin et al. 1987). In Scandinavia the incidence is similar (Buhl-Nielsen 1991).

Data about incidence of ACL injuries in Australia is not available, but it is expected to be similar to the USA. In Australia the incidence of ACL injuries in netball is approximately 4.7 per week, from among 350 000 registered players (Otago et al. 1999) and 950 per year in professional Australian Football (Orchard et al. 2002), though this figure does not include amateur and junior grade players. In skiing the incidence of knee injuries is reported as increasing, despite improvements in binding releases (Sherry et al. 1991). Australian hospital data do not differentiate between ACL injuries and other sports injuries. In the year 1998-9 there were 11,422 people admitted after injuries on playing-fields and 39,966 people admitted for injuries to the knee and lower leg (Australian Institute of Health and Welfare 1999). These data include people admitted with ACL injuries. However, many people with ACL injuries

not admitted to hospital. Therefore, it is not possible to obtain Australian injury statistics from current methods of data collection.

Demographically, knee ligament injuries are most common in individuals between 15 and 25 years of age (Hirshman et al. 1990; Griffin et al. 2000), with injuries in males predominating due to levels of sports participation (Miyasaka et al. 1991). However, there is a reported 4 to 6 times higher risk of ACL injury in those women participating in sport (Roos 1994; Arendt 1997; Gwinn et al. 2000; Huston et al. 2000; Kirkendall et al. 2000), thought to be due to a combination of neuromuscular, hormonal and anatomical factors (Arendt 2001). While sports injuries may account for 74% of ACL injuries, not all ACL injuries occur during sport (Daniel et al. 1994). ACL injuries also occur from falls, during social activities and to pedestrians in vehicular accidents.

Non-contact injuries account for 70% of all ACL injuries (Griffin et al. 2000) occurring in jumping, landing or side-stepping, but the proportion of non-contact injuries may be higher for women (Arendt 1997; Kirkendall and Garrett 2000). The most common mechanism for non-contact injury has been described as a rapid deceleration with the knee close to full extension, associated with a change of direction or rotation factor (Boden et al. 2000). A powerful eccentric quadriceps force is considered to be a major contributor to injury, generating a large anterior tibial shear force. Non-contact ACL injuries are commonly a discrete ACL injury, without damage to other knee structures (Griffin et al. 2000).

Contact injuries are more likely to result in damage to other knee structures as well as the ACL. Contact injuries are most commonly a valgus blow, either from another player, a fall

across an obstacle or occasionally a pedestrian accident. Contact injuries are frequently associated with injury to several structures in the knee, such as the medial collateral ligament (MCL) and menisci, as the direction of the blow and loading through the knee are likely to load to failure both primary and secondary restraints in the valgus direction. O'Donoghue's "unhappy triad" is one description of this pattern of injury, consisting of damage to the ACL, MCL and medial meniscus from a valgus blow to the knee (O'Donoghue 1959). Frequently, the meniscus involved is the lateral meniscus, due to the joint compression and rotation loads involved in the injury forcing the trapped lateral meniscus beyond its available mobility (O'Donoghue 1959; Shelbourne et al. 1991). Evidence of the large joint compression forces in a contact injury may also be seen in the bone bruises found in subchondral bone, particularly of the lateral compartment, visible on MRI (Johnson et al. 1998; Lahm et al. 1998; Johnson et al. 2000). These bone bruises are visible indications of articular cartilage and subchondral bone trauma, even fracture (Johnson et al. 1998). Thus, contact injuries may result in more damage to the knee than the isolated ACL tear of a non-contact injury.

The risk of osteoarthritis is higher after sustaining a contact injury than a non-contact injury to the knee, because of damage to other structures in addition to the ACL. The risk of osteoarthritis in the knee joint following a non-contact, discrete ACL injury may be as low as 15 - 20% (McDaniel et al. 1983; Gillquist and Messner 1999). However, if there is meniscal injury associated with ACL deficiency, the risk of osteoarthritis rises to 65% - 70% (Kannus and Jarvinen 1989; Neyret et al. 1992). Meniscal damage is a common late sequela of ACL deficiency (Myers et al. 2001). There are few studies that report the original mechanism of injury, as opposed to the presence of late meniscal damage, as a risk factor for osteoarthritis in the injured knee. Where a contact injury has also resulted in bone bruising or trauma, the risk

of late osteoarthritis is further increased (Stein et al. 1995). In a study of 141 acute ACL injuries 48 were found to have osseous lesions on MRI, of which 26 were bone bruises, 11 were subchondral fractures and 7 were osteochondral fractures (Lahm et al. 1998). Fifty percent of these osseous lesions can be expected to develop articular cartilage lesions that do not recover (Stein and Fischer 1995). Thus, a discreet ACL injury is a known risk factor for the development of late osteoarthritis, which is increased by other associated damage to the knee.

Development of osteoarthritis in ACL-injured knees

Research into osteoarthritis has depended on animal models. The models that have been used because they consistently develop osteoarthritis are application of blunt impact trauma to the articular cartilage (Oegema et al. 1993; Newberry et al. 1998), meniscectomy (Messner et al. 2000; Calvo et al. 2001) and ACL sectioning of animals (Marshall and Olsson 1971). The ACL-sectioned model has been a reliable model, in that a high percentage of animals develop osteoarthritis (Marshall and Olsson 1971; Bailey et al. 1997; Lopez et al. 2003a). Furthermore, it is less traumatic to experimental animals to perform a discrete ACL section via arthroscopy or radio frequency burn, than the more aggressive meniscectomy or impact trauma (Lopez et al. 2003a). Marshall et al (1971) described in detail the structural changes to the bone, cartilage and soft tissues of dogs' knees following ACL transection via arthrotomy. All 12 dogs studied showed proliferative osteophytes, thickened joint capsule and early cartilage fibrillation (Marshall and Olsson 1971). Lopez et al (2003) similarly found early radiographic signs of osteoarthritis in all the ACL-sectioned dogs with characteristic osteophyte development, subchondral sclerosis and cartilage thinning at 34 weeks.

Differences in kinematics between humans and dogs limit the extrapolation of results from animal studies to humans. Not all ACL-injured people develop osteoarthritis. It is important to look for the relationship between kinematics and degenerative changes in humans.

Currently there are three contributing factors considered responsible for the development of osteoarthritis in ACL-injured knees: damage to joint structures at the time of ACL injury (Lundberg et al. 1997); chronic instability resulting in damage to the menisci and reducing their effectiveness as load sharing structures (Shoemaker et al. 1986; Chorev et al. 2000); and altered tibiofemoral contact patterns changing the contact footprint of the tibia, as well as changing the roll/glide characteristics of motion and shear forces at the tibiofemoral interface (Brandsson et al. 2001).

Firstly, damage to the joint structures at the time of the original injury has long-term consequences. Haemarthrosis and trauma to articular cartilage and menisci produce biochemical changes within the joint, which accelerate degenerative change to the articular cartilage and failure of repair of the proteoglycan matrix (Lundberg et al. 1997; Clatworthy and Amendola 1999). Many ACL injuries, particularly contact injuries, occur with an associated impact trauma to the articular cartilage and subchondral bone. Impact trauma may fail to recover, leading to necrosis of subchondral bone (Stein and Fischer 1995; Lahm et al. 1998). Impact trauma commonly occurs in the posterolateral tibial corner and lateral femoral condyle, due to the mechanism of the original injury. However, articular cartilage degeneration in ACL-deficient knees frequently occurs in the medial tibial compartment, suggesting that impact trauma is not responsible for medial compartment osteoarthritis (Dejour et al. 1994).

Secondly, the instability produced by the loss of the ACL alters the biomechanics of the injured knee and loads previously resisted by the ACL are transferred to the menisci. In the absence of the ACL the secondary restraints to anterior tibial translation are the medial meniscus and medial collateral ligament (Butler et al. 1980). Meniscal damage secondary to cruciate injury is common because while restraining sagittal displacement the menisci are compressed and ground between the condyles (Sherman et al.1988; Gillquist et al.1999; Allen et al. 2000). The damaged menisci are then ineffective in redistribution of weight-bearing forces through the knee. The weight-bearing area of the knee is consequently decreased and the pressure magnified. Weight is also taken in a more posterior position on the tibial plateau (Kurosawa et al. 1980; Arnoczky 1994). The consequences of meniscal insufficiency in the knee are well documented: osteophytosis, flattening of the condyles and articular cartilage degeneration (Fairbank 1948). Meniscal damage and meniscectomy secondary to instability in chronic ACL-deficient knees are known risk factors for the development of osteoarthritis (Sherman et al. 1988; Shirakura et al. 1995a) (Figure 1.1).



Figure 1.1: The knee of a 26-year-old female 10 years after an ACL injury. It demonstrates lateral meniscus maceration (black arrow), full thickness cartilage damage to the lateral tibial and femoral surfaces (white arrows) and extensive osteophytes (triangles).

Finally, the contact pattern of the femoral condyles on the tibial plateau is altered when the restraining influence of the ACL is removed (Dennis et al. 1996; Smith et al. 1999). The area of the tibiofemoral contact footprint and the roll/glide characteristics at the tibiofemoral interface may be implicated in the development of osteoarthritis in ACL-injured knees. Chondrocytes may be able to sense deformations in articular cartilage during stress loading (Mow et al. 2000) and respond quickly to repair minor damage to the cartilage matrix. However, early in knee instability chondrocyte swelling occurs, with a resultant increase in water content of the cartilage matrix. Micro-trauma causing disruption to the collagen fibres also occurs, resulting in a cartilage matrix less able to withstand demands of weight-bearing (Mow et al. 2000). Although this response of articular cartilage to the altered contact stresses caused by ACL sectioning was observed in animal models, the characteristics are likely to be similar in humans. Progressive lesions of the articular cartilage include pitting, fissuring and ulceration (Mow et al. 2000). These gross degenerative changes are most common in the medial aspect of the medial tibial plateau in ACL-deficient knees, with less involvement of

the lateral tibial and femoral surfaces (Mow et al. 2000). The earliest radiographic changes in human ACL-deficient knees are increased horizontal trabeculae in the cancellous bone underlying the medial tibial plateau (Buckland-Wright et al. 2000). This response of the trabeculae may be a response to shear forces within the knee and a precursor to subchondral sclerosis. Aberrant kinematics of unstable ACL-deficient knees can therefore produce changes to the articular cartilage and subchondral bone that are the precursor to osteoarthritis (Radin et al. 1986).

Not all ACL-deficient knees develop osteoarthritis; some remain free of symptoms and signs for many years. The risks for the development of osteoarthritis in the ACL-deficient knee have already been discussed and include a contact mechanism of injury, injury to other knee structures rather than a discrete ACL injury and in particular, damage to the menisci (Gillquist and Messner 1999). However, there are also individual variations in lifestyle that constitute risk factors including continuing to play pivoting sports, thereby subjecting the unstable knee to repeated episodes of trauma (Roos 1994) and neuromuscular coordination factors, that may protect the knee (Chmielewski et al. 2001).

Neuromuscular factors may be involved in the risk of developing osteoarthritis (Chmielewski et al. 2001; Rudolph et al. 2001). Some ACL-injured patients report symptoms of pain and instability of the ACL-injured knee, which they cannot tolerate, while others cope well with ACL deficiency (Noyes et al. 1983). Friden (et al 1993) found symptomatic patients had increased anterior tibial translation during a standing lunge test, viewed radiographically, than asymptomatic patients, irrespective of the passive laxity of the knee (Noyes et al. 1983; Friden et al. 1993). It appeared that asymptomatic patients were dynamically controlling the

knee instability. Dynamic control of knee stability has also been identified in gait analysis and EMG studies (Chmielewski et al. 2001; Rudolph et al. 2001). Thus, it appears that kinematic patterns and kinetic control may be associated with the risk of developing osteoarthritis in chronic ACL-deficient knees.

The natural history of degeneration in knees of community-dwelling chronic ACL-deficient subjects is not clear. The reported incidence of osteoarthritis in chronic ACL-deficient knees ranges widely from 10% (McDaniel et al. 1980; Ciccotti et al. 1994), to 50-76% at 10 years (Roos et al. 1995; Shirakura et al. 1995b; Segawa et al. 2001). This range is probably because some studies sampled patients referred for surgery (Finsterbush et al. 1990; Shirakura et al. 1995a; Chorev and Soudry 2000; Murrell et al. 2001), while other studies re-examined patients injured 10 years earlier (McDaniel and Dameron 1980; Ciccotti et al. 1994; Roos et al. 1995; Segawa et al. 2001). There are many ACL-injured people who do not require surgery, because the level of disability and pain they experience as a result of the ACL deficiency is minor. Nevertheless, when the results of the studies are pooled, it appears the majority of chronic ACL-deficient knees do develop osteoarthritis (Clatworthy and Amendola 1999; Gillquist and Messner 1999).

Epidemiology and pathology of osteoarthritis in the knee

Knee osteoarthritis has been described as the single most common cause of disability in older adults (Peat et al. 2001). In Australia, about 12% of the population and 34% of people aged over 50 years, suffer from osteoarthritis (Chapman et al. 2003). The most commonly affected joint is the knee (Osbourne et al. 2002). The incidence of total knee arthroplasty (TKA) in Australia has risen from 56.4 per 100 000 in 1994 to 76.8 per 100 000 population in 1998 (Wells et al. 2002). In the United Kingdom 20.4 per 1000 people aged over 55 years were self-reported to be so disabled that they would benefit from knee arthroplasty (Tennant et al. 1995). The Framingham study in Massachusetts found a population incidence of 10% among women aged 60-90 years (Zhang et al. 2000). A Beijing cohort study found symptomatic knee osteoarthritis occurred in 15% of women and 6% of men aged 61 to 91 years (Zhang et al. 2001). In Italy the incidence of osteoarthritis was 30% in people aged over 65 years (Mannoni et al. 2003). While there are regional differences in the incidence of knee osteoarthritis, it is clear that it is a widespread problem in the international community.

Knee osteoarthritis is most frequently of the primary idiopathic type and increases in prevalence with increasing age (Praemer et al. 1992). Secondary osteoarthritis is related to age, but also to time since the causative insult (Peat et al. 2001; Wilder et al. 2002). Secondary osteoarthritis may be due to injury or infection, or a variety of mechanical, metabolic or neurological disorders (Buckwalter and Mankin 1997). Knee injuries in youth, for example, increase the risk of later osteoarthritis 5 to 8 times (Gelber et al. 2000; Wilder et al. 2002). Progression of secondary osteoarthritis is influenced by factors including body mass

index, mechanical alignment and occupational load to the knee (Cooper et al. 1994). Idiopathic osteoarthritis is strongly related to age, whereas secondary osteoarthritis is dependent on the time since injury, as well as the age of the subject, due to wear over time.

Injuries to the knee that can cause secondary osteoarthritis include ACL-injury (as previously described), meniscal tears and fractures through the joint. These injuries change the normal load distribution, location of the load and shear stresses at the joint. Meniscectomy, for example, decreases the load-bearing area of the knee and increases the transmitted forces through the knee 2 to 3 times (Kurosawa et al. 1980). Fracture through the joint line creates a step in the articular cartilage, which produces a shearing interface and local area of stress (Bai et al. 2001). Knee trauma can produce a focal defect in the articular cartilage and trauma to the subchondral bone. This focal defect alters load transmission to the underlying bone, which stiffens, causing a shearing effect where the overlying cartilage covers the bridge between the normal and stiffened bone. Thus, secondary osteoarthritis follows a local acute injury (McKinley et al. 2001).

It has been suggested that changes to the kinematics of the knee can also produce damage to the articular cartilage over the long-term. Frankel et al (1971) postulated that changes to the axis of rotation of the knee would alter the roll/glide characteristics of knee motion and cause gouging of the joint surface (Frankel et al. 1971). This change to the axis of rotation could be due to ligament tear or to deformity altering the alignment of the knee. Since then, biomechanical studies, using electrogoniometry and force platforms to record the kinematics as well as direction and magnitude of forces through the knee have been able to demonstrate that pathological shearing occurs during walking and stepping activities, in ACL-deficient

subjects (Marans et al. 1989; Andriacchi 1990; Hollman et al. 2002; Georgoulis et al. 2003). These in vivo studies have increased the understanding of the pathological biomechanics of injured knees, which may be responsible for the osteoarthritic consequences of chronic ACL deficiency and made it possible to model the load and shear forces at the joint surface (Anderson et al. 1993; Steele et al. 1999). However, with the technology now available through MRI, it is now possible to directly visualise the kinematics of the tibiofemoral interface to compare normal and pathological motion.

Characteristics of osteoarthritis in the knee

The osteoarthritic knee is characterised by fibrillation and later eburnation of the articular cartilage, remodelling of the bone with sclerosis of subchondral bone, change to the shape of the condylar surfaces, formation of osteophytes and change to the soft tissues of the knee including synovial thickening, ligament attenuation and muscular atrophy. These changes to cartilage, bone and soft tissue do not occur in a temporal sequence, but rather are intimately related and coexist (Hough et al. 1989). The disease progresses very slowly, making it difficult to follow for any length of time. Consequently, there has been much research, particularly in animal studies, to isolate initiating events from secondary responses in the joint. However, changes to cartilage are rarely seen without subchondral bone changes (Radin and Rose 1986; Hough and Sokoloff 1989) and synovial reactions (Lukoschek et al. 1988) very early in the process. The processes occurring in the articular cartilage, soft tissues of the joint and the bony changes will now be discussed in more detail.

Changes to articular cartilage

The degeneration and progressive loss of the normal structure and function of articular cartilage is an integral part of the disease process in osteoarthritis (Buckwalter and Mankin 1997). The earliest visible sign of osteoarthritis at arthroscopy is fibrillation and softening of the superficial layers of the cartilage (Buckwalter and Mankin 1997). These surface irregularities become clefts, extending deeper into the cartilage, releasing broken fragments of cartilage into the joint space and reaching subchondral bone. Eventually the progressive loss of cartilage leaves only exposed bone (Buckwalter and Mankin 1997). These macroscopic changes reflect the changes occurring to the molecular framework of the cartilage.

Prior to the appearance of visible surface fibrillation, damage to the cartilage has already disrupted the molecular matrix of the cartilage and the water content has increased (Mow et al. 1984). The molecular matrix is made up of collagen fibres and proteoglycan chain macromolecules, along with glycoproteins and chondrocyte cells. In early cartilage damage, the long proteoglycan chains that form the matrix are broken into shorter lengths and the concentration of proteoglycan molecules decreases. Damage to the collagen fibre and proteoglycan framework increases the permeability of the cartilage. The fluid pressurisation in cartilage is essential to its load bearing and lubricating function (Mow et al. 1984). Consequently, the increase in permeability severely reduces the ability of the cartilage to withstand stresses from loading and shearing, thereby increasing the vulnerability of the cartilage tissue to additional mechanical damage (Mow et al. 2000).

In response to the cartilage tissue damage, the chondrocytes begin the repair process. Chondrocytes detect alterations in tissue strain and osmolarity and their response is rapid: the chondrocytes multiply by cloning and synthesise new cartilage matrix (Hough and Sokoloff 1989). The chondrocyte response at this stage is able to restore the cartilage matrix and successfully repair the cartilage (Radin et al. 1984; Buckwalter and Mankin 1997). If the repair response is insufficient to repair the cartilage in the face of increased mechanical loads or as a result of metabolic changes in the tissue that interfere with the ability of the chondrocytes to maintain the matrix, then the loss of cartilage matrix progresses.

Further loss of cartilage presents as visible cartilage damage and frequently symptoms of arthritis (Buckwalter and Mankin 1997). Then activity of chondrocytes reduces and they are less responsive to mechanical and metabolic stimuli. Mechanical damage and death of chondrocytes unsupported by a sound matrix structure means the cartilage is no longer able to restore itself.

Changes to bony structures

Changes to the subchondral bone accompany the articular cartilage changes. It has been suggested that bone changes precede or accelerate changes in the articular cartilage (Radin and Rose 1986; Bailey and Mansell 1997), but it appears difficult to isolate the sequence of events, as the bone and articular cartilage changes happen concurrently (Pugh et al. 1974; Wu et al. 1990). Micro fissures in the subchondral bone heal by formation of new bone tissue in the subchondral bone layer, as part of the bone's remodelling response (Wolff 1892; Churches et al. 1979). The increased density of this remodelled bone results in increased stiffness

(Finlay et al. 1989; Oegema et al. 1997; Day et al. 2001). A large part of the load taken through the joint is normally transmitted to the subchondral bone. The flexibility of the bone improves the ability of the joint to conform under load and increases the load bearing area (Radin and Rose 1986). The subchondral bone normally absorbs the load taken through the compliant articular cartilage, but as the bone stiffens the articular cartilage is compressed between the load and the stiff bone. Shear stress levels within the deepest layer of articular cartilage increase in the presence of calcified tidal cartilage and subchondral plate thickening (Anderson et al. 1993). The remodelled bone has higher mineral density (Madsen et al. 1994; Akamatsu et al. 1997), but it is of poor mechanical quality (Ding et al. 2001). On plain x-rays this remodelled subchondral bone appears as subchondral sclerosis (Fairbank 1948; Kellgren et al. 1957). This bone remodelling process can thicken and stiffen the subchondral bone and can also change the shape of the bone.

Bone remodelling during the progression of osteoarthritis can change the shape of the articulation. The shape of the articulation can become flattened and the joint margins extended by the formation of new bone osteophytes. (Kellgren and Lawrence 1957; Ahlback 1968). Animals with osteoarthritis induced by meniscectomy have developed osteophytes as fast as two weeks post surgery (Moskowitz and Goldberg 1987). The osteophytes are thought to be a response to shearing at the osteochondral junction, as the direction of the osteophyte is governed by the lines of mechanical force exerted on the area of growth and corresponds to the contour of the joint surface from which it extends (Moskowitz and Goldberg 1987; Hough and Sokoloff 1989). Osteophytes, complete with articular cartilage layer, are continuous with the bone at the margin of the joint. They can be painful to palpate and can limit joint flexibility, but they can also be completely asymptomatic (Peat et al. 2001). There is poor

correlation between the extent of bone remodelling in osteoarthritis and pain or disability (Buckwalter and Mankin 1997). Osteophytes have been considered as attempts by the joint to stabilise, since the varus/valgus laxity of the joint is increased by removal of the marginal osteophytes (Pottenger et al. 1990). Osteophytes could therefore be considered a response of bone and articular cartilage to abnormal shearing forces at the joint, extending the articular surface and contributing to the stability of the joint.

Changes to periarticular soft tissues

Pain in osteoarthritis may be due to bony changes and loss of cartilage, but is more likely to be due to chronic synovitis in the joint (Buckwalter and Mankin 1997). Although osteoarthritis is not considered an inflammatory condition, at joint replacement surgery, the synovium is frequently seen to be inflamed, with villous hypertrophy and fibrosis (Hough and Sokoloff 1989). This inflammation may be a secondary reaction to joint detritus, and fragments of articular cartilage have been found embedded in the synovium (Buckwalter and Mankin 1997). The synovium may also become sensitised to the presence of joint detritus and produce an autoimmune response (Hough and Sokoloff 1989). This synovial inflammation is considered responsible for the joint effusion and much of the pain accompanying osteoarthritis as is it a well-innervated and highly vascular structure (Hough and Sokoloff 1989). Capsular thickening as a result of chronic synovial inflammation and fibrosis may also be partly responsible for loss of range of motion in the osteoarthritic knee (Pottenger et al. 1990).

Changes to the soft tissues of the knee may also involve attrition of the cruciate ligaments and menisci within the joint. Frequently during knee replacement surgery, the cruciate ligaments are found to be absent, partly torn or show degeneration. Wada et al (1996) found that the ACL was absent in 50% of knees, partly torn in 25% and in only 25% was the ACL intact (Wada et al. 1996). The PCL, however, was intact in all the knees. Allain et al (2001) found similar incidence of ACL damage in the OA knee, but in 75% of cases where the ACL was torn, the PCL in that knee also had histological evidence of degeneration. They suggested that the attrition of the ACL indicates the quality of the PCL as a viable restraint structure is probably also compromised. The menisci also, are frequently found to be macerated or absent in advanced knee osteoarthritis (Buckwalter and Mankin 1997). It is not clear whether the damage to the menisci precedes the osteoarthritis, since meniscectomy is known to be a predictor of late osteoarthritis (Fairbank 1948). However, it is also likely that the menisci acquire degenerative tears and become macerated during the progress of the disease itself. The soft tissues within the knee, including the synovium, the ligaments and menisci, are therefore involved in the process of osteoarthritis.

Significance of the thesis

When diagnosis of ACL insufficiency is made, whether an isolated lesion or in combination with other ligament structures... the affected joint appears to embark upon a course of progressive deterioration and dysfunction.

(Fetto et al. 1980)

As early as 1938 it was recognized that ACL injury was likely to initiate a cascade of events terminating in osteoarthritis of the injured knee (Palmer 1938), yet the links between abnormal knee kinematics and degenerative change are not well understood. Frankel et al (1971) proposed a model for the damage of the articular surfaces of the knee by injuries that altered the alignment of the flexion/extension axis. This altered axis caused compression, gouging and shearing to damage the articular cartilage. It was a useful model to describe the effects of ligament injury on tibiofemoral interface dynamics, but the description of the flexion/extension axis was based on the method of Reuleaux, which becomes inaccurate when applied to sagittal x-rays of the knee (Soudan et al. 1979: see Chapter 2). Despite some flaws to the model, it may be that the concept is correct, that is, change to the axis of the knee produces degeneration.

Since the model of Frankel et al. (1971), shearing at the articular cartilage interface has been considered an important factor in degeneration of ACL-deficient knees (Anderson et al. 1993). However, there is a knowledge gap regarding whether kinematic characteristics

peculiar to ACL-deficient knees are the cause of degenerative changes or indeed associated with degenerative changes, either prospectively or in a cross-sectional study.

The pattern of tibiofemoral contact describes kinematics at the articular cartilage level. However, there is not a good understanding of the tibiofemoral contact pattern in healthy knees and no information on injured knees. Tibiofemoral contact patterns have been reported in only three papers (Dennis et al. 1998; Smith et al. 1999; Wretenberg et al. 2002), with conflicting findings. There is little published information regarding the effects of ACL injury on tibiofemoral contact patterns (Dennis et al. 1996), although tibiofemoral interface dynamics are believed to be responsible for osteoarthritis in ACL-deficient knees.

Tibiofemoral contact patterns are an important aspect of kinematic behaviour, because the tibiofemoral interface represents the area of load-bearing in the knee, the site of shear forces and hence articular cartilage stress. The tibiofemoral contact patterns of osteoarthritic knees have never been mapped, so associations between tibiofemoral contact patterns and degeneration have not been examined. This is despite events at the tibiofemoral interface being critical to health or failure of articular cartilage. This thesis examines the associations between aberrant tibiofemoral contact patterns in ligament-injured knees and the evidence of degeneration of the articular cartilage and menisci and changes to subchondral bone.

Aim of the thesis

The aim of this thesis was to examine the relationship between kinematics in ligament injury and degenerative change in the knee. This was achieved by firstly establishing a reliable technique for in vivo kinematic analysis of the knee. Then characteristics of kinematic behaviour were examined in terms of damage to the knee and evidence of degenerative change. It was hypothesised that knee injuries and pathology would be exhibited as differences in the tibiofemoral contact pattern.

Kinematics of the knee can be described by recording the pattern of tibiofemoral contact during knee flexion. A technique was established to map the tibiofemoral contact pattern through the range of active knee motion from 0° to 90° knee flexion in subjects performing a supine leg press, using MRI to record the position of the structures of the knee. Characteristics of the tibiofemoral contact pattern were examined.

Injury to the ACL was used as a model for the process of degenerative change in humans. In a cross-sectional study, knee kinematics of healthy control subjects were compared to recently injured subjects, subjects injured at least ten years previously and subjects with established knee osteoarthritis. Damage to the knee joint, menisci and subchondral bone changes, as well as areas of wear, were described and the associations between joint changes and kinematic abnormalities examined.

CHAPTER 2

DEVELOPMENT OF CONCEPTS OF KINEMATICS OF THE NORMAL KNEE

Derivation of the flexion / extension axis of the knee

Geometry of the femoral condyles

Derivation of the flexion / extension axis using three-dimensional imaging technology

Derivation of the axis of longitudinal rotation in the knee

Role of the ligaments in determining knee kinematics

Integrating the bony geometry and ligament restraint in the knee

Four Bar Linkage

Role of the ACL in the screw-home mechanism

Normal motion pathway of the tibiofemoral joint

Passive knee kinematics and active motion

Conclusions

References


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Statement from co-authors confirming the authorship contribution of the
PhD candidate

"As co-authors of the paper "*Development of concepts of kinematics of the normal knee*" we confirm that Jennifer M Scarvell has made the following contributions:

- Searching and collection of the literature
- Analysis of literature and synthesis of concepts under supervision
- Wrote the first draft of the manuscript, and followed through to publication, including proofing and final publication details of the manuscript."

Professor Kathryn M Refshauge Signed:  Date:

Associate Professor Paul N Smith Signed..... Date:.....

Ms Jennifer M Scarvell Signed..... Date:.....

REVIEW ARTICLE

Development of the Concepts of Knee Kinematics

Paul N. Smith, BMBS, FRACS, Kathryn M. Refshauge, PhD, Jennifer M. Scarvell, BAppSc

ABSTRACT. Smith PN, Refshauge KM, Scarvell JM. Development of the concepts of knee kinematics. *Arch Phys Med Rehabil* 2003;84:1895-902.

Objectives: To review the experimental evidence and development of concepts in knee kinematics and to present a synthesis of current theories.

Data Sources: Historical literature from private collections and published journals, from Galen in 160 AD, and Weber and Weber in 1860, through to current research in knee kinematics, sourced through MEDLINE and CINAHL.

Study Selection: Studies of the healthy human knee in vivo and in vitro were included. Other studies were included when relevant, for example, when knee surgery methods have led to a change in kinematic concepts. Of 285 items, 94 were included based on their contribution to original research. When relevant, authors were contacted to resolve issues.

Data Extraction: Sources included were descriptive studies, anatomic dissections, controlled experimental designs, editorials, and review articles.

Data Synthesis: The axes of rotation of the knee are fundamental to kinematic models. The hinge model is contradicted by the ellipsoid shape of the femoral condyles, which results in a moving instant center of motion. However, the "instant center of motion" model was based on analysis of sagittal sections, oblique to the plane of movement and neglecting rotation. The four-bar linkage theory linked cruciate ligament isometry with the roll and glide pattern of knee motion. Recently, however, studies of the biomechanics and histology of the knee ligaments have enabled more accurate kinematic modeling. Three-dimensional imaging and computer modeling have made possible analysis of kinematics parallel to the planes of motion and incorporation of conjoint rotation. Femoral roll back is now described as the manifestation of longitudinal rotation during knee flexion.

Conclusions: Current research concludes that the knee has 4 independent axes: patella, posterior condylar, distal condylar, and longitudinal axes. The axes combine to produce the characteristic helical motion of the knee.

Key Words: Anterior cruciate ligament; Biomechanics; Knee; Knee joint; Rehabilitation.

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IN THE PAST FEW YEARS, a paradigm shift has taken place in the understanding of normal knee kinematics. The previous work using mathematical analysis of sagittal slices

through femora described the axis of rotation of the knee as an *instant center* of rotation, shifting along a predictable, curved pathway as the knee moved through its range of flexion.¹⁻⁹ The model of the knee as a four-bar linkage was also supported by mathematical analysis, when the anterior (ACL) and posterior cruciate ligaments (PCL) were defined as rigid links.^{4,10-13} These 2 theories helped explain the combination of glide and roll that occurs at the articular surface.^{14,15} However, both of these theories were based on 2-dimensional descriptions of motion, whereas the knee moves through 3 dimensions, with 6 degrees of freedom.¹⁶⁻¹⁸ These 2-dimensional models were unable to analyze the flexion and extension axis offset from the anatomic sagittal plane¹⁹ or to account for the concomitant longitudinal rotation occurring around an axis independent from the flexion and extension axis of the knee.²⁰

Magnetic resonance imaging (MRI) has enabled great advances in research into knee kinematics by permitting accurate study of the knee in 3 dimensions, initially in vitro,^{20,21} and then in vivo.²²⁻²⁴ In November 2000, in the *British Journal of Bone and Joint Surgery*, the international research team of Freeman published a set of articles²⁵⁻²⁷ describing the kinematic motion of the normal knee by using MRI. As a result of this research, kinematic theory has seen a fundamental revision of the concept of the axes of motion of the knee, which has implications for understanding the effect of knee pathology on kinematics, for rehabilitation, and surgery.²⁸ We therefore present a review of the development of concepts of knee kinematics and present a synthesis of current theories and experimental evidence.

DERIVATION OF THE FLEXION AND EXTENSION AXIS OF THE KNEE

Geometry of the Femoral Condyles

The axis of knee flexion and extension was derived from the geometry of the femoral condyles, as early as the late 19th century, by analysis of true sagittal plane sections through the femoral condyles.^{1,2,10} From these sagittal sections, it was clear that the femoral condyles were not circular, but were elongated. The femoral condyles were described as spirals, with the lateral condyle having a greater variation in curvature than the medial condyle. If the femoral condyles were circular, the axis of flexion and extension of the knee would be fixed at its center, like a hinge. However, the changing curvature of the condyles seen on sagittal sections results in an axis that moves as the knee flexes and extends. This was described as "the instant center of motion" moving along a predictable curved pathway during knee flexion.^{1,4,5}

The instant center of motion model was useful because it linked the shape of the condyles to the motion characteristics of the knee (fig 1). In the flexed position, the instant center is closest to the joint surface, and the radius of curvature is short: only 12mm at the lateral condyle and 15mm at the medial femoral condyle.⁵ This allows the collateral ligaments and the ACL to slacken. In knee extension, the radius of curvature of the condyles is longer, and the axis of motion is furthest from the articular surface; thus, the collateral ligaments and the ACL become fully tensed, bringing the knee into its most stable position.^{29,30}

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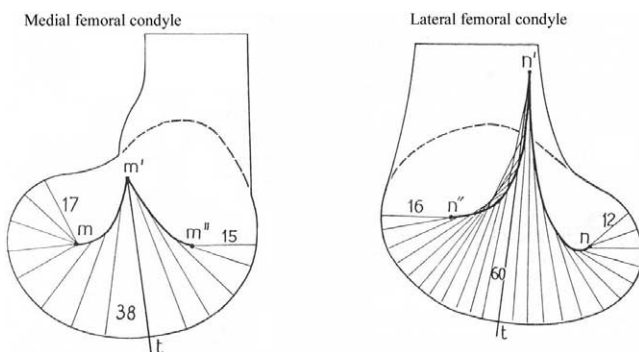


Fig 1. Anatomically sagittal diagram of the medial and lateral femoral condyles. The axis of knee flexion and extension or "instant center" moves as the knee flexes, following a predictable pathway. Here the instant center pathway is shown for the tibiofemoral joint and the patellofemoral joint for the medial and lateral femoral condyles. The distance from the instant center to the joint surface is the radius of curvature, which appears to vary throughout knee flexion.⁵ NOTE. The radius of curvature m to m' ranges from 17 to 38mm for the medial femoral condyle; the radius of curvature n to n' ranges from 12 to 60mm for the lateral femoral condyle. Legend: m to m' , instant center pathway for tibiofemoral joint; m' to m'' , instant center pathway for patellofemoral joint; t , anterior limit of tibiofemoral contact; n to n' , instant center pathway for tibiofemoral joint; n' to n'' , instant center pathway for the patellofemoral joint. Reprinted with permission.⁵

The principal criticism of the instant center theory is that it assumes the flexion and extension axis lies exactly in the sagittal plane. Fick in 1911³ reanalyzed the condyle shapes by using 3 rather than 2 dimensions and concluded that the flexion-extension axis of the knee did not lie in the sagittal plane but was offset by several degrees. This offset orientation of the flexion-extension axis would result in a single, fixed axis, rather than an instant center.²⁰ The method of Reuleaux³¹⁻³³ used to map the instant center of motion depends on the plane of motion being accurate.¹⁹ If the plane of movement is offset, then the calculated axis appears to move. From Braune and Fisher in 1891,¹ until at least the 1970s, researchers had based their calculations on this assumption.^{5,32}

The second argument against the instant center of motion theory is that, for most human articulations, the simplest articular surface shape has evolved to produce the most efficient movement. A circular articular surface with a fixed axis, such as occurs in the ankle,^{34,35} shoulder,³⁶⁻³⁸ elbow,³⁹ and hip,⁴⁰ satisfies this requirement. A constantly moving axis is inefficient because the movement of inertia is also constantly changing. However, the instant center of motion theory prevailed until recently.⁷⁻⁹

Contemporary movement toward the concept of a fixed flexion-extension axis began in the field of total knee arthroplasty. The dynamic growth in knee arthroplasty in the 1970s required that knee kinematics be understood for prosthetic design. The prosthesis must possess the stability and flexibility characteristics of the knee,^{33,41} and the axes of the knee must be reproduced as closely as possible, because malalignment had been associated with loosening and accelerated wear of prostheses.³³ Either the posterior femoral condyles or the epicondyles⁴² were used as landmarks for alignment. These landmarks define the posterior condylar axis and the epicondylar axis of the knee (fig 2). Use of the epicondylar axis to align the prosthesis during surgery indicated that a conceptual shift was taking place in the view of knee kinematics at this time. These researchers and surgeons were beginning to consider a fixed flexion-extension axis.^{41,43-46}

Derivation of the Flexion-Extension Axis by Using 3-Dimensional Imaging Technology

Progress in imaging and computational technology have enabled major advances in analysis of the geometry of the knee. MRI, radiostereometry, and cine computed tomography provide the ability to build 3-dimensional models of knee motion and computer representations of the articular surfaces,^{41,44,47-52} but with the aim of improving prosthetic design, rather than kinematic analysis.

The theory of a fixed axis of motion offset to the sagittal plane²⁹ was tested in vitro²⁰ by using an engineering tool, the "axis finder." Although this research was performed on a small sample, and the technique simple, the accuracy of the technique was excellent, and the conclusions have been supported by subsequent studies. The work of Hollister et al²⁰ supported the concept of a fixed axis of flexion in the knee offset to the sagittal plane by 7°, and with a second and independent axis for the longitudinal rotation of the knee. After this, further research in vitro was performed combining MRI to analyze movement and anatomic study to analyze geometry.^{25,53,54} This work has been followed up by using open-field MRI to analyze knee motion of volunteers sitting, standing, and squatting. This in vivo research will be discussed later.

The in vitro research has led to the development of a model of the tibiofemoral joint with 3 independent axes of motion, about which the knee moves during different kinematic events. These 3 axes are as follows.

One, the posterior condylar axis is effective from 15° to 150° of knee flexion. This axis passes through the origins of the medial (MCL) and lateral collateral ligaments (LCL) and passes through the intersection of the cruciate ligaments.^{20,21} It closely approximates the epicondylar line⁴⁵ and is offset from the sagittal plane by 7° (fig 3).

Two, as the knee reaches extension, the axis of motion shifts from the posterior condylar axis to the distal condylar axis. The flattened distal femoral condyles are in contact with the tibial plateau as the knee extends. The radius of this curve is much larger than the patellar or posterior condylar radii, locating the distal condylar axis proximal to the roof of the intercondylar notch.²¹

Three, the longitudinal axis of rotation of the knee is controlled more by the ligaments and tibial geometry of the knee than by the femoral geometry. Its derivation will be discussed in the next section.

There is an independent axis for the patellofemoral joint, which is anchored by the patella retinaculum.²¹ However, patellofemoral biomechanics are beyond the scope of this review.

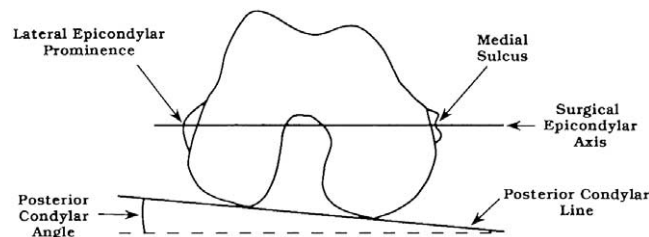


Fig 2. Diagram of the axial view of the right distal femur as seen from below by the surgeon during total knee arthroplasty with the knee flexed at 90°. The posterior condylar angle is the angle between the posterior condylar surfaces and the surgical epicondylar axis, defined by the medial and lateral epicondyle. From: Berger RA, Rubash H, Seel M, Thompson W, Crosssett L. Determining the rotational alignment of the femoral component in total knee arthroplasty using the epicondylar axis. Clin Orthop 1983;Jan(286): 40-7. Lippincott Williams & Wilkins. <http://www.lww.com>. Reprinted with permission.⁴²

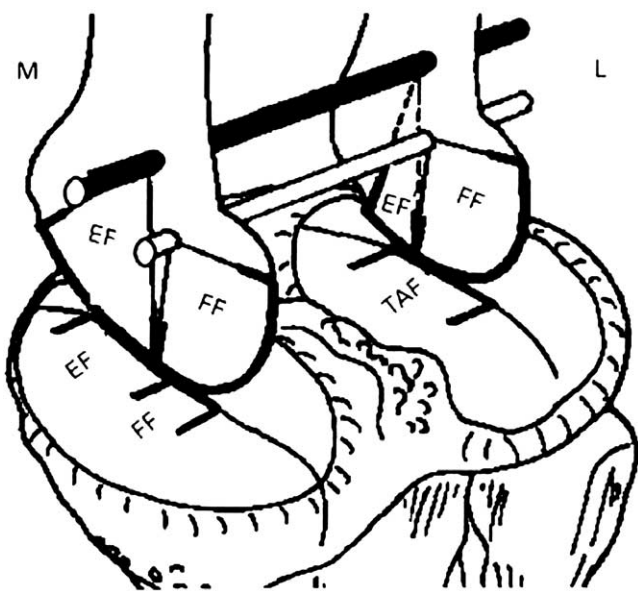


Fig 3. Diagram representing the 2 flexion axes of the knee. Abbreviations: EF, extension facet, with the corresponding distal condylar facet; FF, flexion facet, with the corresponding posterior condylar axis; TAF tibial articular facet, where the lateral femoral condyle articulates with the tibia; M, medial; L, lateral. Reprinted with permission.²⁵

DERIVATION OF THE AXIS OF LONGITUDINAL ROTATION OF THE KNEE

Methods used to derive the flexion-extension axis were applied to the longitudinal axis of the knee: in vivo by applying the method of Reuleaux to radiographs,^{5,6} in vitro using the "axis finder,"²⁰ or biplanar photography⁵⁵ by using MRI⁵⁶ and radiostereometry.⁵⁰ However, wide variations in interpretation still exist. There are aspects of asymmetry to the anatomy of the knee that determine the freedom and control of its longitudinal rotation. Although there are distinct medial and lateral tibiofemoral compartments, there are important differences that render the knee asymmetrical. The angle of the femur to the tibia is slightly valgus.⁵⁷ The articular surface of the medial condyle is shorter and wider than the lateral condyle (fig 4).⁵⁸ As a result of this asymmetry, rotation around the longitudinal axis of the knee occurs during knee flexion and extension.

Details in the asymmetry of the tibial plateau contribute to the longitudinal rotation. The medial tibial plateau is slightly concave and deepened by the medial meniscus.⁵⁷ The lateral tibial plateau is saddle-shaped, that is, concave laterally and convex anteroposteriorly. The lateral meniscus provides congruity for the femoral condyles, but the ligamentous restraints of the lateral meniscus permit some mobility. The net result of the saddle-shaped lateral tibial plateau and the concave medial plateau is to center the axis of longitudinal rotation of the knee through the medial side of the knee.^{25,59}

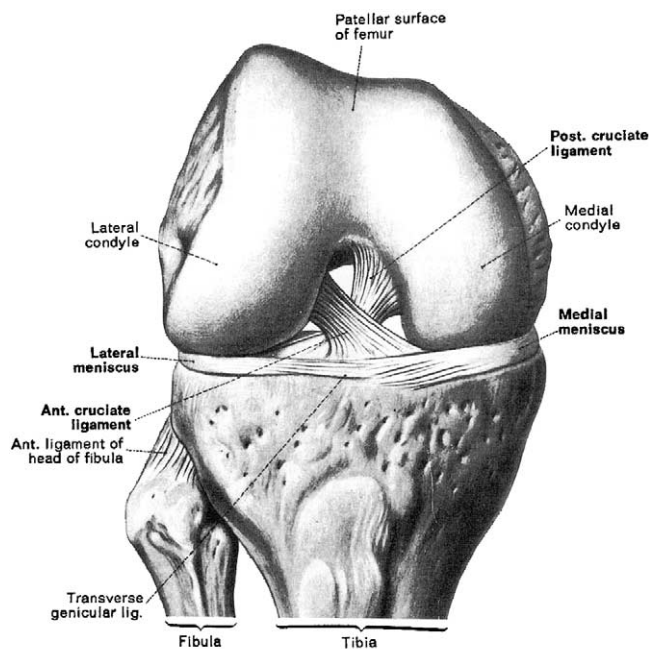
The differences between the medial and lateral menisci further illuminate the concept of longitudinal tibial rotation. The ligamentous tether to the tibia is flexible enough to allow the menisci to slide anteroposteriorly with knee motion. The medial meniscus has a deeper femoral surface and is more firmly anchored than the lateral meniscus. The lateral meniscus is permitted more mobility by its restraining ligaments.³⁰ In vivo MRI studies have observed that the lateral meniscus displaces posteriorly as much as 2 times further than the medial

meniscus on knee flexion,^{60,61} suggesting that the longitudinal axis of rotation is medially located.

Attempts to map the longitudinal axis have resulted in wide variations in interpretation. Cadaveric studies have described a fixed longitudinal axis passing through the medial femoral condyle,³⁰ through the intercondylar eminence^{20,62} (fig 5), or as an instant center.⁵⁵ In vivo studies have shown longitudinal rotation occurring throughout the range of knee flexion by using MRI to measure the displacement of the menisci^{60,61} and by measurement of bony landmarks.⁶³ However, even with 3-dimensional imaging, the location and nature of the axis have been difficult to describe with consistency.

Evidence of the importance of the longitudinal axis of rotation of the knee is illustrated by the development of knee prostheses. Early knee prostheses were designed to constrain longitudinal rotation of the knee, but became loose or broke under this constraint. Mobile bearing knee prostheses have allowed longitudinal rotation around a centrally located axis, with better longevity of the prosthesis.⁶⁴ Recently, a knee prosthesis has been designed with the medial compartment acting as a ball-and-socket joint and the lateral compartment as an outrigger, but it has not yet been in use long enough to show whether drawing the longitudinal axis of the knee medially reduces wear and thus extends the life and function of the prosthesis.⁶⁵

The wide variation of opinion regarding both the location of the axis of longitudinal rotation, and whether the axis is fixed is because of the axis being subject to many more factors than just the bony architecture. The bony architecture could be used reliably to derive the flexion-extension axis of the knee, so cadaveric dissection experiments and in vivo experiments of passive or active knee motion could give consistent results. The longitudinal rotation axis, by contrast, is subject to the influ-



From: Clemente C. Anatomy: a regional atlas to the human body. Baltimore: Urban & Schwarzenberg; 1987. p 452. Reprinted with permission.⁵⁸

Fig 4. The anatomy of the flexed knee showing the asymmetry of the joint. Ant., anterior; lig., ligament; post., posterior. Reprinted with permission.⁵⁸

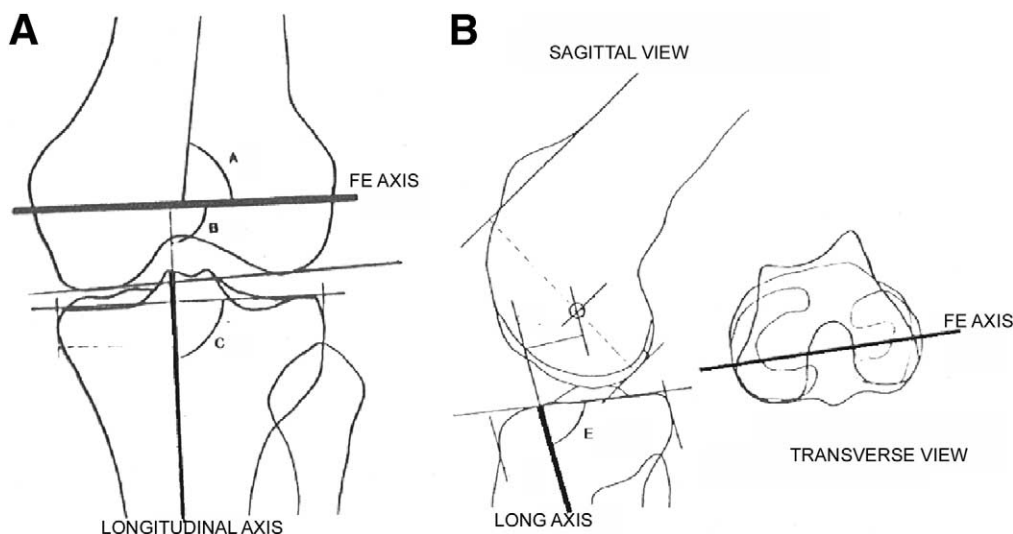


Fig 5. The location of the rotation axes of the knee. (A) Diagrammatic representation of axes in anteroposterior (AP) view with axis parallel to the plate. A is the angle the flexion-extension (FE) axis makes with the shaft of the femur (mean, 84°); B is the angle between the flexion-extension and left-right (LR) axes in the AP plane (mean, 88°). C is the angle between the longitudinal rotation (LR) axis and the tibial plateau (mean, 89°). (B) Diagrammatic representation of axes in sagittal view with x-ray beam parallel to the flexion-extension axis. E is the angle between the LR axis and the tibial plateau in the axial lateral plane (mean, 85°). From: Hollister A, Jatana S, Singh A, Sullivan W, Lupichuk A. The axes of rotation of the knee. *Clin Orthop* 1993;May(290):259-68. Lippincott Williams & Wilkins. <http://www.lww.com>. Reprinted with permission.²⁰

ence of the bony architecture in addition to the effect of ligamentous restraints, load-bearing forces, and muscle activity. It is therefore necessary to review these factors before drawing conclusion about the nature of the longitudinal rotation axis in the knee.

ROLE OF THE KNEE LIGAMENTS IN DETERMINING KNEE KINEMATICS

Galen in 160 AD described the role of ligaments as supporting structures, stabilizing the joints and preventing abnormal motion. Since then, the knee ligaments have been subject to rigorous scrutiny, probably because the knee derives its stability from ligament structures, rather than its bony architecture. Much of the current understanding of the guidance of knee motion by the knee ligaments is based on the work of Brantigan and Voschell in 1941.³⁰ They observed that the MCL, LCL, and ACL were taut in knee extension, but the LCL slackened in flexion. They observed tension in different regions of the MCL as the knee flexed. Both cruciates appeared taut, although not tense, throughout flexion. Advances in technology and biomechanics have enabled modeling of the various bundles and fascicles within the structure of the ligaments, and description of their roles during knee movement.⁶⁶⁻⁶⁸ Belief that the cruciate ligaments were isotonic through flexion have been superseded by the concept of ligament loads shifting between intraligamentous bundles at different stages of knee movement.^{69,70}

The most recent role designated to the knee ligaments is to act as the fulcrum for the axes of motion.²¹ This is made possible by the ability of discrete ligament bundles within the major ligaments to maintain steady tension while the ligament twists during movement.^{67,70} This is particularly true of the MCL and ACL. Frankel et al³² found damage to joint structures resulted from pathologic shifts in the axis of rotation in the sagittal plane. Radiostereometry studies have recorded the change in the longitudinal rotation after ACL injury, with the assumption being that this change is implicated in the genesis

of osteoarthritis in the injured knee.⁷¹⁻⁷³ Further 3-dimensional research is required to determine the effect of ligament damage on the axis of motion and resultant in vivo knee kinematics.

INTEGRATING THE BONY GEOMETRY AND LIGAMENT RESTRAINT IN THE KNEE

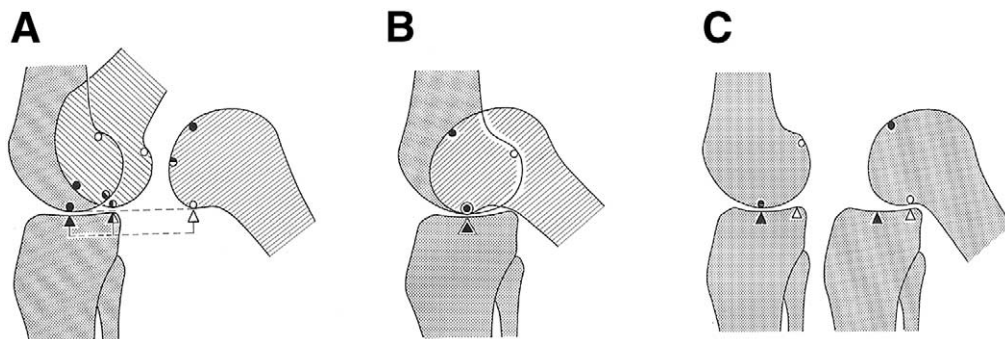
The passive motion characteristics of the knee are determined by the bony architecture and the ligament structures working in concert. The geometry of the femoral condyles, together with the direction of action of the ligaments, control the flexion and extension motion and determine the location of the flexion-extension axis of rotation. The flat shape of the tibial plateau surfaces, the relative mobility of the menisci, and the action of the ligaments of the knee determine the motion pathway of the femoral condyles on the tibial plateau.

Several models have attempted to integrate the structure and function of the knee, ranging from a hinge at its most simplistic, to a complex roll-glide mechanism.^{5,11,13} The knee does not purely glide on the tibia, as evidenced by the position of the femoral condyle at the end of flexion seated posteriorly on the tibial plateau. Nor does it purely roll, because the articular surfaces of the femoral condyles are much longer than the tibial plateau¹¹ (fig 6).

Four-Bar Linkage

The four-bar linkage theory defines the 4 rigid links as the ACL and PCL and the bony structure of the femur and tibia. This model married cruciate ligament isometry with the roll-glide pattern of knee motion.^{11,13} It has been a useful theory in modeling the placement of grafts in reconstruction surgery.⁷⁴ It has also provided a model to explain the appearance of roll back of the femoral condyles on the tibial plateau, which is seen on plain radiographs^{32,75} and fluoroscopy⁷⁶ (fig 7). Roll back was therefore incorporated into the design of knee prostheses and was the basis of the posterior cruciate retaining prosthetic designs.^{12,77-79} Recently, however, studies^{66,70} of the biomechanics and histology of the knee ligaments have enabled

Fig 6. Roll and glide movements of the femoral condyle. (A) Pure rolling motion: the femur rolls off the tibial plateau before full flexion is complete. (B) Pure sliding motion: the femur impinges the posterior tibial plateau before full flexion is achieved. (C) Combined rolling and gliding of the femoral condyles allows full range of flexion. Reprinted with permission.¹¹



more accurate kinematic modeling. To describe the cruciates as isometric may be an oversimplification.^{70,80} Study into the tensions in the various fascicles of the cruciate ligaments during knee flexion has enabled better understanding of their dual roles in controlling rotation and tibiofemoral stability in the terminal extension of the knee as compared with flexion. When performing knee reconstruction, surgeons have also found that ACL graft tension is highly dependent on knee position, and they have found more flexibility in graft placement than is expected under the four-bar linkage model.⁷⁴

Role of the ACL and Screw-Home Mechanism

The stability of the knee in extension is thought to be more because of ligament restraint than the bony architecture.¹¹ Knee extension is characterized by increasing tension in the MCL, LCL, and ACL and the checkrein effect of the posterior capsule.¹¹ The increased radius of curvature of the distal femoral condyle increases the distance between the articular surfaces and the ligament origins, thus creating tension in these ligaments and decelerating the joint. The stability of the extended knee is therefore highly dependent on ligament integrity.

The external rotation (or screw-home) of the tibia in terminal extension of the knee provides a further check to extension.¹¹ This passive phenomenon was thought to be because of tension of the ACL at the limit of extension, causing external rotation of the tibia.^{5,81} The asymmetry of the femoral condyles also contributes to the terminal rotation: the longer medial femoral condyle continues to roll after the lateral condyle has reached its limit of motion.^{5,30} The curve of the intercondylar notch of

the medial femoral condyle forces the tibia to rotate as it glides against the tibial spine.⁸²

However, Blankevoort et al¹⁶ and La Fortune et al⁸³ found no evidence of screw-home in vivo. Blankevoort suggested that, within the envelope of passive motion, which characterizes the knee, there is a pattern of joint motion for each specific task, which is a combination of the passive motion characteristics and the external loads of muscle forces and weight bearing. Therefore, screw-home can be overcome by external forces during active movement.^{16,83,84}

The description of the screw-home mechanism implies that the external rotation of the tibia is restricted to terminal extension. However, recent work on the longitudinal axis of rotation in the tibia shows that the rotation of the tibia during knee extension is a continuation of the rotation that occurs throughout the range of knee movement from flexion into terminal extension.^{20,26}

NORMAL MOTION PATHWAY OF THE TIBIOFEMORAL JOINT

The ability to reconstruct 3-dimensional interpretations of in vivo knee kinematics using MRI has led to review of knee kinematics theory by permitting separate analysis of the medial and lateral compartments and accurate alignment with the axes of motion.^{23,24} Todo et al²³ analyzed MR images perpendicular to the flexion-extension axis, rather than sagittal images. They concluded that roll back, if present at all, is small, perhaps 2mm, and can be suppressed in either the medial or lateral compartment by the longitudinal rotation of the knee (fig 8).

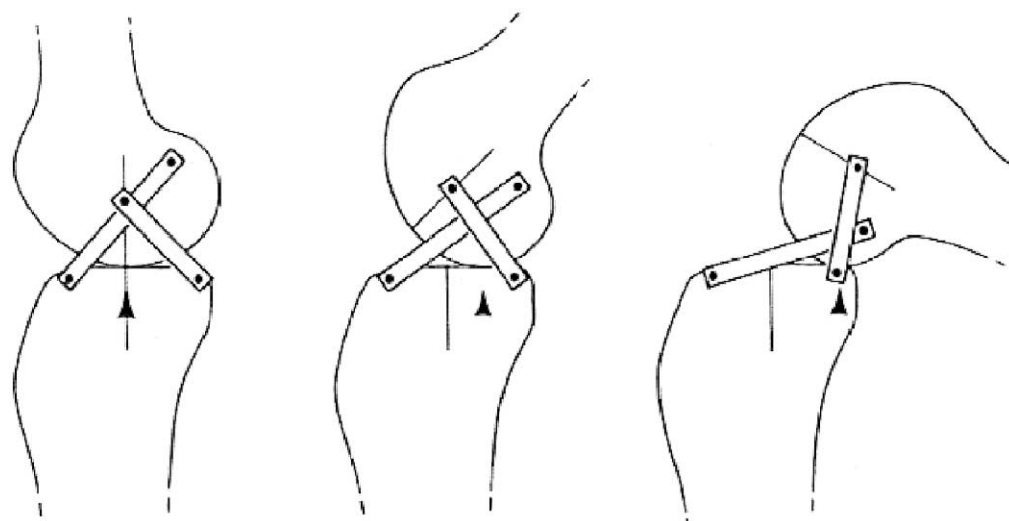


Fig 7. Schematic representation of the knee as a four-bar linkage system showing posterior displacement of the point of tibiofemoral contact with flexion. Reprinted with permission.¹¹

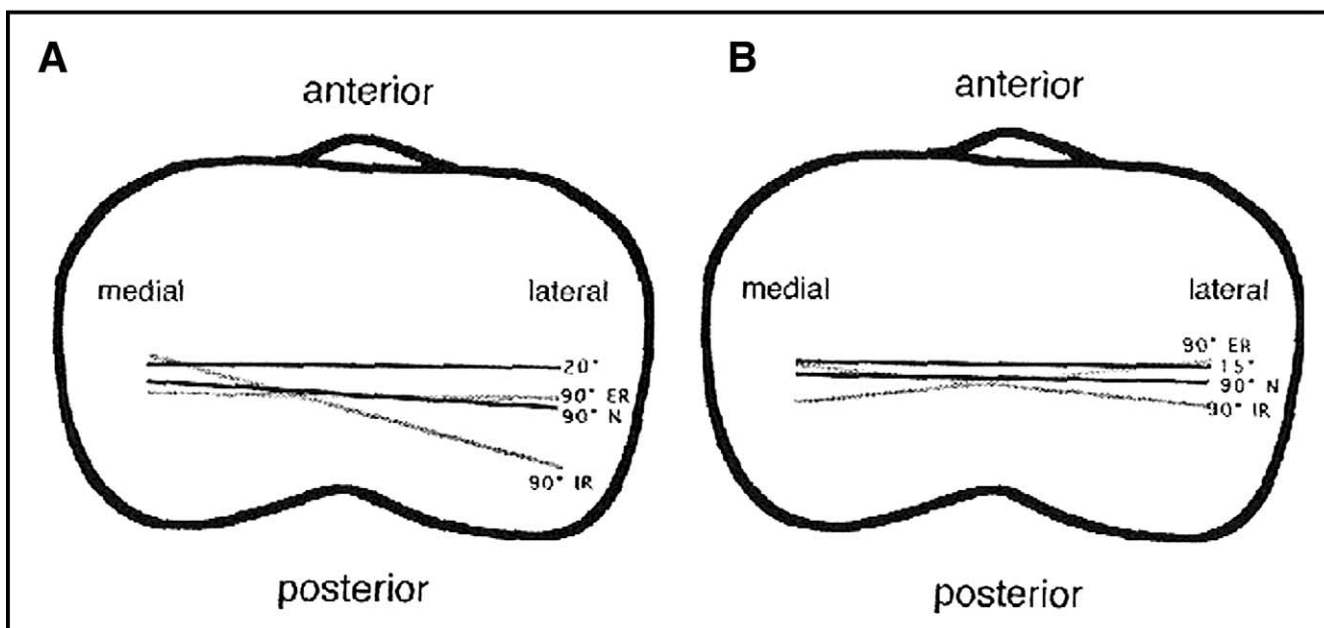


Fig 8. The position of femoral condyles on the tibial surface.²² Mean tibiofemoral contact positions of normal knees of (A) 4 female volunteers and (B) 10 male volunteers. Four positions are shown: early flexion (15° or 20°) with neutral rotation, 90° of flexion with neutral rotation (90° N), 90° of flexion with internal rotation (90° IR), 90° of flexion with external rotation (90° ER). From: Todo S, Kadoya Y, Moilanen T, et al. Anteroposterior and rotational movement of the femur during knee flexion. *Clin Orthop* 1999;May(362):162-70. Lippincott Williams & Wilkins. <http://www.lww.com>. Reprinted with permission.²³

The study was limited by small sample and small effect size and by positioning the subjects in side lying, possibly increasing medial ligament tension. However, the results have been reproduced by subsequent research.^{26,85}

Motion of the femoral condyles on the tibial plateau can be explained by the longitudinal rotation of the knee. The knee rotates about its longitudinal axis simultaneously with flexion about its flexion-extension axis (in helical motion)⁵⁰ throughout knee flexion.⁸⁶ The medial location of the longitudinal axis results in a greater posterior displacement of the lateral femoral condyle than the medial femoral condyle during knee flexion.^{23,87}

Our team performed a similar mapping of the tibiofemoral contact points in a closed kinetic chain "legpress."⁸⁷ Tibiofemoral roll was shown from knee extension to 30° of flexion, then from 30° to 90° of flexion the knee showed more femoral gliding on the tibial plateau. However, the axis of longitudinal rotation did not appear to pass through the medial tibial plateau, as it has with open kinetic chain studies. This suggests the longitudinal rotation of the knee was constrained by the placement of the feet in the closed chain position. Other research has also shown the constraint of longitudinal rotation by weight bearing.²⁶

Passive Knee Kinematics and Active Motion

Structural characteristics of the knee determine its limits of flexibility and stability. Within this envelope of passive motion the muscular forces and weight-bearing loads imposed during active movement will direct the pattern of knee motion.¹⁶ The muscular forces of the hamstrings and gastrocnemius are able to control the longitudinal rotation of the knee in flexion, for example, putting a toe into a sock (open chain) or during a cutting maneuver (closed chain). Kinematic characteristics such as screw-home, which are evident in vitro, may not be shown in vivo, because they can be constrained by the dynamic

forces occurring. Much research in vivo has been plagued by the problem of inconsistency of movement: subjects stepping up onto a box will step up 5 times, in 5 different ways. This is because many variations of active movement are available within the envelope of passive motion of the knee. The longitudinal rotation of the knee may not be a fixed axis, as occurs with the flexion-extension axis, because it is influenced less by the bony anatomy of the knee, and controlled by the summation of passive soft tissue structures and dynamic forces occurring around the knee. Thus, the location of the longitudinal rotation axis of the knee differs for each activity.

CONCLUSION

Several concepts of knee kinematics have been used to explain the relationship between the structure of the knee and its movement characteristics. The major paradigm shift in recent years was from the instant center of motion theory to fixed axes of motion. Three-dimensional imaging and computer modeling have made possible analysis of kinematics parallel to the planes of motion and incorporation of conjoint rotation. Femoral roll back is now described as the manifestation of longitudinal rotation during knee flexion. The integrity of the MCL and ACL are crucial to knee kinematics under this model, because the restraint provided by the various fascicles influence knee stability throughout the range of motion. The model of the knee as a ball-and-socket joint through the medial compartment, with the lateral compartment acting as an outrigger, may be one set of conditions in which the knee can function, but the axis of longitudinal rotation is more likely to vary, depending on the dynamic forces of the particular situation. The designers of knee prostheses must therefore consider, not the incorporation of roll back into design, but rather the freedom or control of longitudinal rotation. Knee brace design likewise must consider the constraint to longitudinal rotation.

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CHAPTER 3

KINEMATICS IN KNEE PATHOLOGY

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Structure of the thesis

Structure and function of the ACL

Anatomy

The anterior cruciate ligament (ACL) arises from the lateral femoral condyle on its medial surface (Jobe and Wright 1994). The fibres run with a lateral twist to the tibia. The tibial insertion is oval-shaped and the ligament inserts over a wide area at the tibial eminence. The ACL is described as having two or three bundles, although often these bundles are not grossly discernable (Dye and Vaupel 2000). The bundles are defined by the tibial attachment: the anteromedial bundle, the posterolateral bundle (Girgis et al. 1975) and sometimes an intermediate bundle (Dye and Vaupel 2000). The complex twisting geometry and discrete fibre bundles allow the ACL to be functional over a wide range of flexion and rotation angles (Woo et al. 1994).

The role of the ACL as a primary and secondary restraint

The ACL is the primary restraint to anterior tibial displacement in the knee (Butler et al. 1980). The ACL resists the anterior component of the quadriceps mechanism load, thus stabilising the knee joint (Boden et al. 2000). The ACL is also a primary restraint to medial tibial translation (Woo et al. 1994). A secondary role of the ACL is to control the coupled rotation of the knee during flexion and extension (Lane et al. 1994; Anderson and Dyhre-Poulsen 1997). The attachment to the medial side of the lateral femoral condyle provides a rotary component to the tension in the ligament (Brantigan and Voshell 1941; Markolf et al.

1990). In terminal extension of the knee the ACL acts to produce some external rotation of the tibia in the final locking or screw-home of knee extension (Hallen and Lindahl 1966; Fuss 1992).

Tension in the anteromedial and posterolateral bundles

There has been lack of agreement regarding the tension in the bundles of the ACL. Every combination of tension in flexion, tension in extension and tension midrange has been proposed by various research groups. Brantigan and Voschell in 1941, described the entire ACL as taut in extension and relaxed in knee flexion (Brantigan and Voschell 1941). However, his study was limited to manual manipulation of cadaver sections. France et al (1983) using strain gauges, found the anteromedial bundle of the ACL was under little tension in knee extension, peak tension at 70° and relaxed again further into flexion (France et al. 1983). The posterolateral bundle was under peak tension at 0°, slackened towards 70° and tightened again beyond 100° knee flexion. This pattern of ligament tensioning midrange has not been supported by other authors. This lack of support could be because strain gauges are sensitive to temperature variations, which are complex to account for or because the unloaded knee is capable of a wide variety of kinematic patterns. Markolf et al (1990) found the anteromedial bundle was under peak tension at 0° and relaxed to zero by 20°, but between 20° and 90° there were erratic strain patterns in the anteromedial bundle (Markolf et al. 1990). These conflicting experimental results regarding tension in the bundles of the ACL were clarified by later research enabled by advances in experimental design and the development of constrained testing machines fitted with force and direction sensors.

Constrained testing machines have enabled in vitro studies that simulate directional forces acting across the knee joint. The advantage of customised testing jigs is that test conditions can be strictly controlled. Force, direction and displacement can be recorded accurately. The contribution of each ligament or portion of a ligament to restraint, can be calculated by testing, sectioning the ligament and retesting. Ligaments may be sectioned in different sequences to distinguish the roles of primary and secondary restraints. Experiments using the Oxford rig (Markolf et al. 1990; Shoemaker and Daniel 1990; Zavatsky 1997) were able to record the loads through various ligaments during a simulated stance phase of gait. Freedom of motion could be permitted or constrained at the knee in prescribed directions. Displacement could then be measured and compared to directional loads while the ligaments were sectioned in sequence (Pizziali et al. 1980; Lipke et al. 1981; Fukubayashi et al. 1982; Grood et al. 1988; Shoemaker and Daniel 1990). Thus, the relative contributions of ligaments could be derived. This method has developed as technology has progressed. Now six-degrees-of-freedom motion and load sensing can be permitted (Rudy et al. 1996). This six-degrees-of-freedom jig has been named the Robotic Universal Force Moment Sensor (UFS). Motion with six degrees-of-freedom also permits coupled movements to be analysed: the result of ACL sectioning was laxity in anterior translation and internal rotation of the knee (Rudy et al. 2000).

The primary and secondary restraint roles of the ACL were derived from experiments with the Oxford rig and robotic UFS (Butler et al. 1980; Woo et al. 1994; Livesay et al. 1995; Sakane et al. 1996; Livesay et al. 1997; Sakane et al. 1997; Sakane et al. 1999; Woo et al. 1999). Under an anterior load of 100N applied to the tibia, the in situ load in the ACL exceeded the applied load (up to 129N: (Takai et al. 1993). The distribution of the load between the anteromedial and posterolateral bundles varied with the knee flexion angle: in extension the

load was evenly distributed between the bundles, but at greater than 45° flexion the load was borne through the anteromedial bundle alone (Takai et al. 1993; Sakane et al. 1997).

In vivo experiments have also been performed to determine the tension in the anteromedial bundle of the ACL, using strain gauges implanted under local anaesthetic in consenting volunteers (Beynnon et al. 1990; Fleming et al. 1993; Beynnon et al. 1995; Beynnon et al. 1997; Beynnon and Fleming 1998; Fleming et al. 1998). These experiments recorded the strain in the anteromedial bundle of the ACL during functional activities including cycling (Fleming et al. 2002), active knee motion and rehabilitation exercises (Beynnon et al. 1995; Beynnon et al. 1997). The anteromedial bundle of the ACL consistently bore higher tension in knee extension, the tension decreasing with increasing knee flexion.

The consensus at present from in vitro testing, is that tension in the anteromedial bundle of the ACL remains relatively constant through knee flexion, whereas the posterolateral bundle relaxes as the knee flexes (Dye and Vaupel 2000). On the contrary, the in vivo tests tend to suggest that tension in the anteromedial bundle decreases as the knee flexes (Beynnon et al. 1997). This difference may be accounted for by other dynamic forces acting across the knee in vivo, such as joint compression and muscle loads. The effect of these dynamic forces will now be discussed.

The role of the ACL in stabilising the knee during dynamic activities is relevant for exercise prescription post-injury or reconstruction. The quadriceps tendon exerts an anteriorly directed load on the tibia, particularly in the early part of knee flexion, for which the ACL is the primary restraint. In vitro and in vivo studies have found that quadriceps activation produces

an anterior shear force at the tibial plateau in knees between 0° and 45° flexion (Arms et al. 1984; Hsieh and Draganich 1998; Li et al. 1999). However, between 60° and 90° the resultant force of the quadriceps was a posterior shear force (Smidt 1973; Howe et al. 1990; Beynnon and Fleming 1998). The strain produced in the ACL by quadriceps activity may be countered by co-contraction of hamstrings muscles or increasing joint compression (O'Connor 1993). Activation of the hamstrings muscles reduced the anterior shear force produced by quadriceps muscles in mathematical models of knee activity (O'Connor 1993; MacWilliams et al. 1999; Pandy and Shelburne 2000; Toutoungi 2000). However, in vivo, Beynnon et al (1995) found that activation of hamstrings muscles was not able to fully eliminate the ACL strain during open-chain exercises. Beynnon et al (1995) concluded there was less strain on the ACL if quadriceps muscle strengthening exercises were performed at greater than 60° knee flexion or with hamstrings muscle co-contractions between 35° and 60° knee flexion.

Joint compression may also reduce the strain on the ACL during physical activity (McGinty 2000). In vitro the knee has more stability when joint compression is applied (Markolf et al. 1981), implying that joint compression, such as occurs during closed-chain exercises, would protect the joint from shear forces. Closed-chain exercises are those where the kinetic chain is closed by fixation of the foot to the ground or another solid structure, enabling joint compression due to body weight and ground reaction forces to occur (Fu et al. 1992). Open-chain exercises are those where the foot is free, for example, non-weight bearing exercises and the forces across the joint are muscular. In vivo, closed-chain exercises result in less ACL strain than open-chain exercises (Wilk et al. 1996; Escamilla et al. 1998). Anterior tibial translation is reduced in joint compression conditions, which suggests that joint compression can be used to protect the ACL from strain (Torzilli et al. 1994). Rehabilitation protocols for

patients following ACL injury or reconstruction have therefore been designed to protect the ACL from strain by combining joint compression, closed-chain conditions and quadriceps/hamstrings muscles co-contraction (O'Connor 1993; MacWilliams et al. 1999; McGinty 2000; Toutoungi 2000).

Kinematics of the ACL-deficient knee in vitro

In vitro studies have shown that the knee kinematics change when the ACL is sectioned. In the absence of the ACL the knee has additional anterior and rotational laxity (Butler et al. 1980; Lipke et al. 1981; Anderson and Dyhre-Poulsen 1997). Secondary restraints to anterior and rotational motion become important for stability and are subject to increased or unusual strain (Butler et al. 1980). The secondary restraints to anterior translation are the medial meniscus (Allen et al. 2000) and the medial collateral ligament (MCL) (Markolf et al. 1976; Butler et al. 1980). The role of the ACL was derived from in vitro studies where the ACL was sectioned before and after the MCL (Markolf et al. 1976) and before and after meniscectomy (Markolf et al. 1976; Allen et al. 2000). The medial meniscus, for example, is subjected to significantly greater strain after the ACL is sectioned. The strain on the medial meniscus is greatest at 60° knee flexion (Allen et al. 2000). The MCL is also subject to increased strain after the ACL is sectioned, particularly from 0° to 45° (Markolf et al. 1976). The MCL is the primary restraint to valgus rotation, but is also the secondary restraint to anterior translation and medial translation in the ACL-sectioned knee, particularly in knee extension (Woo et al. 1994). Further into knee flexion the medial capsule also contributes. In the ACL-sectioned knee the

MCL and medial meniscus become important as the secondary restraints to anterior translation and internal rotation. They are therefore also vulnerable to increased strain loading (Allen et al. 2000). Thus, the kinematics of the ACL-deficient knee are characterised by increased joint laxity during motion and loading conditions and the roles of the secondary restraint structures, particularly the MCL and medial meniscus are emphasised.

Kinematics of the ACL-deficient knee in vivo

Recent advances in technology, particularly radiography and in data transformation, have enabled in vivo imaging to move from two to three dimensional images and improve in accuracy. The new technologies have enabled advances in the understanding of normal knee kinematics, as was described in Chapter 2. They have also been applied to the study of kinematics of ACL-deficient knees. It is logical at this point to describe the methods that have been used for the study of in vivo knee kinematics and then to describe their findings.

Methods of kinematic analysis in vivo

In vivo studies of kinematics in the ACL-deficient knee have been conducted in order to understand the resultant pathological motion and to assess the effectiveness of interventions such as surgery and bracing. These studies have used a variety of methods including non-invasive electrogoniometry (Alkjaer et al. 2002; Beynnon et al. 2003); highly invasive implants into the tibia and femur using intracortical pins as bone markers for the measurement of kinematics from 3-dimensional video footage (La Fortune et al. 1992; Ramsey et al. 2001) or tantalum pellets imaged by roentgen stereo photogrammetry (RSA)(Karrholm et al. 1988a; Brandsson et al. 2001). MRI has not yet been used to examine ACL-deficient knee kinematics. These kinematics studies have enabled an understanding of kinematics of the ACL-deficient knee, in particular the differences in translations and rotations that occur in the knee unconstrained by the ACL.

2-dimensional analysis of kinematics

Some of the earliest investigations of kinematics in ACL-deficient knees were performed using plain x-rays, as this was the best available technology at the time for direct visualisation of the knee (Frankel et al. 1971). Plain x-rays use accessible technology but are limited to still images at intervals of knee flexion, not motion. Measurement of longitudinal rotation is not possible from x-rays because the two femoral condyles are superimposed and there are inadequate tibial landmarks in the images.

Fluoroscopy produces a 2-dimensional image, but records images rapidly (10 frames per second), so that motion is recorded. Banks and Hodge (1996) initiated the use of this method

of kinematic analysis in normal and replaced knees (Banks and Hodge 1996). The technique was developed subsequently to assess the effect of ACL and PCL sectioning on kinematics after total knee arthroplasty (TKA) (Dennis et al. 1996). Researchers have found fluoroscopy to be limited due to its 2-dimensional nature and poor image resolution. The method has been refined by computer-assisted image fitting, that is, by superimposing images of knee prostheses or 3-dimensional computerised tomography images, in order to improve the accuracy of the technique (Banks and Hodge 1996; Dennis et al. 1998; Stiehl et al. 2001; Komistek et al. 2003). The technique is currently being used to describe kinematics in prosthetic knees (Bellemans et al. 2002; Komistek et al. 2003).

3-dimensional analysis of kinematics

Biplanar video

To investigate the 3-dimensional nature of knee kinematics, technologies such as biplanar video, electrogoniometry and biplanar x-ray techniques have been used. Biplanar video analysis with skin markers has the advantages of being non-invasive, not involving radiation and it is easily synchronised with other data collection, such as electromyography (EMG) and force platform data (Wexler et al. 1998; Chmielewski et al. 2001; Alkjaer et al. 2002; Hollman et al. 2002; Georgoulis et al. 2003). Using this combination of data it is possible to build up a model of kinematics and kinetics in vivo. Motion of the subject is not inhibited by this method, so that activities such as walking, jumping, running and stair climbing are possible. The primary disadvantage in this method of video analysis is that the skin markers move independently of the underlying bone. As a method of kinematic research it is indirect and consequently lacks accuracy. There has been a modification of the method, using point

clustering to define a body segment (Andriacchi et al. 1998) and this improves its accuracy. Another problem with using biplanar video for kinematic study of the knee, is that the model used for analysis of kinematics usually defines all joints as spherical socket joints (Wexler et al. 1998), which is inaccurate. The model may be built with the knee as a multiaxial joint (Hollman et al. 2002), but this model is insufficient to describe the axes of rotation at the knee. Because of these limitations (movement of the skin markers and the simplistic model of the knee), the method is not able to measure the finer details of knee motion, such as anterior translations, longitudinal rotations and tibiofemoral contact behaviour. Despite the limitations of video analysis, it is still very useful, because it is able to describe kinematics and kinetics in ACL-deficient knees during a wide variety of functional activities.

A variation of biplanar video techniques using skin markers, is biplanar video with bone markers (La Fortune et al. 1992; Ramsey et al. 2001). In this method, intraosseous pins are implanted into the tibia and femur under local anaesthetic (Figure 1). Three-dimensional positional data is recorded by up to six high-speed video or infrared cameras. Digital analysis provides accurate and reliable kinematic information. This method has been used only in small numbers of subjects, because of its highly invasive and painful nature and consequent high risk to subjects (La Fortune et al. 1992; Iishi et al. 1997). Non-invasive methods may be less accurate in some cases, but have advantages in ease of use and low risk to subjects.

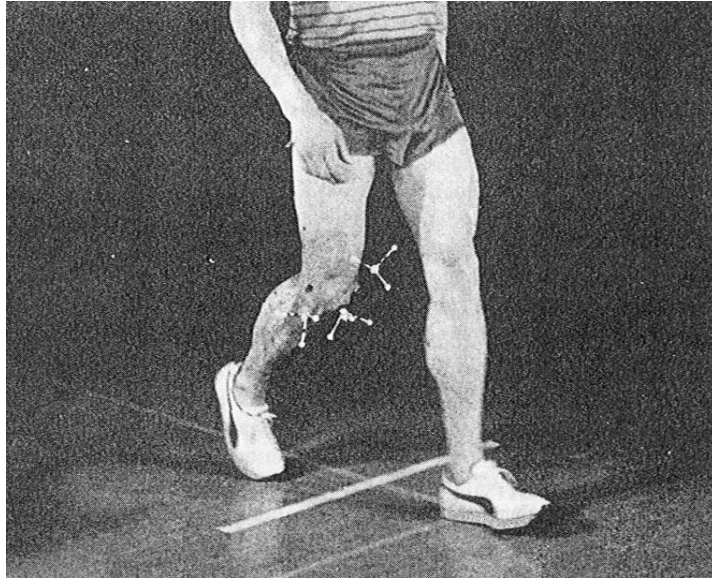


Figure 3.1: View from one of the high-speed video cameras as a subject walks with three point-markers attached to tibial, femoral and patella intraosseous pins (La Fortune et al. 1992).

Electrogoniometry

Electrogoniometry is a non-invasive, in vivo method of analysis, which purports to enable six-degrees-of-freedom in analysis of knee motion (Marans et al. 1989; Yack et al. 1994; Vergis et al. 1997; Kvist and Gillquist 2001; Vergis et al. 2002; Witvrouw et al. 2002). It is low risk to volunteers as it is non-invasive and involves no radiation exposure. However, like video analysis, it is an indirect measure and is limited in its ability to characterise motion of the bony structures and the electrogoniometry frame is prone to slipping against the skin. Vergis et al (2002) recently compared the CA-4000 electrogoniometer with lateral fluoroscopy images for measurement of tibial translation. They found a correlation of $r = 0.89$ between the electrogoniometer and fluoroscopic measurements in healthy knees, with lower reliability for ACL-injured knees. However, it would have been more rigorous to validate electrogoniometry

against image enhanced fluoroscopy or the reference standard in kinematics analysis, RSA (Brandsson et al. 2001). Electrogoniometry is non-invasive 3-dimensional method of kinematic analysis and as such is a useful tool in the research and clinical environment (Beard et al. 2001; Kvist and Gillquist 2001; Kvist et al. 2001; Hollman et al. 2002).

Roentgen Stereophotogrammetric Analysis (RSA)

The gold standard for kinematic analysis of the ACL-deficient knee is RSA (Karrholm et al. 1988a; Jonsson et al. 1989). The ACL-deficient knee was studied using RSA as early as 1988 by implanting tantalum beads into the tibia and femur during arthroscopy (Karrholm et al. 1988a; Karrholm et al. 1988b; Jonsson et al. 1989). These beads are more accurate than anatomical landmarks from which to reference motion and are fixed within the bone, so they do not slip or move independently of the underlying tissues. In vivo kinematics is then recorded via biplanar x-ray. The accuracy of this method is reported as 0.2° rotation and 0.2mm translation (Karrholm et al. 1988a). Sample sizes are limited by this method due to its invasive nature and high exposure to ionising radiation. Brandsson et al (2002) reported that in subjects performing a step-up, standard deviations in recorded kinematics were ten times higher than the measurement error, indicating that variations in performance were markedly larger than the error of the method.

While RSA is considered the gold standard for kinematic analysis, it has distinct disadvantages: it is expensive, has limited availability and is invasive. It is an invasive technique, requiring transcutaneous implantation of intraosseous tantalum beads under anaesthetic (Jonsson et al. 1989; Karrholm et al. 2000). It is suited to subjects having a

procedure such as arthroscopy, but difficult to ethically justify in healthy or conservatively managed subjects. RSA exposes the subjects to high doses of ionising radiation, which increases risk, particularly to young subjects. The tantalum beads are left in situ permanently and while the tantalum is an inert material, migration of the beads may be possible. RSA is limited to the acquisition rate of the films, usually 2-4 per second, which is quite slow for kinematic analysis (Brandsson et al. 2002). Fluoroscopy image acquisition, for example operates at 10 frames per second (Komistek et al. 2003) and optoelectrical and video systems at 60 per second (Hollman et al. 2002). Due to these constraints, RSA is limited to surgical populations, in small sample groups.

Magnetic Resonance Imaging (MRI)

Knee kinematics is amenable to study by MRI because it permits clear measurement from bony or soft tissue landmarks, the medial and lateral compartment kinematics can be analysed separately and 3-dimensional kinematic behaviour of the knee can be reconstructed from the layers of images. For example, the ability to acquire images along any plane enables images to be selected through the medial compartment of the knee or aligned with any chosen plane or axis. It is non-invasive, involves no exposure to ionising radiation and is accessible.

MRI is of low risk to subjects (Shellock and Kanal 1998; Westbrook 2002). Subjects are exposed to three forms of electromagnetic radiation: a static magnetic field, gradient magnetic fields and radio frequency electromagnetic fields (Shellock and Kanal 1998). It is necessary to exclude subjects with ferromagnetic implants in soft tissues that have the potential to become dislodged (such as some earlier aneurysm clips and shrapnel) and electrically or magnetically

activated implants (such as pacemakers or cochlear implants) (Shellock and Kanal 1998). While MRI is not believed to be hazardous to the developing foetus, safety committees in the UK, USA and Australia recommend not using MRI in the first trimester, unless there is a clear risk-benefit advantage (Shellock and Kanal 1998; Westbrook 2002). Anxiety may be encountered in 5-10% of patients referred to MRI, because of the restricted dimensions of the magnetic core and the noise generated (Shellock and Kanal 1998). This anxiety can be alleviated by maintaining verbal or physical contact with the subject, calm music, clear explanation of the procedure and good lighting. Bearing in mind these precautions for its use, MRI remains a useful clinical and research tool.

Traditionally, diagnostic MRI has required the patient to be prone or supine to enter the magnetic core (Todo et al. 1999). Recently interventional and open field MRI have been developed to create a vertical open space approximately 450mm wide, between two magnetic coils. This enables a subject to stand or perform activities such as a standing squat or a step up within the MRI field (Hill et al. 2000). The flexibility of MRI for imaging particular tissues and possibilities of positioning in open field MRI has useful applications to study of knee kinematics. However, the subjects must remain still during image acquisition. This is the greatest limitation of MRI for kinematics at present.

Image acquisition is becoming faster as new sequences are written for imaging. However, the fastest sequences still have an acquisition time of approximately 30 seconds. These sequences were developed to image tissues such as the liver, which are influenced by motion of the diaphragm so that image acquisition can take place in a breath hold (Behrens and King 2000). MRI of the moving knee will be the next advance (Rebmann and Sheehan 2003). The method

is being developed whereby a tracking device maintains the scanning plane and a single slice image is acquired at a rate of 20 per second (Niitsu et al. 1990; Niitsu 2001). This technique still has problems in terms of image resolution, reliability of the plane of the acquired slice and difficulty in handling out of plane motion, because a single 2-dimensional image is generated using this method. It may be possible to generate 3-dimensional images of the moving knee with reliable precision in the future, but image resolution and motion artefact on the images are presently a problem (Rebmann and Sheehan 2003). Once these issues are resolved, MRI will be able to image true kinematics, rather than a series of still shots.

Summary of findings of kinematics of the ACL-deficient knee in vivo

Examination of ACL-sectioned knees in vitro demonstrated increased anterior translation and internal rotation under loads. This laxity was also seen in vivo in subjects performing a standing lunge onto the injured leg (Friden et al. 1993). Furthermore, increased anterior translation was correlated with subjects' complaints of instability. During a step up exercise increased anterior tibial translation and internal rotation of the tibia on the injured side were recorded by electrogoniometry (Vergis and Gillquist 1998). Electrogoniometry has also demonstrated that tibial translation during open chain is greater than during closed chain exercises, enabling safer exercise prescription (Yack et al. 1994; Kvist et al. 2001).

The kinematic behaviour of the lateral compartment seems to be more affected by ACL deficiency than the medial compartment (Brandsson et al. 2001), which also suggests that the ACL has a role in rotary stability.

In one of the earliest RSA studies, Karrholm et al (1988a) studied knee flexion in prone subjects with x-rays acquired at 2-4 frames/second. They found that the ACL-deficient knees exhibited less internal rotation as the knee flexed than the intact knees (by 2.6°). Karrholm et al (1988a) also found the ACL-deficient knee exhibited greater posterior translation of the tibia during knee flexion than the intact knee. Translation was measured from the intercondylar eminence, as referenced from images of the knee taken with the subject relaxed and supine in knee extension. This appears to be counterintuitive, that the tibial intercondylar eminence of the ACL-deficient knee would be posterior to the intact knee. However, the interpretation could be in the semantics: Karrholm et al (1988a) are not referring to the anterior tibial translation laxity of the ACL-deficient knee, but to the motion of the tibia referenced to the femur as the knee flexes, that used to be called roll-back. Karrholm et al (1988a) concluded that the ACL injury caused a multiplanar instability that had previously been unrecognised, with components of instability in the adduction/abduction rotations, medial and lateral translation and anteroposterior planes.

This multiplanar instability was recognised by Brandsson et al (2001), in their analysis of ACL-deficient subjects performing a step-up. They found the tibia was more posteriorly displaced and externally rotated on the injured side. They analysed the medial and lateral femoral condyle position separately and found that the lateral femoral condyle of the injured side was displaced more anteriorly. Conversely, the medial femoral condyle was unchanged from

the intact side. Thus, the ACL deficiency had more effect on the kinematics of the lateral compartment than the medial compartment.

Tibiofemoral contact point behaviour has been described in the ACL-deficient knee in only one study of ten subjects, who demonstrated a wide variation of individual contact patterns (Dennis et al. 1996). In some subjects the femur was posterior on the tibial plateau in extension compared to normal knees and then paradoxically rolled forward during knee flexion. In other subjects the contact began in the normal position on the tibial plateau (quite anterior) and then rolled back during early knee flexion and forward again to 90° knee flexion. Other subjects had a more normal contact pattern. This fluoroscopic analysis of walking kinematics was limited by 2-dimensional information and could not report on rotations in the knee.

It appears that ACL-deficient subjects walking and ascending stairs avoid using their quadriceps muscles at heelstrike (Berchuk et al. 1990). This analysis of the kinematics and kinetics of gait was achieved by combining biplanar video and force platform data. “Quadriceps avoidance gait” appeared logical since quadriceps muscles exert an anterior drawer at the knee which ACL-deficient subjects may be attempting to minimise in their gait. Beard et al (1996) reproduced this experiment using EMG in addition to video and force platform data and found that the quadriceps were working in the same manner in the ACL-deficient and healthy knees, but in the ACL-deficient knees the onset of hamstrings activity was earlier and with activity greater, resulting in a net flexion moment at the knee, protecting the knee from anterior translation (Beard et al. 1996). This increased hamstrings activity has

been linked to the ability to dynamically stabilise the ACL-deficient knee (Steele and Brown 1999; Fitzgerald et al. 2000; Chmielewski et al. 2001; Rudolph et al. 2001).

Kinematics of jumping and landing using video, EMG and force platform data showed subjects with ACL-deficient knees delay hamstring muscle activation so that peak hamstring activity is more synchronous with initial contact and with the high impact and shear forces that occur after initial contact (Steele and Brown 1999). The delayed hamstring activation was proposed to be an adaptation developed to stabilize the injured limb against a giving-way episode via increased joint compression and posterior tibial drawer. Jumping was also examined using intracortical pins (Ramsey et al. 2001). In this study of six subjects, one subject lacerated the quadriceps muscle and bent the intracortical pin through the force of his quadriceps muscle contraction and in another subject the data were corrupted. The remaining four subjects did not provide an adequate sample size for any conclusions beyond a descriptive comment on the wide variation in kinematics among subjects.

However, this study was useful in demonstrating the wide variation in kinematics among subjects with ACL deficiency and the small magnitude of change produced by an intervention (in this case application of a Donjoy knee brace: dj Orthopaedics, Vista, California). In kinematic analysis it is very useful to use the contralateral knee as a healthy control, due to the high between-subjects variation, but low intra-subject variation (Smith et al. 1999). However, in a highly invasive method such as the use of intracortical pin markers, discomfort and risk to the subject are increased if both legs are incised and pinned. Consequently, motion analysis using intracortical pins has never been performed bilaterally.

Clinical examination of ACL deficiency

Diagnosis of ACL deficiency in the clinical setting has been founded on the features of anterior and rotary instability in the knee. The three most commonly performed clinical tests for the diagnosis of ACL deficiency are the anterior drawer at 90° knee flexion with the addition of internal or external rotation, the anterior drawer at 30° knee flexion (the Lachman test) and the pivot shift test.

The anterior drawer test is performed by applying a manual anterior force to the proximal tibia with the knee at 90° flexion. Addition of internal or external rotation may give added information regarding rotational laxity (Sandberg et al. 1986). Sensitivity of the anterior drawer test in the alert patient has been reported as 41% (Harilainen 1987). The anterior drawer test may be more sensitive in the chronic ACL-deficient knee or knee with injuries in addition to the ACL tear. The laxity of the knee in flexion may become more pronounced as the secondary restraint structures of anterior stability are lost (Malanga et al. 2003).

Pivot shift is both a clinical symptom, described by the subject as causing the knee to give way and a physical sign that can be elicited on examination (Malanga et al. 2003). The knee is held passively in flexion and a valgus and internal rotation force is manually applied, causing the lateral tibial plateau to sublux anteriorly (Galway and MacIntosh 1980; Lucie et al. 1984). The knee is passively extended until at approximately 30° the displaced tibia reduces (Galway and MacIntosh 1980). The pivot shift can also be demonstrated in vitro (Tamen and Henning 1981; Matsumoto 1990). Quantification of the pivot shift using an electrogoniometer demonstrated the pivot shift in stable knees post reconstruction and the subjects' contralateral

asymptomatic knees (Gillquist and Messner 1995). The electrogoniometer appeared to be very sensitive in measuring the pivot shift or anterior subluxation of the tibia during a rotation-extension manoeuvre. Disadvantages of the pivot shift are that it is uncomfortable for the subject, who may find it difficult to relax and prevent the pivot occurring. The sensitivity is low (0.18 to 0.48) but specificity is high (0.97 to 0.99), meaning that it has good predictive value (Scholten et al. 2003). The pivot shift is most reliable in anaesthetised subjects (Sandberg et al. 1986).

The Lachman test has better sensitivity (0.63 to 0.93) and reasonable specificity (0.55 to 0.99) (Scholten et al. 2003). The Lachman test is performed by applying an anterior force to the proximal tibia with the knee at 30° flexion (Torg et al. 1976). When performed manually, internal and external rotation can be applied to the knee to assess rotary components of instability. An instrumented quantification of the Lachman test has been developed, using a device called the KT1000 (Daniel et al. 1985b). When an anterior drawer is applied to the tibia, the displacement of the tibial tubercle is measured, a difference of 3mm or greater between the injured and contralateral knee is considered indicative of ACL injury with a specificity of 0.85 (Daniel et al. 1985a). Advantages of the Lachman test are its simplicity and comfort for the subject. One final interesting consideration is that measured passive anterior laxity of the knee has no association with dynamic instability (Friden et al. 1993; Sernert et al. 1999; Tyler et al. 1999; Patel et al. 2003). The dynamic stabilisation of the knee is produced by neuromuscular coordination and may be highly effective in stabilising the knee during active movements, despite substantial passive laxity of the knee (Steele and Brown 1999; Chmielewski et al. 2001; Rudolph et al. 2001).

These clinical tests for ACL deficiency aim to test the anterior and rotary components of instability permitted in the ACL-deficient knee. The anterior laxity of the knee is best to examine and to quantify at 30° knee flexion, with the subject relaxed, using the Lachman test. The rotary components of instability are demonstrated clearly by the pivot shift, but it may be best used with restraint as a diagnostic tool.

Kinematics of the osteoarthritic knee

There are some clear relationships between changes to the cartilage, bone and soft tissues of the knee and the altered kinematics exhibited. The standing alignment of the knee has been linked to the areas of wear on the tibial plateau (Harman et al. 1998). Wear and bony remodelling changes the shape of the knee, causing varus or valgus malalignment. As the medial compartment collapses the knee drops into varus alignment, especially at 30° knee flexion, when the femur is sitting in the deepest wear cupola of the medial tibial plateau. In a study of 173 tibial plateaus collected during TKA, wear-patterns of the ACL-intact varus knees occurred on the anteromedial aspect of the medial tibial plateau (Harman et al. 1998). Wear patterns of the ACL-deficient varus knees were posterior on the medial tibial plateau. The ACL-deficient knees showed a wear pattern that was consistent with posterior femoral subluxation and posterior tibial contact observed after ligament injury. The varus and valgus

knees showed wear at the medial and lateral compartments respectively and the ACL-deficient knees showed wear more posteriorly on the tibia plateau (Harman et al. 1998).

Bone and cartilage changes are also demonstrated by the joint space width appearing greater in x-rays in knee extension than in weight-bearing films at 30° flexion (Buckland-Wright et al. 1995). In healthy knees in knee extension, the femur is positioned above the anterior tibial plateau (Todo et al. 1999). As the knee flexes the femur rolls a little more posteriorly. Joint space width is narrowest on x-rays when the knee is flexed at 30° because the area of cartilage thinning is greatest in this tibiofemoral contact area (White et al. 1991). From this it could be surmised that the joint space width is wider in knee extension, narrower at 30° and widens again as the femur moves posteriorly out of the area of deepest wear in knee flexion. However, there is no research to describe the tibiofemoral contact pattern in the osteoarthritic knee, only the position of the femur in healthy knees (Todo et al. 1999; Hill et al. 2000; Iwaki et al. 2000). It could be that the bone and cartilage remodelling that occurs in knee osteoarthritis changes the tibiofemoral contact pattern.

Remodelling of the bone and cartilage also changes the degree of laxity in the osteoarthritic knee. Disease progression has been linked to laxity, especially varus/valgus laxity associated with varus/valgus malalignment (Maquet 1976). As the medial compartment narrows, for example, the knee develops a varus malalignment, which increases the transmitted load through the medial compartment. Increased load bearing in turn increases the wear on the medial compartment. In a longitudinal study of 230 subjects the risk of disease progression increased four-fold when medial compartment disease was associated with varus alignment and risk of disease progression was increased five-fold when lateral compartment

osteoarthritic was associated with valgus alignment (Maquet 1976; Sharma et al. 2001). One of the solutions to this in the past was high tibial osteotomy, which is able to correct the varus alignment and spare the medial compartment from some of the load-bearing by resecting a bone wedge from the tibia (Akamatsu et al. 1997). This intervention delayed the progression of osteoarthritis in the knee (Maquet 1976). The unicompartmental knee replacement is now frequently used in place of osteotomy, restoring the normal joint space width to reduce weight bearing through the medial or lateral compartment as appropriate (Callaghan et al. 2000). It remains unclear, however, if the valgus laxity is present before the medial compartment osteoarthritis or is a result of the osteoarthritis.

Varus/valgus laxity has been measured in healthy and age matched control subjects and both knees of osteoarthritic subjects in order to examine whether increased laxity is the cause or the result of the unicompartmental wear. In a cross-sectional study of 69 control subjects and 169 osteoarthritic subjects, the varus/valgus laxity increased without evidence of osteoarthritic change and increased with the severity of the disease (Sharma et al. 1999b). It has been suggested that laxity contributes to disease progression (Wada et al. 1996; Sharma et al. 1999b). Anterior-posterior (AP) laxity and rotational laxity, however, do not seem to be proportional to the severity of the disease. AP laxity is highest in the contralateral knee of osteoarthritic subjects and those with very early osteoarthritic changes, it then decreases in severe osteoarthritis, but does not return to normal age-matched levels (Brage et al. 1994). This pattern is similar for internal and external rotation laxity (Brage et al. 1994; Wada et al. 1996; Sharma et al. 1999a). Another interesting finding from Wada et al (1996) was that although most of the subjects had ACL deficiency, they also had decreasing AP laxity as the severity of the disease progressed. They concluded that the capsular stiffness and osteophyte

growth restricting the passive laxity of the joint was able to override the laxity due to ACL deficiency. These studies all measured passive laxity of the knee; kinematics studies can confirm whether this passive varus/valgus laxity and AP and rotational restriction are apparent in active knee motion.

Range of motion demands of the osteoarthritic knee, measured with electrogoniometry, showed that osteoarthritic subjects used less of their available knee flexion range than normal controls performing the same activities (Walker et al. 2001). Functional range did not correlate with available range, suggesting that subjects limited joint excursion due to pain on activity. Gait analysis has also found that osteoarthritic knees exhibit decreased flexion range during gait (Messier et al. 1992; Al-Zahrani and Bakheit 2002). These findings may be dependent on severity of the disease, however, as in early osteoarthritis, magnitude of flexion range used to ascend and descend stairs and for normal gait was not found to be different (Kaufman et al. 2001). The range of motion used by the osteoarthritic knee may be limited as much by pain as structural limitations (Gok et al. 2002).

Pain may be responsible for some other characteristics of gait in osteoarthritic subjects, including reduced flexion and adduction moments measured through a force platform during gait analysis (Hurwitz et al. 1999; Kaufman et al. 2001). The reduced external flexion moment recorded during stance phase in osteoarthritic knees is an indication of reduced net quadriceps activity (Hurwitz et al. 1999). Osteoarthritic subjects walk and descend stairs with reduced quadriceps activity either because the quadriceps are weak or the subjects are attempting to reduce joint compression loads by reducing quadriceps activity (Hurwitz et al. 1999; Kaufman et al. 2001). Patients with medial compartment osteoarthritis also demonstrate an increased

adduction moment during stance phase of gait (Hurwitz et al. 1998; Andriacchi et al. 2000; Baliunas et al. 2002). The increased adduction moment is related to the severity of the disease, causing an increased load through the medial side of the joint (Sharma et al. 1998) and therefore also a higher ratio of medial to lateral bone density (Madsen et al. 1994; Hurwitz et al. 1998). The adduction moment at the knee is also related to joint pain: patients in pain reduce the adduction moment and patients given non-steroidal anti-inflammatory or analgesic medication show an increased adduction moment (Hurwitz et al. 2000). Since a greater adduction moment is associated with disease progression, Hurwitz et al question the wisdom of prescribing analgesics to patients with medial compartment osteoarthritis (Hurwitz et al. 1999). Thus kinematics of the osteoarthritic knee is a function of not only the structural changes, ligament laxity or alignment of the knee, but also the requirements of patients to avoid pain.

The tibiofemoral contact points in the osteoarthritic knee have not been studied. Two studies explored joint loading patterns (Fukubayashi and Kurosawa 1980; Riegger-Krugh et al. 1998), but these were both cadaver studies that limited the research question to loading in knee extension, not tibiofemoral contact patterns during knee flexion. Two studies found a loss of coupled rotation of the knee during active flexion and extension (Koga 1998; Nagao et al. 1998) and loss of screw-home in osteoarthritis. Indeed, in severe osteoarthritis the knee may even internally rotate in terminal extension (Koga 1998). Koga et al (1998) relate this loss of rotation to the area of wear observed on the tibial plateau: the larger the area of wear, the less rotation present. However, it is not possible to examine the relationship between tibiofemoral contact patterns and wear from this study. Further research is required to map the tibiofemoral

contact patterns in the osteoarthritic knee and examine the relationship between contact patterns and wear.

Measurement of bone mineral density by Dual Energy X-ray Absorptiometry

Measurement of bone mineral density (BMD) is integral to this thesis because it measures the subchondral sclerosis that is an indicator of early osteoarthritis in the knee. BMD can be used, not only to compare osteoarthritic subchondral bone to healthy bone, but also to compare medial and lateral compartment ratios that indicate the presence of unicompartmental osteoarthritis (Madsen et al. 1994). In animal studies, BMD has increased at the same time or even earlier than the first detectable articular cartilage changes (Bailey and Mansell 1997; Pastoureaux et al. 1999). In particular, changes to the articular cartilage that are evident on MRI are indications of already quite advanced osteoarthritic changes (Balkisoon 1996; Blackburn et al. 1996; Disler et al. 2000; Murphy 2001). Bone density measurement has the additional advantage of being possible through dual energy x-ray absorptiometry (DEXA), which is minimally invasive.

Very early osteoarthritic changes are often examined by histology. Since this thesis includes ACL-injured and osteoarthritic subjects who are managed conservatively, surgical retrieval and histology of subchondral bone sections is not possible. Subchondral bone changes may be

the earliest indicator of osteoarthritic change in the knee and these can be measured by radiography (Buckland-Wright et al. 2000)

Non-invasive methods of measuring BMD include dual photon absorptiometry (Bohr and Schaadt 1987; Petersen et al. 1996a), quantified computerised tomography (QCT) (Odgaard et al. 1989; Hans et al. 1997; Majumdar et al. 1997) fractal signature analysis of trabecular bone (Lynch et al. 1991; Majumdar et al. 1993; Buckland-Wright et al. 1994; Buckland-Wright et al. 1996) and DEXA (Casez et al. 1994; Genant et al. 1994; Mottet et al. 1996; Link et al. 2000; Petley et al. 2000; Abrahamsen et al. 2001; Cure-Cure et al. 2002).

Dual photon absorptiometry was developed in order to assess osteoporosis in individuals at risk of osteoporotic fractures (Hans et al. 1997). Photons are emitted by an injected radioisotope, gadolinium 153. Bone and soft tissue absorb the photon energy at different levels and relative density can be calculated (Ott 1998). Disadvantages of this method are accuracy of only 4-6% and the use of radioisotope, which decays fast and makes for poor test-retest reliability. It is also very expensive (Moyer-Metzler and Daniels 1999).

QCT is highly accurate and reliable (Hans et al. 1997). It can be used to measure small regions of interest and it is three-dimensional (Majumdar et al. 1997; Muren et al. 2001). Because it is three-dimensional, it is able to measure cortical bone independently of cancellous bone, which the two-dimensional absorptiometry methods cannot (Moyer-Metzler and Daniels 1999). The disadvantage of QCT is the level of radiation exposure. It is estimated that worldwide CT made up 5% of radiological examinations in 2000, but made up 34% of the collective radiation dose (United Nations Scientific Committee on the Effects of Atomic Radiation 2000). For

example, a skull x-ray delivers a radiation dose of 0.07 mSv, whereas a cranial CT delivers a dose of 2.3mSv (European Commission 2000).

Fractal signature analysis of trabecular bone enables details of trabecular structure to be examined, for example, comparison of vertical with horizontal trabecular density may be relevant in increased load bearing responses (Lynch et al. 1991; Majumdar et al. 1993; Buckland-Wright et al. 1994; Buckland-Wright et al. 1996). To image the fine detail of trabecular bone either a digitised image taken from a microfocal radiograph or an MRI is used. The digital data is analysed using a fractal pattern. This method is not widely used outside of specialised research institutions (Lynch et al. 1991).

The most commonly used method for assessment and monitoring of osteoporosis is DEXA (Casez et al. 1994; Petley et al. 2000; Iki et al. 2001; Cure-Cure et al. 2002). It is non-invasive and the technology is widely available. Repeatability is reported to be 0.5 – 2%, but accuracy in measuring the true BMD is 5-10% (Szucs and Jonson 1992; Genant et al. 1994; Blake 1996; Hans et al. 1997). Possible sources of inaccuracy include inconsistency in measurements recorded using different machines (Genant et al. 1994), measurements calibrated against phantoms may not represent accuracy in vivo (Blake 1996) and regions of interest that are small may result in heterogeneous areas of tissues being sampled by the scanner and lead to low repeatability (Szucs and Jonson 1992). Despite these limitations, the availability and low radiation dose make DEXA popular for measurement of BMD.

The principle indication for DEXA is the diagnosis and monitoring of osteoporosis, not knee research (Moyer-Metzler and Daniels 1999). The sites measured are sites prone to

osteoporotic fractures, i.e. the hip, spine and forearm. Techniques and computer programs designed for analysis of DEXA data are designed to sample tissues at these sites. Computer programs have been designed to analyse the x-ray absorption by the tissues in the field of the scan, including bone, muscle and fat (Ott 1998). Programs developed for analysis of forearm scans also include samples of air in the scan field. Different tissues absorb x-ray energy generated by the scanner at different rates and the different absorption of the tissue samples is compared to calculate bone mineral density. The knee is a narrow structure, so the region included in a knee scan will include samples of bone, muscle, fat and air. Forearm programs are suitable for scanning the knee, as air will be included at either side of the scan field (Murphy et al. 2001). Spine or hip programs may be used, but air must be excluded from the scan fields by placing rice bags or other tissue substitutes, beside the knee. The DEXA programs were not designed for analysing data from the knee, so reliability issues needed to be addressed to ensure the analysis system compares sampled tissue and sampled bone regions appropriately.

Reliability of BMD measured at the knee by DEXA has been tested by several authors (Sievanen et al. 1992; Murphy et al. 2001; Nilsson 2001). A comparison was made between dual photon absorptiometry and DEXA at the knee, using a Norland DEXA scanner (MkII, Norland Corp, WI, USA), (Petersen 2000). Petersen reported that DEXA measurement in vivo at the proximal tibia had a coefficient of variation (CV) of 0.8% and at the distal tibia CV 1.4%, in 8 subjects scanned 3 times in one week. When smaller regions were measured in the subchondral trabecular or cortical bone the precision was lower: 3.4 – 5.2% (Petersen et al. 1996a). Using an Hologic DEXA scanner (Hologic, MA, USA) inter- and intra-observer repeatability of measurement at the proximal tibia were 2.9% and 2.8% respectively, but this

was repeat analysis of data from 20 subjects scanned once and analysed three times (Hulet et al. 2002). In the distal femur, the measurement of BMD was found to have a CV of 3.1 – 3.7% in TKA patients (Lui et al. 1995). Using a Lunar (GE, USA) DEXA scanner, the precision was found to be lower: 3.2 – 16.9% at the proximal tibia (Nilsson 2001). One of the problems scanning the knee is the insufficient soft-tissue sample for comparison with bone. The soft tissue sample can be supplemented by substitutes such as rice bags placed beside the knee or a plexi-glass rod in the detector opening. There appears to be a range of precision values reported by researchers, ranging from that quoted for the spine and hip, of 0.5 – 1%, to 0.7 – 16% around the knee. It is likely that the computer programs for analysis of DEXA are less reliable at the knee, than at the sites for which they were designed, but are nevertheless sufficiently reliable to enable research into BMD at the knee.

Research has demonstrated that BMD changes occur in at least three conditions affecting the knee: ACL reconstruction, knee osteoarthritis and TKA. A single case study showed BMD after knee injury and reconstruction fell by 17.4% at the proximal tibia and by 18.5% at the distal femur (Sievanen and Kannus 1994). BMD reached the lowest point 15 weeks post reconstruction and was still reduced by 10-14% at 12 months. Findings were similar in a larger (n=33) ACL-injured group (Leppala et al. 1999). Conservatively managed subjects were found to have less marked BMD loss than surgically managed subjects. The difference between the BMD loss in surgical and conservatively managed ACL-injured knees was probably not due to kinematic changes in the initial 12 months inducing bone remodeling, but rather to surgical management, including chemical factors involved in inflammation and surgery and disuse of the operated limb (Leppala et al. 1999).

The bone mineral density of knees with osteoarthritis is higher where there is sclerosis of the subchondral bone (Petersen et al. 1996b; Pastoureaux et al. 1999). This increase in bone density due to sclerosis is often countered by a decrease in bone density due to pain avoidance decreasing the mechanical load through the knee (Hurwitz et al. 2001). It is possible to see lower bone mineral density in osteoarthritic knees than in age matched control subjects, particularly in very early symptomatic osteoarthritis (Karvonen et al. 1998). However, bone density changes in osteoarthritic knees reflect what is happening at the joint. Increase in BMD due to subchondral sclerosis is proportional to the severity of the osteoarthritis (Madsen et al. 1994). Also the relative density of the medial to lateral compartments change as the alignment of the knee changes, reflecting the increased loading in one compartment and load-sparing in the other (Madsen et al. 1994; Hulet et al. 2002). The progress of unicompartmental knee osteoarthritis can be delayed by performing high tibial osteotomy (Maquet 1976). The osteotomy corrects the alignment of the knee (or over-corrects) and mechanically unloads the effected compartment. At one year following high tibial osteotomy the ratio of medial / lateral BMD at the knee had fallen significantly (Akamatsu et al. 1997). However, in this study it is not possible to isolate the effects of the high tibial osteotomy in mechanically unloading the medial compartment, from BMD changes due to osteoarthritic subchondral sclerosis, because both factors may be involved. It is not possible from this study of BMD alone, to say the osteoarthritic had been reversed by osteotomy. The value of measuring BMD at the subchondral regions of the knee in osteoarthritis is in monitoring medial to lateral compartment ratios in unilateral osteoarthritis, in detecting changes due to disease progression and changes due to therapeutic interventions to unload the knee joint.

In joint arthroplasty, BMD is measured as an indicator of events happening at the prosthesis-bone interface. Early post-operative BMD loss has been seen following TKA (Li and Nilsson 2000a). The subsequent study investigated whether this early BMD loss was related to migration of the prosthesis into the subchondral bone (or subsidence of the subchondral bone) (Li and Nilsson 2000b). Fixation of the prosthesis (either using bone cement or hydroxyapatite coated stems that are uncemented) may be influenced by the reaction of the bone. Bone mineral density has been studied in the peri-prosthetic regions of the femur and tibia as an indicator of bone resorption or formation, that may influence fixation (Lui et al. 1995). Much of the testing of the reliability of BMD testing at the knee was performed by the research teams investigating peri-prosthetic bone quality in TKA (Bohr and Schaadt 1987; Lui et al. 1995; Li and Nilsson 2000a; Murphy et al. 2001; Nilsson 2001; Shahid et al. 2001).

In this thesis bone mineral density is used to indicate changes to the structure of the bone that may be indicative of early osteoarthritis (Boyd et al. 2000; Shahid et al. 2001). The BMD at the knee in recently ACL-injured knees can be compared to those knees injured many years ago, in order to examine the effects of chronic ACL deficiency on structural changes of the subchondral bone. Subchondral BMD can also be measured in those subjects with established osteoarthritis of the knee. Thus the changes to the density of the subchondral bone may be examined at several stages of knee degeneration.

Structure of the thesis

This thesis includes seven studies, each of which may be read independently. The studies examine the kinematic consequences of ACL deficiency. The kinematics of osteoarthritic knees are examined representing the end stage of degeneration of the knee. The changes in kinematics of the knees are compared to the wear and degeneration observed in the knees.

The list of references cited in the thesis is to be found at the end of the thesis. The exception to this is where the published manuscript is included as a chapter, then the references are included at the end of that manuscript and also at the end of the thesis.

CHAPTER 4

EVALUATION OF A METHOD TO MAP TIBIOFEMORAL CONTACT POINTS IN THE NORMAL KNEE USING MRI.

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Statement from co-authors confirming the authorship contribution of
the PhD candidate

"As co-authors of the paper "*Evaluation of a method to map tibiofemoral contact points in the normal knee using MRI*" we confirm that Jennifer M Scarvell has made the following contributions:

- Contributed to discussions on design of the study
- Collection, analysis and synthesis of literature
- Submission for ethics approval, recruitment of subjects, and collection of data under supervision
- Analysis and interpretation of data under supervision
- Wrote the first draft of the manuscript, and followed through to publication, including proofing and final publication details of the manuscript."

Professor Kathryn M Refshauge Signed:  Date:.....

Associate Professor Paul N Smith Signed: Date:.....

Dr Howard R. Galloway Signed: Date:.....

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Ms Jennifer M Scarvell Signed: Date:.....

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Evaluation of a method to map tibiofemoral contact points in the normal knee using MRI

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Abstract

A technique using Magnetic Resonance Imaging (MRI) is proposed for analysis of knee motion that is practical in the clinical situation. T1 weighted fast spin echo (FSE) and spoiled gradient echo (GE) sequences were compared to image both knees at 15° intervals from 0° to 90° flexion, while unloaded and loaded. The medial and lateral tibiofemoral contact points were mapped reliably using both FSE sequences and GE sequences with intra-class correlation_(2,1) of 0.96 (CI 99% = 0.94–0.97) and 0.94 (CI 99% = 0.91–0.97), respectively. Results were consistent with the current literature on knee motion: the medial and lateral tibiofemoral contact pathways were different ($F_{1,80} = 253.9$, $p < 0.0001$) reflecting the longitudinal rotation of the knee, the loaded and unloaded knees were not different in the healthy knee ($F_{1,80} = 0.007$, $p = 0.935$), and the left and right knee were consistent for each individual ($F_{1,80} = 0.005$, $p = 0.943$). Therefore, right to left differences may be attributed to pathology. MRI analysis of knee kinematics as described by this technique of tibiofemoral contact point mapping provides a robust and reliable method of recording the tibiofemoral contact pattern of the knee.

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Keywords: Knee; Magnetic resonance imaging (MRI); Knee motion; Knee physiology

Introduction

Detailed understanding of normal knee kinematics is essential for both conservative and surgical treatments aimed at restoring kinematics after knee injury or disease. Knee ligament injuries are common, with an incidence of at least 31 per 100,000 population per year [13], and have significant implications in terms of subsequent loss of function, and degenerative changes in the injured knee [5,9]. Altered knee kinematics are thought to contribute to the development of articular cartilage pathology [4,8]. A method of analysing knee kinematics in the clinical situation will provide accurate data for surgical decision-making and outcome studies.

Magnetic resonance imaging (MRI) has advanced the study of knee kinematics by enabling a three-dimensional analysis of movement with great accuracy [17,18], permitting separate analysis of the medial and lateral compartments of the knee and accurate alignment with

the axes of motion. Open field MRI systems have been used to perform three-dimensional Fourier transformation gradient echo sequences for knee kinematic study pertaining to the motion of the menisci [1]. The tibiofemoral kinematics between 15° and 90° has been described from analysis of T1 gradient echo images [18], importantly describing the mediolateral asymmetry of motion which demonstrates the longitudinal rotation of the knee during flexion. The study was limited by small sample size and effect size. However, the findings have since been reproduced in cadaveric studies [12,15] and in vivo [10,11,14]. Access to open field MRI systems, however, is not widely available in clinical practice.

Knee kinematics can be described by the movement of the centres of the posterior femoral condyles, or by tibiofemoral contact mapping. The movement of the femoral condylar centres is particularly relevant to design of total knee arthroplasty prostheses, as knee prosthesis designs aims to replicate the normal motion of the knee and the malalignment of the axis of motion has been linked to loosening of the prosthesis [20]. Tibiofemoral contact patterns, in contrast, can be used to describe events at the tibiofemoral interface that may

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contribute to articular cartilage damage and wear, such as altered roll/glide characteristics, sheering and instability [6,19].

This study has established a technique for kinematic analysis of knee motion by tibiofemoral contact mapping, achievable in the clinical situation, using a readily available 1.5 T closed field MRI unit and a fast gradient echo sequence that makes the technique practical for clinical use. A comparison is made between the T1 weighted fast spin echo technique which enables thinner slices but has longer scanning times and the T1 weighted spoiled gradient echo sequence, which is fast enough to take images in a breath hold [3]. Both sequences image bone well, but the gradient echo sequences lose some detail in imaging soft tissue. Thus, we have devised a technique that utilises available technology and is achievable in the clinic. This technique may be applied to investigate the impact of knee injury to the kinematics of the knee, the response to corrective surgery, or the effectiveness of rehabilitation to restore the dynamic stability of the knee.

Methods

Subjects

Twelve healthy subjects (seven male, five female, aged 20–50 years) were recruited to the study and provided informed consent. Subjects for whom MRI was contraindicated, were over 180 cm tall (to permit knee flexion within the tunnel), or with a history of knee symptoms were excluded. Approval to conduct the study was obtained from the Health Department and university ethics committees.

MRI procedure

Imaging of both knees was performed using a 1.5 T Siemens Magnetron Vision (Erlangen, Germany), using a body coil to generate parasagittal images, perpendicular to each tibial plateau.

Five subjects (one male, four female), were scanned using a fast spin echo sequence (FSE) (repetition time [TR]=660.0; echo time [TE]=12.0) with a field of view of 15–16 cm. Fourteen slices were obtained in each knee (slice width 7 mm). Scan time was 2.31 min, giving a complete session time of 1.5 h. These films were scanned and imported into Photoshop® version 5.0.2 (Adobe Systems Incorporated, San José, USA) as Tiff (tagged image format) files for analysis. The Photoshop® measuring tool was calibrated from scale printed on the films.

Seven subjects (six male, one female), were scanned using a spoiled gradient echo sequence (GE) [3]. Sixteen slices were generated (eight through each knee) approximately 10 mm apart (TR = 160.0, TE = 2.3/1, TA = 00:46), with a 256×256 matrix. Scans took 38 s each, with a complete session time of 35 min. These images were directly downloaded from the MRI machine as Dicom format files. The images were analysed using Osiris® software version 4.11 (Université de Genève, Switzerland).

A wooden frame was fitted to the MRI couch, to enable positioning and loading of the subjects' knees (Fig. 1). Elastic straps maintained neutral tibial rotation and thigh adduction. Images were taken of both knees, loaded and unloaded, at seven 15° intervals from full knee extension to 90° flexion.

Loaded knee images

Subjects were scanned at each knee flexion position twice: once while relaxed, and again pressing down against a footplate weighted

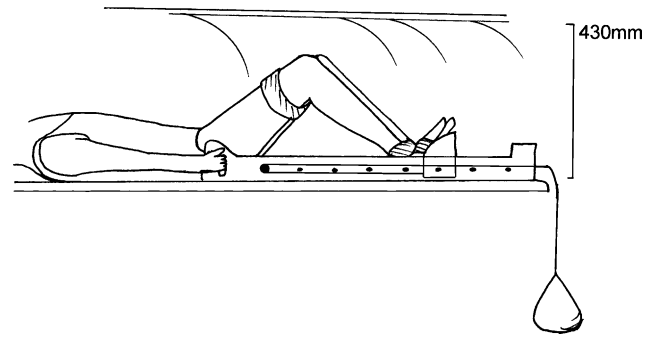


Fig. 1. Diagram of a subject performing a loaded leg-press in the MRI tunnel. Knee flexion angle was controlled by the position of a sliding wooden footplate. A 150 N weight and pulley provided resistance to knee extension.

with a sandbag imposing a force in line with the axial skeleton of 150 N. This results in a lower flexion moment at the knee at 0°, than at 90°. However, this increasing flexion moment at the knee with flexion is a normal physiological condition for the knee. This experimental design aims to simulate a leg-press, during which the flexion moment at the knee normally changes with knee flexion. Subjects were requested to press down firmly with the feet against the footplate, in order to prevent it sliding towards them. Exerting more than the required load would not result in movement of the footplate, but less than the required load would cause the footplate to slide. The load at the footplate therefore represents a minimum level: subjects could have exerted a greater load than required, particularly at 0° knee flexion.

One hundred fifty Newtons is a small load for the knee, which physiologically is capable of loads greater than bodyweight. However, it is difficult for a subject to remain still maintaining a maximal muscle contraction for the time required for imaging. To test the effect on knee kinematics of a greater weight, a comparison between a 150 and 250 N load was made in one subject. Tibiofemoral contact points were compared for the two conditions 150 and 250 N using a paired *t*-test.

Testing for image distortion

Gradient echo sequences were tested for evidence of distortion, since at 90° flexion the knees of the subject are close to the roof of the tunnel. A cylindrical specimen jar 41×50 mm filled with water was placed in the tunnel in six positions evenly spaced from the couch, to the roof of the tunnel, all just lateral to the centre of the tunnel. A seventh position was sited 150 mm proximal to the tunnel centre. The diameter, length and diagonal dimensions of the jar were recorded at each position. The diagonal dimension was 63 ± 0.9 mm. Dimensions of the jar were tested for correlation with proximity to the roof of the MRI tunnel, using a Pearson's correlation.

The anteroposterior (AP) dimension of the medial tibial condyle was recorded at each knee flexion position, for both knees of each subject imaged using GE. Tibial AP dimensions were tested for correlation with knee flexion to assess the influence of proximity to the tunnel roof on distortion, using a Pearson's correlation.

Tibiofemoral contact point measurement

The point at which the femur contacted the tibial plateau was recorded for the medial and lateral compartment of each knee at 15° intervals from 0° to 90°, in the loaded and unloaded knee. Measurement of the tibiofemoral contact point was referenced from the posterior tibial cortex to the centre of the tibiofemoral contact area (Fig. 2). This generates a motion pathway of contact point behaviour for each compartment representing the motion characteristics of the knee.

From the set of knee images for each subject at each knee position, an image was chosen which was closest to the centre of the medial and centre of the lateral compartment. This chosen image was used to record the tibiofemoral contact position. The tibial plateau is rounded, so AP dimensions are sensitive to the mediolateral positioning. For example, the distance from the posterior tibial cortex to the tibio-

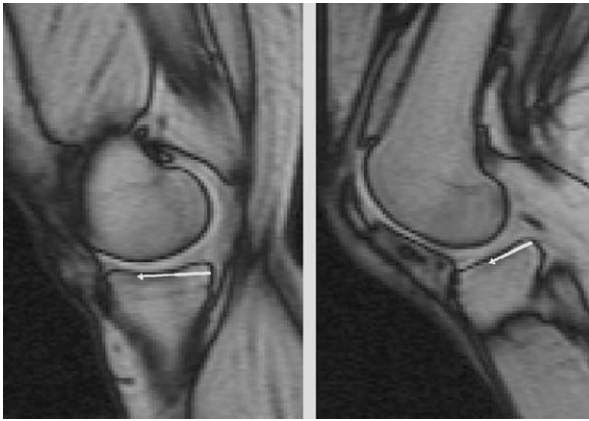


Fig. 2. Measurement reference points for tibiofemoral contact mapping. T1 weighted gradient echo sagittal MRI images through the medial compartment at 0° and the lateral compartment of the knee at 45°, demonstrating the posterior edge of the tibial cortex and the contact point of the femoral condyle (arrows).

femoral contact position is smaller if the chosen image is situated too far lateral in the knee. Slices were chosen to represent the centre of each compartment at each knee position from extension to flexion. Consistency in choice of image for analysis was tested by repeating observations on separate occasions more than a week apart.

Reliability of contact point measurement was tested by a single trained observer, blinded to previous results, repeating the measurement process on two occasions more than a week apart. This measurement process includes two possible sources of error: choice of slice and measurement error. The measurement reliability of FSE images scanned into Photoshop® was compared with the GE images, analysed using Osiris® software.

Controlling for variation in size of subjects

To account for variation in the size of subjects, tibial cortex to contact point distance measurements were scaled in proportion to mean tibial dimensions. The mean AP diameter of the medial tibial plateau was 50 ± 4.4 mm, and the lateral tibial plateau was 42 ± 2.17 mm. The distance recorded for each tibiofemoral contact point for each subject was then scaled proportionally to give a tibiofemoral contact position represented on a tibial plateau of standard dimensions.

Testing for construct validity

There is not a gold standard for analysis of knee kinematics with which to compare this method. The method was therefore tested for construct validity, by developing three statements based on contemporary kinematic research [11,12,14], and testing the results of this kinematic study against the kinematic behaviour predicted by the statements. The statements were:

1. That the contact patterns of the medial and lateral compartments of the knee would be different, reflecting the longitudinal rotation of the knee during knee flexion [11,12,14].
2. That the loaded and unloaded knee would not demonstrate a difference in contact pattern [11].
3. That the right and left knee would not demonstrate a difference in contact pattern [16].

Statistical analysis

The agreement between test re-test measurement of contact points from the knee images was determined by $ICC_{(2,1)}$ with 95% confidence

interval. This measure included reliability of choice of image for analysis, and of measurement of contact point distance.

To compare the right to left, loaded to unloaded knees and medial to lateral contact patterns, a repeated measures ANOVA design was used. Factors were the knee (left or right), the condition (loaded or unloaded), the compartment (medial or lateral), and the knee flexion angle. Significance was set at $p < 0.05$ for all parameters. Statistical analyses were performed using SPSS® version 9.1.

Results

Load

The heavier load demonstrated no significant difference in the tibiofemoral contact points of the knee ($p = 0.95$).

Distortion

There was no correlation between position of the jar, or its proximity to the roof of the tunnel, and the dimensions of the jar measured ($r = 0.003$, $p = 0.990$). None of the dimensions of the jar (width, depth or diagonal) demonstrated distortion.

The mean tibial AP dimension for subjects scanned using GE ranged from 51.5 ± 3.7 mm at 0° knee flexion to 49.8 ± 3.1 mm at 90° knee flexion. However, there was no correlation between knee flexion and tibial AP dimension ($r = -0.14$, $p = 0.13$).

Consistency of choice of image slice for analysis

Both FSE and GE images were analysed for consistency of choice of representative slice. Day to day reliability for FSE sequences was high, with $ICC_{(2,1)} = 0.99$ (CI 99% = 0.99998–0.99999). Representative images were also chosen with high reliability from the GE sequences, with an $ICC_{(2,1)} = 0.99998$ (CI 99% = 0.99998–0.99999).

Measurement reliability

The reliability of the tibiofemoral contact point measurement includes the effects of choice of image and measurement error incorporated in the image resolution and the accuracy of measurement with the Osiris® and Photoshop® software (Figs. 3 and 4). For FSE and GE scans measurement reliability was $ICC_{(2,1)} = 0.96$ (CI 99% = 0.94–0.97) and 0.94 (CI 99% = 0.91–0.97), respectively.

Construct validity

There was no significant difference demonstrated in the tibiofemoral contact pathway between the left and right knee ($F_{1,80} = 0.005$, $p = 0.943$), or between the loaded and unloaded knee ($F_{1,80} = 0.007$, $p = 0.935$).

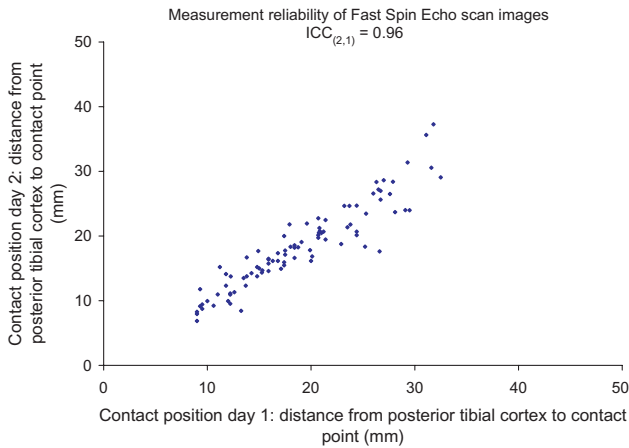


Fig. 3. Reliability of tibiofemoral contact point measurements, recorded from fast spin echo images on two occasions more than a week apart ($ICC_{(2,1)} = 0.96$).

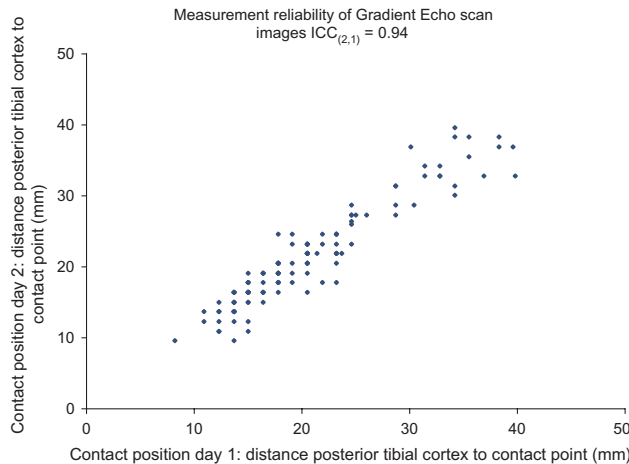


Fig. 4. Reliability of tibiofemoral contact point measurements, recorded from gradient echo images on two occasions more than a week apart ($ICC_{(2,1)} = 0.94$).

The tibiofemoral contact pathway in the healthy knee did demonstrate mediolateral asymmetry ($F_{1,80} = 253.9$, $p < 0.0001$). In knee extension, the medial femoral condyle rested further anteriorly on the tibial plateau than the lateral femoral condyle (the medial femoral condyle resting at mean 35 ± 4.0 mm and the lateral femoral condyle resting 25 ± 4.0 mm from the posterior edge) (Fig. 5). During knee flexion to 90° , the medial femoral condyle moved back on the tibial plateau 15 ± 2 mm to a location 20 mm from the posterior tibial cortex. The lateral femoral condyle, however, continued to move back on the tibial plateau to 12 ± 2 mm from the posterior tibial cortex at 90° flexion (Fig. 6). These findings are in agreement with the theoretical construct and therefore the technique has high construct validity.

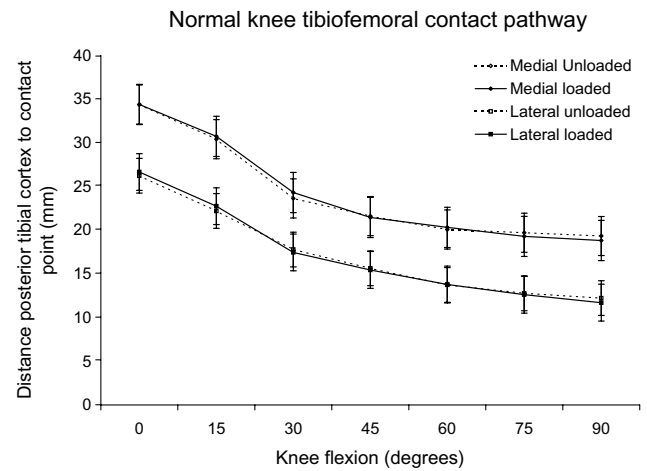


Fig. 5. Graph plotting the tibiofemoral contact points from 0° to 90° of knee flexion (mean \pm standard error). The similarity in the tibiofemoral contact pathway between the loaded and unloaded knee is demonstrated. The medial compartment shows a contact pattern located more anteriorly on the tibial plateau, than the lateral compartment.

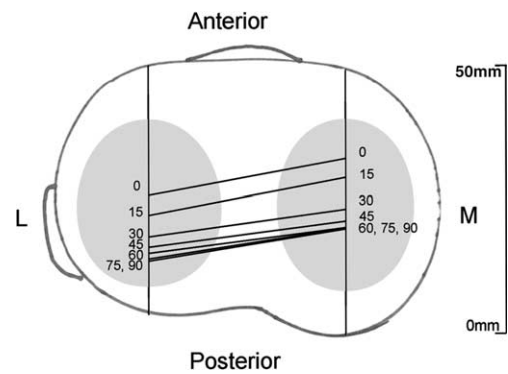


Fig. 6. Diagram of the tibial plateau, showing the tibiofemoral contact pattern from 0° to 90° of knee flexion, in the loaded knee. In both medial and lateral compartments, the femoral condyle rolls back along the tibial plateau from 0° to 30° . Between 30° and 90° the lateral condyle continues to move posteriorly, while the medial condyle moves back little.

Discussion

The study thoroughly tested this technique of kinematic analysis of the knee for practical application, reliability and construct validity. The FSE and GE sequences that were used during this study were both useful. GE sequences gave clearer definition of bony landmarks, in particular clear definition of the posterior tibial cortex reference point used for measurement, than FSE sequences, and little loss of image resolution at the magnification used for image analysis. FSE sequences gave better articular cartilage detail, hence making the tibiofemoral contact point more distinct, but less definition of the posterior tibial cortex reference point. Both sequences generated suitable slice intervals. The prac-

tical advantages of GE sequences were in the saving of time for the patient, staff and use of resources.

Precise measurement of the tibiofemoral contact point using MRI is complicated by the contact occurring over an area, not a point. This contact area is small in knee flexion and the centre point easily measured, but contact area widens in knee extension as the distal femoral condyle has greater congruence with the tibial plateau. Visual estimation of the contact point with reference to the centre of the articular contact area (seen on the MRI scans by distortion of articular cartilage and the position of the menisci) is prone to subjectivity. Wretenberg attempted to solve this by measuring the extremes of the contact area, and calculating the centroid [21]. In this study direct visualisation of the centre of the contact area provided accurate and reproducible measurement.

Description of knee kinematics with reference to the femoral condylar centres also has limitations. Todo et al. [18] superimposed a circle representing the posterior femoral condylar arc over the MRI image, in order to draw a line from the centre of the circle perpendicular to the tibial plateau, thus defining the position of the femoral condyle above the tibial plateau. This method is useful in knee flexion, when the posterior femoral condyle is in contact with the tibial plateau and the axis of the knee is through the posterior condylar centre. However, in knee extension the posterior condylar arc is not relevant as the axis of rotation of the knee has shifted to the extension facet centre [7]. Todo et al. did not study knee kinematics from 0° to 20°, and so did not face this limitation to their method [2,18]. Tibiofemoral contact mapping can be applied throughout the range of knee motion, including the terminal extension of the knee.

High construct validity of this technique is important for the clinical application of the technique in assessment of normal and injured knee kinematics. Recent MRI studies have described the differences between the movement of the medial and lateral femoral condyles, which showed the longitudinal rotation of the knee during flexion [10–12,14]. The femur rolled back on the lateral tibial plateau throughout flexion, with less backward movement in the medial compartment. The tibiofemoral contact pattern also shows this longitudinal rotation. The medial femoral condyle rested anteriorly in knee extension and for the first 30° of knee flexion simply rocked back to the centre of the tibial plateau. For the remainder of knee flexion the medial condyle rolled back very little. In contrast, the lateral femoral condyle continued to move posteriorly throughout flexion, indicating the rotation of the knee.

Rotation of the knee may be constrained by loading the knee, or by fixing the position of the feet. Hill et al. reported that in squatting, the longitudinal rotation of the knee was suppressed, when compared with sitting [11]. In the current study, longitudinal rotation of the knee was still evident, despite the fixed foot and ankle

position. The suppression of rotation may be due to the load imposed. While the current study did not demonstrate differences between the loaded and unloaded knee using a 150 or 250 N weight, the weight may have been insufficient to induce the kinematic behaviour seen in a standing squat. Weight-bearing and position constraints are some of the biomechanical differences between a egress in the supine position and a full squat.

For this technique of kinematic analysis to be applicable in the clinical situation or to the study of pathological knee behaviours, it is important that the right and left knees are symmetrical. Research has shown that while there is considerable variation between individuals, there is symmetry within individuals [16]. Other studies have analysed single knees, both in vitro and in vivo, but the symmetrical kinematic behaviour has not been reported [11,12,14]. This study has established the symmetry of healthy knees, so that differences in right to left behaviour may be confidently attributed to pathology.

This technique of tibiofemoral contact point mapping provides a tool which has been previously used only for research, but is practical in the clinical situation, using available technology. MRI analysis of knee kinematics as described by this study provides a robust and reliable method of recording the tibiofemoral contact pattern of the knee. This method is reliable using FSE and GE sequences (with the latter having advantages in efficiency), and has produced results consistent with other methods used to map tibiofemoral contact patterns.

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CHAPTER 5

KINEMATICS OF THE ANTERIOR CRUCIATE LIGAMENT INJURED KNEE

This chapter is in press as:

Scarvell JM, Smith PN, Refshauge K, Galloway HR, Woods KR. Comparison of kinematics in the healthy and ACL injured knee using MRI. *Journal of Biomechanics*, Knee monograph 2003 (in press)

Abstract

Magnetic Resonance Imaging (MRI) was used to examine the characteristics of abnormal motion in the injured knee by mapping tibiofemoral contact. Twenty subjects with a unilateral ACL injury performed a leg-press against resistance. MRI scans of both knees at 15° intervals from 0° to 90° of flexion were used to record the tibiofemoral contact pattern.

The tibiofemoral contact pattern of the injured knees was more posterior on the tibial plateau than the healthy contralateral knees ($p = 0.012$). The tibiofemoral contact pattern of the loaded knees did not differ from the unloaded knees ($p = 0.46$). The difference in the tibiofemoral contact pattern in the ACL-injured knee was more pronounced in patients with more severe knee symptoms ($r = 0.40$), irrespective of the passive anterior laxity of the knee ($r = 0.12$).

Introduction

Anterior cruciate ligament (ACL) injuries are common, the incidence being 30 per 100 000 of the population per year in the United States of America (Miyasaka et al. 1991). Unfortunately, the risk of developing osteoarthritis after ACL injury is 60 - 90%, with the highest incidence being in those who return to sport (Otto et al. 1998; Clatworthy et al. 1999; Gillquist et al. 1999). Degenerative change in the ACL-injured knee is due in part to instability leaving the knee vulnerable to repeat trauma, and in part to altered kinematics including shearing at the articular cartilage (Friden et al. 1993; Vergis et al. 1997; Osternig et al. 2000). An understanding of how the kinematics of the knee are altered by ACL injury is important in order to relate the aberrant kinematics to the process of degeneration.

Kinematics of the ACL-injured knee have been studied using plain films to measure anterior instability in patients performing a standing lunge (Friden et al. 1993) and fluoroscopy (Dennis et al. 1996). However, 2-dimensional data have limited application for analysis of the complex 3-dimensional motion of the knee. 3-dimensional analysis using electrogoniometry (Vergis et al. 1997; Vergis et al. 1998) and roentgen stereophotogrammetric analysis (RSA) (Brandsson et al. 2001) has demonstrated anterior translation and external rotation of the tibia in the ACL-injured knee, but these are complex, expensive and invasive techniques and are impractical in the clinical situation.

Magnetic Resonance Imaging (MRI), like RSA, enables visualisation of the bony structures and is consequently able to record the position and hence the motion of the knee with

precision. Advantages of MRI are that it is non-invasive and is readily accessible in the clinical situation. Open field (Vedi et al. 1999; Hill et al. 2000) and standard closed MRI (Smith et al. 1999; Todo et al. 1999; Scarvell et al. 2003a) have been used to analyse knee kinematics in three dimensions. The kinematics have been described in terms of a tibiofemoral contact pattern (Chapter 4). This allows the roll/glide characteristics and the longitudinal rotation of the knee to be visualised and quantified. In healthy subjects studied using this technique, a high degree of variation has been demonstrated, but with good right to left consistency within individuals, indicating that the uninjured knee may be reliably used as a control (Smith et al. 1999; Scarvell et al. 2003a). The purpose of this study was to record the motion of the ACL-injured knee using MRI, in order to assess the characteristics of abnormal motion in the ACL-injured knee that may contribute to progressive degenerative change. Finally this technique may be useful in predicting those patients at risk of osteoarthritis due to the kinematic behaviour.

Method

Twenty subjects aged between 21 and 52 years, with a unilateral ACL injury were recruited. The exclusion criteria were the same as for Chapter 4, including subjects contraindicated to MRI and subjects over 180cm tall. Subjects were also excluded if history of injury or symptoms were present in the contralateral knee, so that the uninjured knee could act as the control. Eight subjects were male and 12 were female. All ACL injuries were sustained within 3 years of testing. All subjects provided informed consent. This study was approved by the

Australian Capital Territory Department of Health and Community Care and the University of Sydney Human Ethics Committees.

Tibiofemoral contact point measurement from MRI scans

To study kinematic effects of ACL injury, subjects performed a supine leg-press between 0° and 90° knee flexion in the same manner as described in Chapter 4. The leg-press was weighted by a 150N load via a rope and pulley to resist leg extension. Sagittal images were generated through both knees simultaneously using MRI. The position of the tibiofemoral contact was recorded as the distance from the posterior tibial cortex to the point of tibiofemoral contact for both the medial and lateral femoral condyles respectively (Scarvell et al. 2003a; Chapter 4).

Knee injury variables recorded

In addition to recording the pattern of tibiofemoral contact, the passive anterior laxity, function and knee damage visible at arthroscopy were recorded for each subject. The KT 1000 arthrometer was used to measure passive anterior laxity, by quantifying the anterior displacement in the Lachman's manoeuvre (Daniel et al. 1985). We measured anterior displacement of the tibial tubercle in both knees five times: at 15lb, 20lb, 30lb of anterior drawer, during an unloaded straight leg raise and with a maximum manual anterior drawer using the KT 1000 (Figure 5.1). A side-to-side difference of 3mm or more on a manual maximum anterior drawer is considered indicative of an ACL injury, with a sensitivity of 85% (Daniel et al. 1985).



Figure 5.1: Passive anterior laxity at the knee was measured using the KT 1000 arthrometer. An anterior force was applied to the knee via the calibrated handle and the displacement was measured as the difference between the patella pad and tibial tubercle pad.

The Cincinnati knee score (Barber-Westin et al. 1999) was used to measure the symptoms, functional limitations, activity levels of subjects and details of the physical examination. The result was a score out of 100, where 100 was normal, with full function. The Cincinnati knee rating score has high reliability and is sensitive to changes in the ACL-injured population (Barber-Westin et al. 1999).

Injuries to the ACL are frequently associated with injuries to other structures of the knee, either at the time of injury or subsequent to the injury. These associated injuries may impose confounding effects on the kinematics of the injured knee. All subjects in this study underwent arthroscopic knee reconstruction within two months of testing. At arthroscopy, visible joint damage, meniscal damage and cartilage wear were recorded according to the location, area and depth of cartilage lesion (Noyes et al. 1989). The presence of meniscal damage and

damage to other knee ligaments was also recorded. Thus, the effects of meniscal damage and articular cartilage damage on knee kinematics could be tested.

Statistical Analysis

The tibiofemoral contact points recorded for the healthy and ACL-injured knees were compared using repeated measures ANOVA. Factors included in the analysis included the angle of knee flexion, the injured or contralateral knee, the loaded and unloaded condition, and the medial and lateral compartments of the knee.

Difference between the passive anterior laxity of the injured and the contralateral knee was analysed using a paired student's t-test.

The deviation from the healthy knee contact pathway exhibited by the injured knee is quantified as the side-to-side difference in the tibiofemoral contact points. Articular cartilage damage recorded at arthroscopy was compared to the mean of the side-to-side difference recorded at each knee flexion angle, using one-way ANOVA. Passive anterior laxity and Cincinnati knee score were analysed for their correlation with the mean of the side-to-side difference in the contact points, using Pearson's r .

Results

The mechanism of injury for the twenty ACL-injured subjects included 18 playing sports and one fall from a height of 1 metre and one cycling accident. Sports participation included 7 injured playing soccer, 5 at netball, 2 at martial arts, 2 at basketball, 2 skiing and one at Australian Rules football. There were 9 left knees injured and 11 right knees.

At arthroscopy, internal knee joint damage was present in 9 subjects. Damage included femoral cartilage fibrillation of the medial femoral condyle in 2 subjects, (1b, 2a and 2b changes) and patellofemoral cartilage damage in 2 subjects (1a and 1b; Noyes et al. 1989). There was no tibial cartilage damage in any of the subjects. In 3 subjects there was damage to the medial meniscus, including one small tear of the posterior horn, one large displaced tear of the posterior horn and one bucket handle tear. In 3 subjects there was damage to the lateral meniscus, including one mild crush, one small tear and one old partial meniscectomy seen, all of the posterior horn. There were 11 subjects with ACL tears with no other associated joint damage.

Passive anterior laxity at 30lb, the straight leg raise and maximum manual anterior drawer, of the injured knee were each significantly greater than the healthy knee ($p < 0.01$). In 19 of the 20 subjects passive anterior laxity of ≥ 3 mm side-to-side difference at manual maximum anterior drawer supported the clinical diagnosis of ACL injury. The side-to-side difference in anterior displacement of the tibia for the 30lb anterior drawer was 2.7 ± 2.7 mm (mean \pm SD),

for the straight leg raise test was 3.9 ± 2.6 mm and for the manual maximum anterior drawer was 5.5 ± 3.3 mm. ACL tear was confirmed at arthroscopy for all 20 subjects.

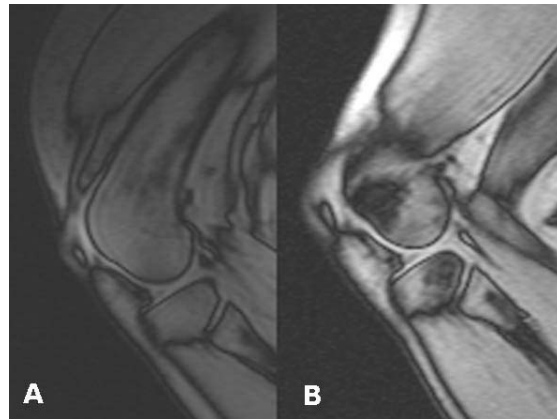


Figure 5.2: Sagittal images of the lateral compartment of the A) healthy contralateral and B) ACL-injured knee of a subject at 75° knee flexion, in the loaded condition. The tibiofemoral contact occurred more posteriorly in the ACL-injured knee than the healthy knee.

MRI knee kinematics

The tibiofemoral contact pattern was located posteriorly on the tibial plateau in the ACL-injured knee ($F_{(1, 152)} = 6.5$, $p = 0.012$) than the healthy knee (Figure 5.2). In the medial compartment of the knee this difference was most pronounced at 0° and 15° (Figure 5.3). At 0° the medial femoral condyle was located 32.2 ± 5.1 mm from the posterior tibial cortex landmark in the ACL-injured knee and 33.2 ± 3.9 mm in the healthy knee. Further into knee flexion the medial compartment of the injured knee followed a similar pattern to the healthy knee. In the lateral compartment of the knee, the femur was more posteriorly positioned on the tibial plateau throughout the range of knee flexion. The lateral femoral condyle was located

24.7 ± 4.5 mm from the posterior tibial cortex landmark in the injured knee and 25.9 ± 3.1 mm in the healthy knee. The posterior femoral contact position appeared more pronounced in the lateral than the medial compartment of the knee (Figure 5.3), but this was not significant ($F_{(1,152)} = 1.3, p = 0.25$). To achieve 80% power a sample size of 144 subjects would have been required, due to the wide variation between individuals. The femoral contact point was on average 1.3mm more posterior than the healthy knee in the lateral compartment of the loaded knee and 0.5mm more posterior in the medial compartment of the loaded knee (Figure 5.4). However, individuals exhibited as much as 9.7mm of posterior femoral contact displacement in the medial compartment and 9.5 mm in the lateral compartment.

In healthy contralateral knees the medial and lateral tibiofemoral contact patterns were distinctly different, reflecting the longitudinal rotation of the knee through flexion. The mediolateral asymmetry of the contact point pattern was preserved in the injured knees ($F_{(1,152)} = 310, p < 0.001$). The healthy contralateral and ACL-injured knees both exhibited internal tibial rotation about the longitudinal axis during knee flexion from 0° to 90°.

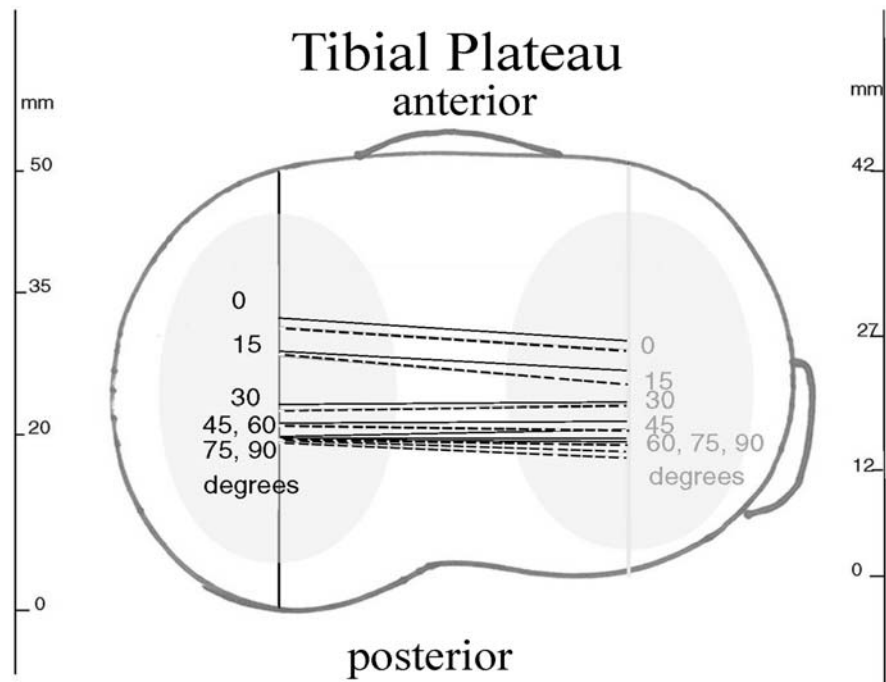


Figure 5.3: Diagram of the tibial plateau indicating the tibiofemoral contact pattern from 0° to 90° knee flexion for the loaded condition in the healthy contralateral (solid line) and ACL-injured knees (dotted line).

Loading the injured and healthy knees did not alter the tibiofemoral contact pattern ($F_{(1,152)} = 0.557$, $p = 0.46$). There was no difference in the contact pattern between subjects when pressing down through the weighted footplate and when relaxed.

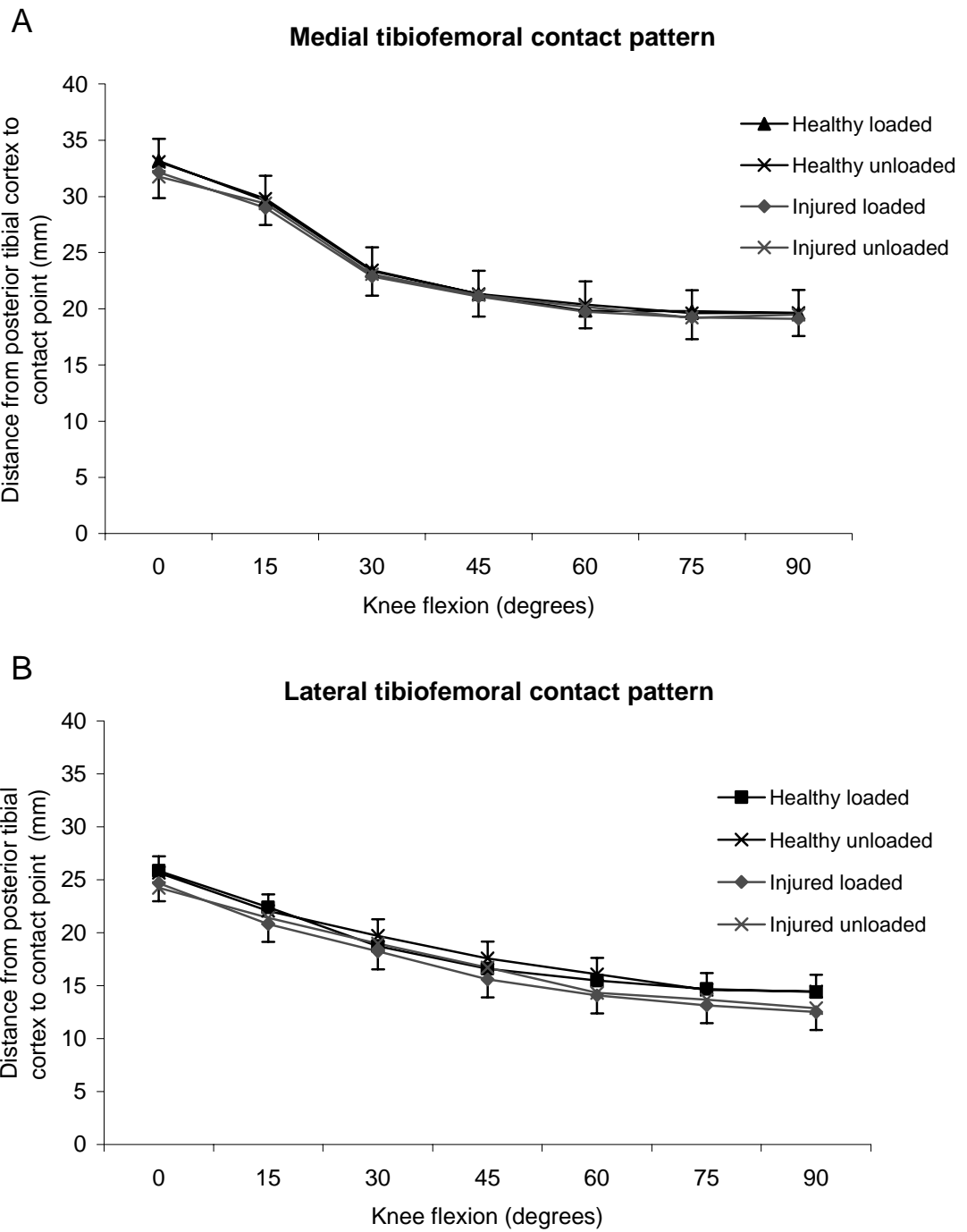


Figure 5.4: Tibiofemoral contact patterns for the A) medial and B) lateral compartments, in the ACL-injured and healthy contralateral knees. The tibiofemoral contact pattern on the tibial plateau in the ACL-injured knees was slightly posterior compared to the healthy contralateral knees, but profile of the curve was preserved.

Kinematics and knee injury variables

The difference in tibiofemoral contact pattern due to injury was moderately correlated with the Cincinnati knee score. The average score for the ACL-injured subjects was 57 ± 10 (range 35 to 76/100). The average pain score was 6 ± 1.7 out of 10 and self-reported grade was 5 ± 1.8 (range: 2 to 8/10), which is defined as fair/good. The difference in tibiofemoral contact pattern due to injury for each subject was measured as the mean of the side-to-side difference in the contact points, at each knee flexion angle. The Cincinnati knee scores were weakly correlated with the mean of the side-to-side difference in the contact points in the unloaded knee (Pearson's $r = 0.40$, $p = 0.07$), but not in the loaded knees ($r=0.25$, $p= 0.28$). The reported knee symptoms taken in isolation (scored out of 20) were also weakly correlated with the mean of the side-to-side difference ($r=0.37$, $p=0.10$). Thus, there is some indication that the subjects with poorer knee scores have greater difference in contact pattern in the injured knee.

Damage to the articular cartilage and menisci were not correlated with a greater difference in the tibiofemoral contact pattern of the knee. The subjects with meniscal damage did not have a significantly different mean side-to-side difference in contact points ($F_{(1,19)} = 2.48$, $p= 0.133$), nor did the subjects with chondral damage ($F_{(1,19)} = 3.69$, $p= 0.71$).

Increased passive anterior laxity of the knee did not correlate with a greater difference in contact pattern. Side-to-side difference at manual maximum anterior drawer, quantified by the KT 1000 measurement did not correlate with side-to-side difference in the contact pattern ($r = 0.12$, $p = 0.60$). Hence, neither passive instability nor damage to the knee joint was related to the active instability demonstrated on MRI.

Discussion

The tibiofemoral contact pattern of the ACL-injured knees was significantly different to the healthy contralateral knees. The high right to left consistency within subjects (Smith et al. 1999, Scarvell et al. 2003a), indicates that the contralateral knee can reliably be used as a control for tibiofemoral contact pattern analysis. The difference in contact pattern between the ACL-injured knee and the healthy contralateral knee can therefore be attributed to the knee pathology.

The posterior tibiofemoral contact pattern due to ACL injury has been reported by other authors. Dennis et al (1996) used videotaped fluoroscopic images to examine the tibiofemoral contact patterns in the ACL-deficient knee in standing deep knee flexion. They described the ACL-injured subjects moving from posterior tibiofemoral contact positions in knee extension, to variations in normal and posterior contact positions through flexion. Three subjects demonstrated paradoxical roll-forward of the femoral condyles on knee flexion. The average difference in contact position between the injured and intact knee was only 1.6mm, but the difference ranged from 0.5mm to 13.7mm in some knees. That study used 2-dimensional imaging at low resolution, so was unable to draw out information regarding rotation. Our study shows a more consistent tibiofemoral contact pattern, with preservation of the roll/glide characteristics, but a more posterior pattern overall.

The shift in the axis of longitudinal rotation of the knee due to ACL deficiency was not confirmed in our study, due to the wide variations in the kinematics of subjects. It appeared,

however, that the effect of the ACL injury tended to generate a tibiofemoral contact pattern more posterior in the lateral compartment than the medial compartment of the knee. This might indicate that the axis of longitudinal rotation of the knee had shifted medially. It was observed by Brandsson et al (2001) using RSA that while there was no difference in anteroposterior displacement of the medial compartment, there was a difference in the lateral compartment of the injured knees. The injured knees also maintained more external tibial rotation than the healthy knees. The reference points used in the analysis by Brandsson et al. are different to the reference points used in our MRI study, so parallel analysis is difficult. However, the medial/lateral characteristics of the injured knee motion are similar. The role of the ACL in the control of longitudinal rotation of the knee is recognised (Grood et al. 1988; Buckland-Wright et al. 1994; Anderson et al. 1997; Benvenuti et al. 1999; Woo et al. 2002), so it is reasonable to suspect that having lost the primary restraint of the ACL, the secondary restraint of the medial collateral ligament may play a greater role in controlling knee rotation.

The magnitude of the difference in tibiofemoral contact between the healthy contralateral and ACL-injured knees (1.3 ± 1.64 mm) was similar to that reported by Brandsson et al (2001), in subjects performing a step-up activity. In subjects performing a standing lunge the anterior tibial displacement recorded using plain radiography was 12mm (Friden et al. 1993) and in a step up activity measured by electrogoniometry was 5mm (Vergis et al. 1997; Vergis et al. 1998). A study of passive anterior displacement of the tibia confirmed that the magnitude of the difference measured using RSA is smaller than that measured using a KT 1000 arthrometer or plain radiography (Fleming et al. 2002). This discrepancy in measurement between the different tools may be because plain radiography and KT 1000 arthrometry, being 2-dimensional, are unable to account for longitudinal rotation of the knee and describe anterior

tibial shift as a 2-dimensional feature of the ACL-deficient knee. RSA and MRI techniques are able to analyse rotation and anterior translation independently. Consequently MRI and RSA record the difference in displacement of similar magnitude.

In our study, loading the knees did not change the tibiofemoral contact pattern. Hill et al (2000), using open field MRI, reported rotation was suppressed in subjects performing a standing squat.. However, there are several biomechanical differences between a supine leg press and a standing weight-bearing activity (Friden et al. 1993; Dennis et al. 1996; Hill et al. 2000). Subjects in supine experience different proprioceptive feedback to standing subjects. Additionally, joint compression in standing is greater and contact occurs over a wider area. Our subjects were pressing down against a 150N load, substantially less than the body weight supported in a standing lunge, although we found no difference when the weight was increased to 300N (Scarvell 2003a). This protocol is intended to be suitable for clinical application. As such, it has been designed to use a clinically available closed tunnel MRI unit and weights sustainable by an injured population. Thus, comparison of findings between a supine leg press and standing lunge may be inappropriate.

Closed-chain resisted exercises have been advocated for rehabilitation after ACL injury and reconstruction (McGinty 2000; Toutoungi 2000), in which the foot is fixed and the axial force in line with the tibia. Closed-chain exercises minimise stress on the ACL, theoretically by increasing joint compression, decreasing tibiofemoral shear forces and increasing muscular co-contraction (Beynon et al. 1995; MacWilliams et al. 1999). The supine leg-press exercise is therefore one that has been commonly prescribed. The present study showed that the tibiofemoral contact pattern of the ACL-injured knee was different from the healthy

contralateral knee performing a supine leg-press. In effect, that to press down through the feet did not normalise the tibiofemoral contact pattern. It may be that further testing of the ACL strain in vivo is necessary to clarify the relative “safety” of the leg-press exercise.

It has been suggested that increasing knee symptoms correlate with instability during activity, regardless of passive anterior laxity (Friden et al. 1993). Friden et al (1993) suggested that subjects with less displacement had learnt to actively control the instability of the knee and therefore experienced fewer symptoms. Our study has also shown a relationship between symptoms and active instability, reflected by the tibiofemoral contact pattern, but no relationship between passive anterior laxity and difference in the tibiofemoral contact pattern.

The tibiofemoral contact appears to occur over a more posterior region of the tibial plateau in the ACL-injured knee, and this may be implicated in the degeneration of the articular cartilage of the knee. While it is difficult to isolate damage to the knee joint caused by repeat episodes of trauma over time from damage caused by aberrant kinematic behaviour, there is an increasing body of evidence that aberrant knee kinematics contribute to articular cartilage damage. Damage to the medial articular cartilage has been associated with time since ACL injury (Myers et al 2001). Harman et al (1998) also found ACL deficiency was associated with articular cartilage wear over a more posterior area of the tibial plateau. The menisci suffer from chronic loading and repeat trauma in the ACL-deficient knee (Finsterbush et al. 1990; Irvine et al. 1992; Keene et al. 1993; Bellabarba et al. 1997; Allen et al. 2001). Damage to the menisci contributes to the development of osteoarthritis (Fairbank 1948). In this manner, the aberrant kinematic behaviour of the knee is responsible for the degeneration of the menisci and the consequential osteoarthritis. Our study shows that there is a change in the tibiofemoral

contact pattern resulting from ACL injury, which is likely to contribute to accelerated wear over the long term (Bellabarba et al. 1997; Clatworthy et al. 1999; Gillquist et al. 1999).

Conclusion

The characteristics of abnormal motion in the ACL-injured knee are firstly, that the tibiofemoral contact in the ACL-injured knee occurs over a posterior area on the tibial plateau. Secondly, that the longitudinal rotation evident in the healthy knee does occur in the ACL-injured knee, however, the axis of rotation may have shifted medially. The difference in the tibiofemoral contact pattern in the ACL-injured knee is more pronounced in patients with more severe knee symptoms, especially pain, irrespective of the passive anterior laxity of the knee.

CHAPTER 6

COMPARISON OF KINEMATIC ANALYSIS BY MAPPING TIBIOFEMORAL CONTACT WITH MOVEMENT OF THE FEMORAL CONDYLAR CENTRES IN HEALTHY AND ANTERIOR CRUCIATE LIGAMENT INJURED KNEES.

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Statement from co-authors confirming the authorship contribution of
the PhD candidate

"As co-authors of the paper "*Comparison of kinematic analysis by mapping tibiofemoral contact with movement of the femoral condylar centres in healthy and anterior cruciate ligament injured knees*" we confirm that Jennifer M Scarvell has made the following contributions:

- Contributed to discussions on design of the study
- Collection, analysis and synthesis of literature
- Recruitment of subjects and collection of data under supervision
- Analysis and interpretation of data under supervision
- Wrote the first draft of the manuscript, and followed through to publication, including proofing and final publication details of the manuscript."

Professor Kathryn M Refshauge	Signed: 	Date:.....
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Comparison of kinematic analysis by mapping tibiofemoral contact with movement of the femoral condylar centres in healthy and anterior cruciate ligament injured knees

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Abstract

Two methods of analysis of knee kinematics from magnetic resonance images (MRI) *in vivo* have been developed independently: mapping the tibiofemoral contact, and tracking the femoral condylar centre. These two methods are compared for the assessment of kinematics in the healthy and the anterior cruciate ligament injured knee.

Sagittal images of both knees of 20 subjects with unilateral anterior cruciate ligament injury were analysed. The subjects had performed a supine leg press against a 150 N load. Images were generated at 15° intervals from 0° to 90° knee flexion. The tibiofemoral contact, and the centre of the femoral condyle (defined by the flexion facet centre (FFC)), were measured from the posterior tibial cortex.

The pattern of contact in the healthy knee showed the femoral roll back from 0° to 30°, then from 30° to 90° the medial condyle rolled back little, while the lateral condyle continued to roll back on the tibial plateau. The contact pattern was more posterior in the injured knee ($p = 0.012$), particularly in the lateral compartment. The medial FFC moved back very little during knee flexion, while the lateral FFC moved back throughout the flexion arc. The FFC was not significantly different in the injured knee ($p = 0.17$).

The contact and movement of the FFC both demonstrated kinematic events at the knee, such as longitudinal rotation. Both methods are relevant to design of total knee arthroplasty: movement of the FFC for consideration of axis alignment, and contact pattern for issues of interface wear and arthritic change in ligament injury.

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Introduction

Since the introduction of interventional MRI there has been renewed interest in the study of *in vivo* knee kinematics that MRI has made possible. The development of knee arthroplasty has fuelled the need for better understanding of the location and governance of the axes of knee rotation and the tibiofemoral (TF) interface dynamics due to issues of component wear. Thus, the analysis and interpretation of the MRI recordings can be approached from a variety of ways. Emphasis on the axis of rotation for knee prosthetic design has led to analysis of knee kinematics by tracking the axis through the femoral condyles [8,15,21,31]. Researchers studying

the effects of motion on wear at the TF component interface have examined the TF contact dynamics [2,5,18,30,33,37]. This study compares the two approaches to interpretation of knee kinematics *in vivo*.

The flexion axis of the knee was described in early documents as a fixed axis passing through the centre of the posterior femoral condyles [35]. This view was revised later, as cadaveric studies described sagittal sections through the femoral condyles as spirals, not circles, and the axis thus defined as an instant centre of rotation at any moment during the flexion arc [4,16,34]. The three dimensional nature of MRI has enabled the plane of sagittal sections to be redefined as perpendicular to the line through the centres of the posterior femoral condyles [13,15,19,23,25]. This alignment of the sagittal plane to the posterior condylar centres has demonstrated the femoral condyles to be indeed circular, and the theory of the fixed flexion axis has been re-examined.

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Tracking the movement of the femoral condyles during the knee flexion arc has enabled a description of knee kinematics based on the reference position of the femoral condyles over the tibial plateau [31]. In vivo studies have compared the movement of the femoral condyles in passive and active knee extension in side-lying [31] and in a standing squat [13]. The centre of the posterior femoral condyle has been termed the flexion facet centre (FFC) since it is this facet that articulates with the tibia during the flexion arc [8]. Tracking the FFC has been particularly useful to describe the longitudinal rotation of the knee during the flexion arc [13–15,17].

Independent researchers have analysed knee kinematics from the perspective of the TF contact areas [26,29,37]. Standard closed field MRI machines have been used for these studies, to examine the TF contact pattern of the knee when passively repositioned, or actively pressing down through the feet. Fluoroscopic analysis of contact areas in standing and walking has also been reported [2,6,18]. The contact areas provide information regarding the roll/glide motion at the joint surface, and the pattern of loading through the joint [10,20]. There has not yet been a study combining these two methods of kinematic analysis in order to compare both characteristics of knee motion.

In order to compare the two characteristics of knee kinematics described by the tracking of the FFCs with the TF contact pattern, a third variable is introduced, that of anterior cruciate ligament (ACL) deficiency. The ACL has been described as an essential part of the governance of the roll/glide motion of the knee [22,24], and a major determinant of both the flexion axis and the longitudinal axis of rotation [24,36]. Thus, the impact of ACL deficiency on the motion of the femoral condylar centres and TF contact pattern may be examined.

The aim of this study was therefore to examine the differences in kinematic terms of the behaviour of TF

contact points versus the FFC in both healthy and ACL-injured knees.

Method

Twenty subjects aged between 21 and 52 years, with a unilateral ACL injury were recruited for a study of kinematic effects of ACL injury [27]. Eight subjects were male, 12 female. Subjects were excluded if there were any contraindications to MRI, may have been pregnant, or they were over 180 cm tall (to permit knee flexion in the MRI tunnel, Fig. 1). Subjects were also excluded if history of injury or symptoms were present in the contralateral knee, so that the uninjured knee could act as the control. All ACL injuries were sustained within 3 years of testing. A complete ACL tear was diagnosed for all subjects at examination by an orthopaedic surgeon, and confirmed at arthroscopy for knee reconstruction following the study. All subjects provided informed consent. Ethics approval for the study was obtained from the local University and Department of Health Committees.

MRI imaging procedure

For the study of kinematic effects of ACL injury the subjects had performed a supine leg-press between 0° and 90° knee flexion (Fig. 1) [27]. To enable standardisation of knee flexion positions between 0° and 90° a wooden frame with a sliding footplate was fitted to the MRI couch. The leg-press was weighted by a 150 N load via a rope and pulley to resist leg extension. Elastic straps stabilised the thighs, feet and ankles. Imaging of both knees simultaneously was performed using a 1.5 T Siemens Magnetron Vision (Erlangen, Germany). A body coil was used to generate parasagittal images, defined as perpendicular to the tibial plateau. Using spoiled gradient echo sequences, eight sagittal slices were generated through each knee, approximately 10 mm apart (TR = 160.0, TE = 2.3/1, TA = 00:46), with a 256 × 256 matrix. Images were saved as digital image files in Dicom and Bitmap formats.

Tibiofemoral contact point measurement

The position of the TF contact with the tibial plateau was recorded as the distance from the posterior tibial cortex to the point of TF contact of the medial and lateral femoral condyle (Fig. 2) [28]. Where contact occurred over a wide area, the area centroid was used. To account for variation in the size of subjects, cortex to contact distance measurements were normalised in proportion to mean tibial dimensions. The mean AP diameter of the medial tibial plateau was 50 ± 4.4 mm, and the lateral tibial plateau was 42 ± 2.17 mm. The TF contact map was scaled onto a tibial plateau of standard dimensions.

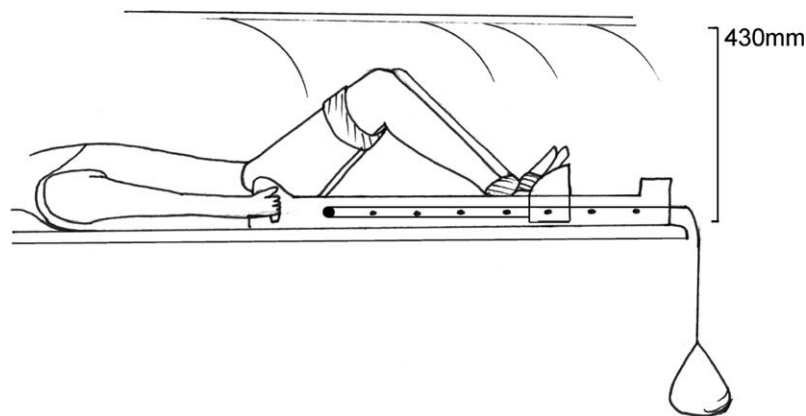


Fig. 1. Subjects' position in the MRI scanning tunnel. The knees were positioned at 15°-intervals between 0° and 90° flexion, relaxed, or pressing down through the feet against a 150 N load.

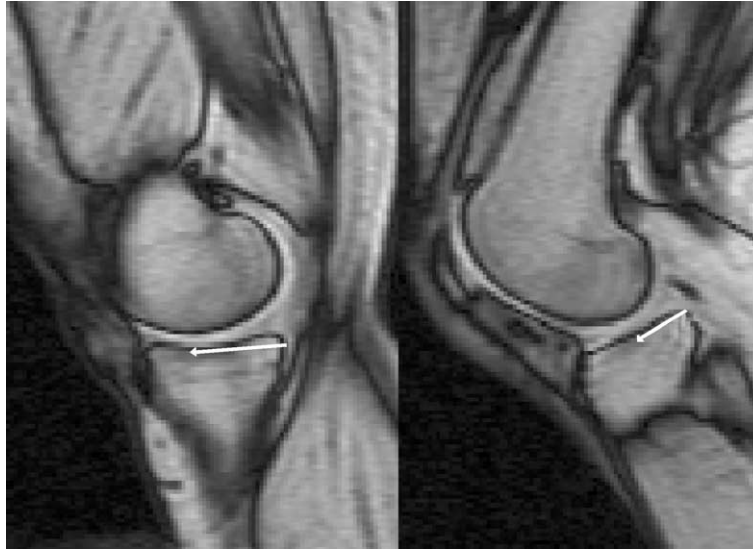


Fig. 2. Sagittal images through the centre of the medial and lateral compartment of the knee were used to measure the tibiofemoral contact. The distance was measured from the posterior tibial cortex to the centre of the area of contact (arrows).

Flexion facet centre measurement

The position of the FFC over the tibial plateau was located by a three-stage procedure with a computer assisted design program (Fig. 3) (IntelliCAD® Technology Consortium, Portland Oregon). First, the FFC was identified by fitting a circle to define the flexion arc of the posterior condyle. Second, the tibial plateau was defined by a line from the posterior tibial cortex to the anterior tibial cortex, parallel to the tibial plateau. Lastly, a line was drawn through the FFC perpendicular to the tibial plateau line to measure the distance from the posterior tibial cortex to the intersection of the perpendicular line.

Reliability

The reliability of both methods of measurement was tested by repeating measurements from the original scan images of seven subjects on two occasions at least 24 h apart. The reliability of mapping the contact points for the medial and lateral compartments was very

high with intra class correlation_(2,1) 0.94 (99% confidence interval was 0.91–0.97). The reliability of measurement of the FFC was also very high with intra class correlation_(2,1) of 0.94 (95% confidence interval was 0.83–0.97). The distances from the posterior tibial cortex to the contact point, or FFC, for the 20 subjects records a 95% confidence interval of the measurement of 1.30 mm.

Results

The contact for the healthy knee in the medial and lateral compartments was anterior in knee extension, and then moved posteriorly during the flexion arc (Fig. 4A). The medial condyle began 33 ± 3.8 mm from the posterior cortex, which was just 17 mm from the

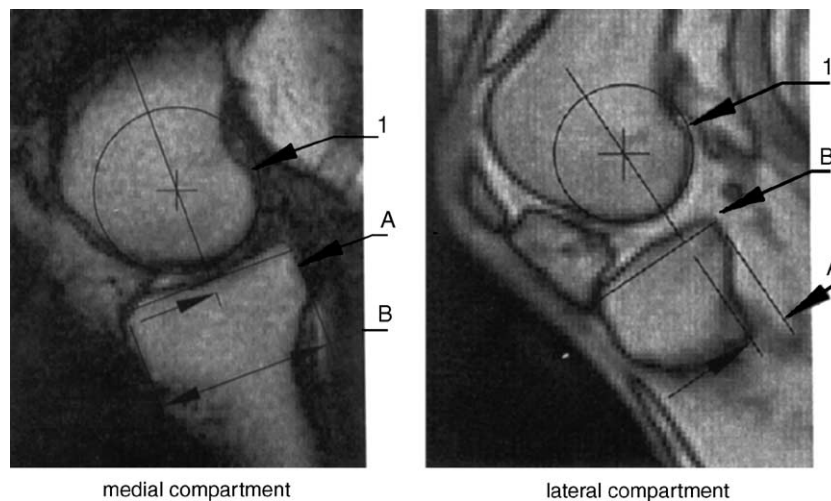


Fig. 3. The position of the flexion facet centre over the tibial plateau was measured in three steps: the arc and centre of the posterior femoral condyle were defined (1), the tibial plateau was defined, and the distance from the perpendicular through the centre to the posterior tibial cortex was measured. (A) Distance from the flexion facet centre to the posterior tibial cortex and (B) anterior and posterior tibial cortex defined the angle and the limits of the tibial plateau.

Table 1

The position of the flexion facet centre and the tibiofemoral contact point in the healthy and ACL-injured knees of 20 subjects (mean and standard deviation), measured in millimetres from the posterior tibial cortex

	Flexion facet centres		Tibiofemoral contact	
	ACL-injured		ACL-injured	
	Mean	SD	Mean	SD
<i>Lateral compartment</i>				
0°	22.0	5.1	25.0	4.5
15°	22.8	3.6	20.9	3.4
30°	21.2	4.1	18.2	3.3
45°	18.8	4.2	15.5	3.4
60°	17.3	3.5	13.7	3.5
75°	15.5	3.9	13.1	3.0
90°	15.4	2.6	12.3	3.0
<i>Medial compartment</i>				
0°	21.2	4.0	32.0	5.0
15°	23.4	3.9	29.0	3.5
30°	24.4	3.0	23.4	3.2
45°	23.1	2.8	21.2	2.4
60°	22.9	1.4	19.7	1.8
75°	22.3	2.3	19.4	2.1
90°	22.9	1.8	19.2	2.3
	Healthy		Healthy	
<i>Lateral compartment</i>				
0°	24.1	3.3	26.1	3.1
15°	23.7	3.4	22.5	2.8
30°	21.7	3.8	18.6	2.4
45°	19.7	3.2	16.5	2.0
60°	18.2	2.4	15.3	2.9
75°	16.8	2.3	14.4	2.8
90°	15.9	2.4	14.2	2.8
<i>Medial compartment</i>				
0°	22.5	3.4	33.0	3.8
15°	24.8	3.3	29.6	3.7
30°	24.3	2.7	23.6	3.5
45°	23.4	2.5	21.3	2.9
60°	22.8	2.4	20.1	2.6
75°	22.6	2.4	20.0	2.9
90°	22.9	2.2	19.8	2.8

anterior edge of the tibial plateau (Table 1). The lateral condyle began 26.1 ± 3.1 mm from the posterior tibial cortex. Both the medial and lateral femoral condyles then rolled back along the tibial plateau. Between 0° and 30°, the medial condyle covered 9.6 mm, which was 0.3 mm per degree. Between 0° and 30° the lateral condyle covered 7.5 mm, which was 0.25 mm per degree. Between 45° and 90° the medial condyle did not move posteriorly as much, 1.5 mm in 45°, or 0.03 mm per degree. The lateral condyle moved a further 2.3 mm posteriorly, which was 0.05 mm per degree. The lateral condyle at 90° knee flexion was situated 14.2 ± 2.8 mm from the posterior tibial cortex. The medial femoral condyle was situated 19.8 ± 2.8 mm from the posterior tibial cortex, so the lateral TF contact had moved more

posteriorly than the medial TF contact. Between 0° and 30° the TF contact pattern was demonstrating roll of the condyles, beyond 30° the condyles were demonstrating more glide, especially the medial condyle.

The TF contact in the injured knee had similar roll/glide characteristics to the healthy knee, but was located significantly more posteriorly on the tibial plateau ($F_{(1,152)} = 6.5$, $p = 0.012$) (Fig. 4B). In the medial compartment of the knee the difference was most pronounced at 0° and 15°. At 0° the medial femoral condyle was located 32.2 ± 5.1 mm from the posterior tibial cortex landmark in the ACL-injured knee, compared to 33.2 ± 3.9 mm in the healthy knee. Further into knee flexion the TF contact of the medial compartment of the injured knee followed closely the healthy knee. In the lateral compartment of the knee, the TF contact of the injured knee was more posterior on the tibial plateau than the healthy knee throughout the range of knee flexion (Fig. 5A). The femoral contact point was on average 1.3 mm more posterior than the healthy knee in the lateral compartment of the loaded knee, and 0.5 mm more posterior in the medial compartment of the loaded knee (Fig. 5A). However, individuals exhibited as much as 9.7 mm of posterior femoral contact displacement in the medial compartment and 9.5 mm in the lateral compartment.

The FFCs of the healthy knee were positioned over the centre of the tibial plateau in knee extension. The medial FFC moved forward slightly as the knee flexes to 15° (Fig. 4C). The medial FFC moved from 22.5 ± 3.3 mm at 0° to 24.8 ± 3.4 mm from the posterior tibial cortex, at 15° knee flexion. Beyond 30° the medial FFC remained centrally located over the medial tibial plateau, at 90° it was 22.9 ± 2.2 mm from the posterior tibial cortex. The lateral FFC moved posteriorly at a steady rate during the flexion arc to 90°, from 24.1 ± 3.3 mm at 0°, to 15.9 ± 2.4 mm at 90° flexion. The movement of the FFC in the ACL-injured knee was not significantly different to the healthy knee ($F_{(1,76)} = 1.86$, $p = 0.17$) (Fig. 5B).

The characteristics of the FFC and TF contact were similar in the medial and lateral compartments, that from 15° to 90° the medial condyle and contact remained central on the tibial plateau while the lateral condyle and contact continued to move back on the tibial plateau. This axial rotation of the knee around a medially located axis was demonstrated by both the motion of the FFC and TF contact.

The FFC was not positioned directly above the TF contact. Between 0° and 15°, the FFC was posterior to the TF contact (Fig. 5C). Between 30° and 90° the FFC was anterior to the TF contact. The greatest difference between the TF contact and FFC position was in knee extension, when the difference between the FFC and TF contact was 10.5 mm in the medial compartment, and 2.0 mm in the lateral compartment.

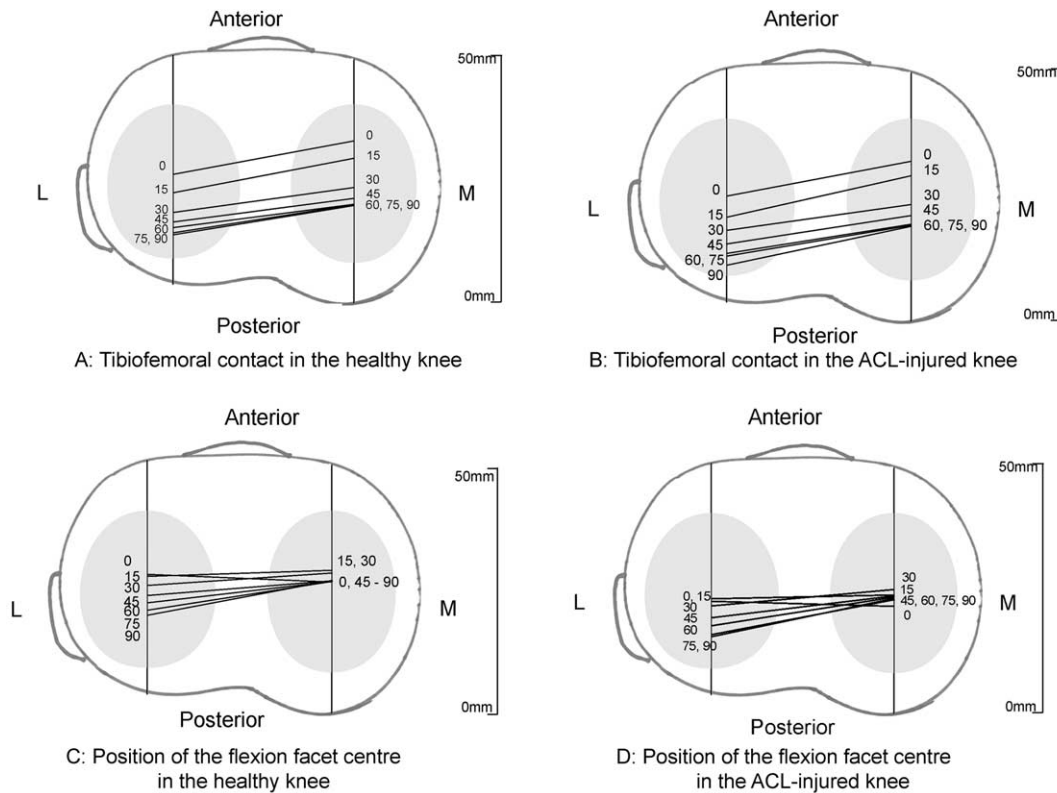


Fig. 4. Comparison of flexion facet centre position with tibiofemoral contact, between 0° and 90° knee flexion. Tibiofemoral contact in the: (A) healthy knee and (B) in the ACL-injured knee. Flexion facet centre position over the tibial plateau in the: (C) healthy knee and (D) ACL-injured knee. (L) lateral and (M) medial.

Discussion

Kinematic events are recognisable in the movement of the FFC and TF contact pattern, including the screw-home in knee extension, longitudinal rotation during flexion, and the effect of ACL injury on the control of rotation. The behaviour of the knee from 0° to 15° was different from the behaviour between 30° and 90° for both the TF contact pattern and position of the FFC. The medial TF contact rolled to its anterior limit in knee extension, and the FFC demonstrated a paradoxical backward rock. This paradoxical backward rock of the FFC is considered to be due to the axis of flexion shifting from the flexion facet to the extension facet centre [8].

The longitudinal axis of rotation of the knee was demonstrated by the TF contact and the FFC movement in the healthy and ACL-injured knees. In the medial compartment, the TF contact and FFC posterior excursion was limited, compared to the greater range of excursion for the lateral TF contact and FFC. The difference between the medial and lateral FFC movement has been described as evidence of the longitudinal rotation of the knee during the flexion arc, the axis of which lies through the medial compartment [13].

The effect of ACL injury over the control of the axis of longitudinal rotation is clearer in the TF contact than the FFC movement. The lateral femoral condyle traced a posterior pathway on the lateral tibial plateau, but the medial condyle was unchanged [27]. The effect of ACL injury on TF contact may be greater than its effect over FFC movement. ACL injury results in both laxity to anterior displacement forces at the knee and also rotary instability. TF contact is able to reflect both of these features.

Knee kinematics exhibited by the pattern of TF contact was been described differently by Wretenberg et al. [37] who performed a study of 16 healthy subjects, where the sagittal MR images were aligned with the long axis of the femur and tibia. A customised knee splint held the knee position. It may be that the orientation of the MR images and the constraints imposed by the knee splint account for the differences between his results and those we have reported. Wretenberg et al. describe similar contact points to those in our study at 0°, but then the contact moves backward at 30° and forward again at 60°. The contact pattern is almost parallel between the medial and lateral compartments. The pilot study of Smith et al. [29] reports the TF contact points moving posteriorly during knee flexion, but more so in the lateral than the medial compartment of the knee.

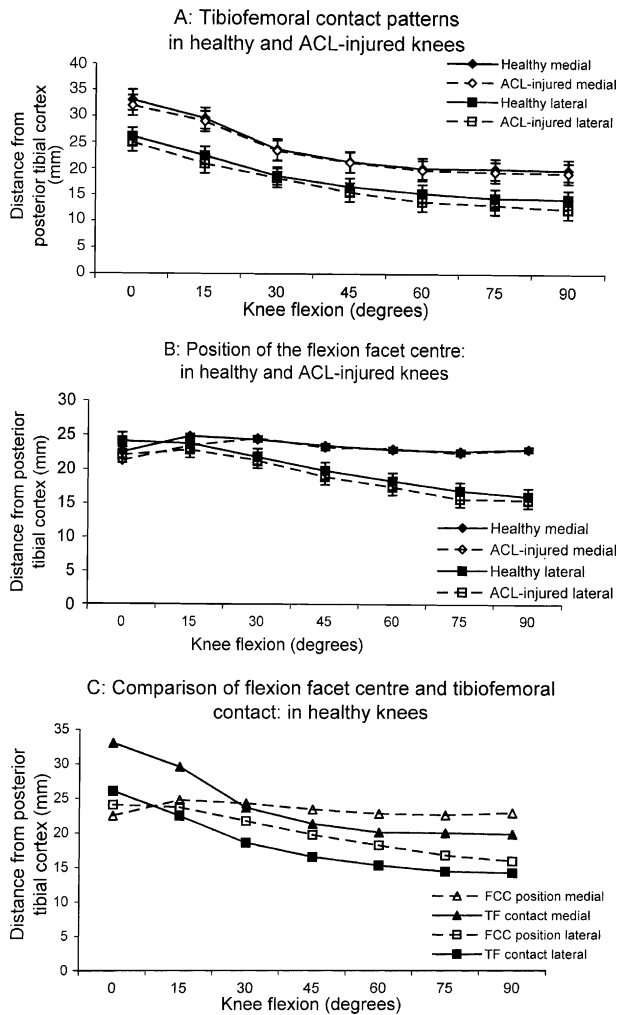


Fig. 5. Graphs comparing tibiofemoral contact and flexion facet centre position in the healthy and ACL-injured knee, performing a leg press against a 150 N load. (A) Tibiofemoral contact positions in healthy and ACL-injured knees, demonstrating the similarity of medial compartment contact in the healthy and ACL-injured knees, but difference in the lateral compartment contact. (B) Position of the flexion facet centre over the tibial plateau in healthy and ACL-injured knees. There was no significant difference between the healthy and injured knees. (C) Comparison of tibiofemoral contact with flexion facet centre position in healthy knees. At 0° flexion the FFC is posterior to the TF contact, but from 30° to 90° the FFC was anterior to the TF contact.

Wretenberg aligned the sagittal imaging plane from the long axis of the femur, rather than from the tibial plateau, as in this study, however, this is unlikely to have made such a difference to the reported contact pattern. It is more likely the kinematics were indeed different in his study.

The movement of the femoral condyles referenced from the FFC has been widely reported in vivo, in a variety of kinematic activities [1,12,13,31]. During most knee flexion activities the medial FFC remains essentially central on the tibial plateau, while the lateral FFC moves posteriorly with regard to the tibial plateau as the

knee flexes [32]. It has been shown that the axis of rotation of the knee can be constrained by loading the knee and fixing the foot in subjects performing a standing squat [17]. Indeed there are a variety of movement patterns available to the knee within the envelope of passive motion, depending on the loading, dynamic forces and constraints of particular kinematic events [3]. The movement of the FFC seen in this study is consistent with that seen by Hill et al. [13] Iwaki et al. [15] and Karrholm et al. [17] within the constraints of the supine leg press activity performed.

To describe kinematic events at the knee by measuring the TF contact pattern was useful in both knee flexion and extension. However the FFC is most useful beyond 30° flexion, when the flexion axis of the knee is through the FFC [8,9,25]. Between 0° and 30° the extension facet is in contact with the tibia: the knee rotates about an axis through the extension facet centre and the FFC is therefore not relevant in this arc [8]. This may be why there appears to be a paradoxical backward rock of the FFC at 0–30°, because the axis of rotation of the knee has shifted from the FFC to the extension facet centre at that time.

The shift of the axis of rotation between the extension facet centre and FFC may explain why the FFC is positioned posteriorly to the TF contact in knee extension, but it does not explain why the FFC would not be directly above the TF contact point at deeper knee flexion angles. One reason for this could be the method of measuring the distance from the FFC to the posterior tibial cortex is dependent on the angle of the tibial plateau, so the perpendicular intersecting line can be drawn. The tibial plateau, however is not orientated horizontally, but rather 13° posteriorly. Therefore the natural weight bearing line is not perpendicular to the tibial plateau, but at 77° to it [24]. If measurement of the FFC position were referenced from a plumb line through the knee, then perhaps the TF contact points would lie directly underneath the FFC.

Measurement of TF contact permits examination of events at the TF interface. These interface events are clinically important to patients and of practical importance to surgeons. Arthritis is an event occurring at the TF interface. One study of 450 tibial plateau specimens resected during knee arthroplasty procedures linked wear patterns to attenuation or loss of integrity of the ACL [11]. Fluoroscopy has been used to concentrate on TF contact because of the relevance to wear in total knee arthroplasty [2,5,6,30]. The goal of total knee arthroplasty has been to replicate the kinematics of the healthy knee. Studies have examined anterior and posterior cruciate retaining and sacrificing prosthesis designs in vivo to see how analogous the TF contact pattern is to that of the healthy knee [5]. Fluoroscopic mapping of TF contact patterns have enabled prediction

of wear patterns for different total knee arthroplasty designs [7].

The two methods for examining knee kinematics, measurement of TF contact patterns and movement of the FFC both describe kinematic events at the knee. Both methods are applicable and valid but the differences between them need to be noted. The TF contact pattern was more sensitive to the effects of ACL injury. The movement of the FFC has limited application between 0° and 30° knee flexion, when the tibia is articulating with the extension facet of the femoral condyles. The position of the extension facet centre may be more usefully examined at these flexion angles. The ability to study kinematic events at the TF interface may be useful in a range of knee pathologies that are vulnerable to degenerative change due to kinematic disturbance.

Acknowledgements

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CHAPTER 7

KINEMATICS FOLLOWING KNEE RECONSTRUCTION

Abstract

It is unclear whether ACL reconstruction prevents the natural history of degeneration in the ACL-deficient knee or whether normal knee kinematics are restored. This prospective study used MRI to measure knee kinematics before and after reconstruction.

20 ACL-injured subjects performed a closed-chain leg-press, relaxed and against a 150N load. MRI recorded the tibiofemoral contact position at 15° intervals from 0° to 90° degrees of knee flexion. Surgical outcomes were measured as passive laxity using a KT1000 arthrometer and Cincinnati rating. All measures were performed preoperatively, at 12 weeks and 2 years postoperatively.

Surgical outcomes were “excellent” or “very good” in 15/20 subjects; 5 were fair, none were poor. Preoperatively the contact patterns for the ACL-injured knees were different to the healthy contralateral knees ($p = 0.014$), but were not significantly different at 12 weeks ($p = 0.117$) or at 2 years postoperatively ($p = 0.909$). However, the lateral compartment contact pattern of the healthy and the reconstructed knees of injured subjects provided some evidence of less tibiofemoral rollback at 2 years than in healthy control subjects ($p < 0.01$).

The knee reconstruction restored the tibiofemoral contact pattern to that of the healthy contralateral knee, but both the healthy contralateral and reconstructed knees showed changes over time.

Introduction

Anterior cruciate ligament injury alters the kinematics of the knee, and this may be responsible for the natural history of degeneration of the ACL-deficient knee. It is not clear whether knee reconstruction surgery restores normal knee kinematics. There is no evidence yet that reconstruction prevents, osteoarthritis. In fact reconstructed patients may have a higher incidence of osteoarthritis than those unoperated (Gillquist 1993; Daniel et al. 1994; Maletius and Messner 1999). A 7% - 18% incidence of osteoarthritis was reported 7 years after bone-patella tendon-bone reconstructions (Jomha et al. 1999; O'Neill 2001) and a 4% - 16% incidence 5 years after hamstrings graft reconstructions (O'Neill 2001; Pinczewski et al. 2002). Incidence of osteoarthritis as evident on x-ray has been reported to be as high as 50% 7 years after hamstrings graft reconstruction (Ruiz et al. 2002). The consequence of the recent rapid advances in knee reconstruction techniques is that longitudinal prospective studies are not yet available beyond 7 years.

Since abnormal kinematics of the ACL-deficient knee have been considered partly responsible for the high incidence of late osteoarthritis (Gillquist 1993), research has emphasised kinematics of the knee before and after knee reconstruction surgery. In vitro studies have demonstrated that the anterior stability of the reconstructed knee is similar to the normal knee (Papergeorgiou et al. 2001; Yagi et al. 2002), but the rotary stability is not restored by reconstruction (Woo et al. 2002). In vitro studies of reconstructed knees have limited application to in vivo knee kinematics, as the motions and forces of normal activity are not reproduced.

In vivo studies of knee kinematics before and after reconstruction surgery have used electrogoniometry (Bulgheroni et al. 1997; De Vita et al. 1998), biplanar video analysis (Georgoulis et al. 2003) and roentgen stereophotogrammetric analysis (RSA) (Brandsson et al. 2001; Brandsson et al. 2002). Knee kinematics do not appear to be restored to normal, as the altered rotation reported preoperatively is maintained (Brandsson et al. 2002) and increased sagittal plane motion sometimes persists (Beard et al. 2001). These studies have included subjects who underwent a variety of different reconstruction techniques. All studies examined weight-bearing activity, such as walking or stepping up. Kinematics of non-weight-bearing activities have not been compared to weight-bearing activities after knee reconstruction. This is relevant given the prescription of open and closed chain exercises for this group of patients.

The characteristics of knee kinematics described by the tibiofemoral contact pattern have not been examined in subjects following knee reconstruction. Recently ACL-injured knees have been shown to demonstrate different contact patterns to healthy knees, particularly in the lateral compartment (Chapter 5; Scarvell et al. 2002a, 2003c). In the chronic ACL-deficient knee it is the medial compartment contact pattern that is affected, as the secondary restraints to anterior translation are attenuated by chronic loading (Chapter 8; Scarvell et al. 2003b). Whether these abnormal contact patterns are corrected by knee reconstruction surgery is not known.

A method has been established using MRI to record the tibiofemoral contact pattern bilaterally, in subjects performing a closed-chain leg-press (Chapter 4; Scarvell et al. 2003a). This study aimed to use the established MRI method to record the tibiofemoral contact pattern preoperatively in injured and healthy contralateral knees and

postoperatively at 12 weeks and 2 years, to determine whether knee reconstruction restores normal knee kinematics.

Method

Twenty subjects with a unilateral ACL injury were recruited. Eight subjects were male, 12 were female. Subjects were aged 19 to 52 years (33 ± 7 yrs; mean \pm SD). The injuries were sustained 1 to 36 months earlier (7 ± 10 months). There were 12 right knees injured and 8 left knees. Subjects were injured at soccer (7), netball (4), basketball (2), skiing (2), martial arts (2), football (1), motor bike accident (1) and a fall from 1m height (1). All ACL injuries were diagnosed clinically by an orthopaedic surgeon and confirmed later at arthroscopy. Eighteen of these subjects were included in the study of ACL-injured knees (Chapter 5; Scarvell et al. 2003c). Because two subjects from the group in Chapter 5 declined knee reconstruction surgery, two additional subjects were recruited to the study. All subjects were available for follow up at 12 weeks postoperatively and 18 were available at 2 years. Of the two subjects lost to follow up at 2 years, one had moved internationally and one had further surgery for cartilage transplantation and was therefore excluded from the study. Subjects were excluded if there were any contraindications to MRI, may have been pregnant or they were over 180cm tall (to permit knee flexion in the MRI tunnel). Subjects were also excluded if history of injury or symptoms were present in the contralateral knee, so that the healthy contralateral knee could act as the control.

Twelve healthy subjects were used as controls for the comparison of knee kinematics at 2 years. These subjects are described in Chapter 4. These healthy subjects were aged 20 to

50 years, 7 were male and 5 female. None had any symptoms or history of injury in either knee.

All subjects provided informed consent. Ethics approval for the study was obtained from the University of Sydney and Australian Capital Territory Department of Health Human Research Ethics Committees.

Surgical procedure

Knee reconstruction was performed by four orthopaedic surgeons using the same technique. A quadrupled graft of semitendinosus and gracilis was harvested and the intraarticular procedure performed arthroscopically. The loops of tendon were anchored proximally by suspension over a transverse femoral pin within the femoral tunnel and anchored in the tibial tunnel by a bioabsorbable interference screw and finished with two tibial staples. Postoperative rehabilitation enabled full weight-bearing as tolerated, with accelerated rehabilitation in physiotherapy to encourage full range of motion, hamstrings and quadriceps strengthening via closed-chain exercises and proprioception training. Return to sport was permitted at 6 months (Shelbourne and Nitz 1990; Fu et al. 1992; Escamilla et al. 1998; Beynnon et al. 2002).

Surgical outcomes

Surgical outcomes for this study were measured by the passive anterior laxity of the knee and the Cincinnati knee rating scale. Passive anterior laxity was measured for each subject preoperatively, at 12 weeks and 2 years postoperatively using a KT1000 knee arthrometer (Medmetric, San Diego, California). The difference between the injured knee and healthy

contralateral knee was recorded at 15, 20 and 30lb of anterior drawer, on activation of the quadriceps muscles and on a maximum manual anterior drawer (by the same tester JMS), according to the protocol established by Daniel et al (1985). Pathological anterior laxity both before and after surgery is defined by a side-to-side difference of 3mm or greater on maximal manual application of anterior drawer (Daniel et al. 1985). Passive laxity of greater than 3mm side-to-side difference is considered a poor surgical outcome (Shelbourne and Nitz 1990).

The Cincinnati knee rating system includes measures of symptoms, difficulty with activities of daily living, examination findings, quantified ligament laxity, pivot shift and radiological findings. The result is a total score out of 100, where 100 indicates a fully functional and healthy knee. There is also a rating of excellent through good, fair and poor, and this indicates the surgical outcome. If a subject has a rating of “poor” for any one of the aspects, for example, passive laxity measurement, a final rating of “poor” is found. The Cincinnati rating system has been validated for use in ACL-injured and reconstructed populations (Barber-Westin et al. 1999). Subjects also reported the perceived condition of their knee on a 10 point Likert scale, with descriptors of poor (0-1/10) through good (5/10) to excellent (9/10).

Tibiofemoral contact point measurement from MRI scans

To study kinematic effects of ACL injury, subjects performed a supine leg-press between 0° and 90° knee flexion in the same manner as described in Chapter 4. The leg-press was weighted by a 150N load via a rope and pulley to resist leg extension. Sagittal images were generated through both knees simultaneously using MRI. The position of the tibiofemoral contact was recorded as the distance from the posterior tibial cortex to the

point of tibiofemoral contact for both the medial and lateral femoral condyles respectively (Scarvell et al. 2004).

Statistical Analysis

Tibiofemoral contact patterns of healthy contralateral and injured knees were compared using a repeated measures ANOVA model. The model was complex, with repeated measures performed at the seven intervals of knee flexion (0°, 15°, 30°, 45°, 60°, 75°, 90°) and at three time frames (including preoperatively, 12 weeks and 2 years postoperatively). To analyse the effects of surgery on tibiofemoral contact patterns three separate analyses were performed. The ACL-injured knees were compared to the contralateral knees at the preoperative time point. The ACL-reconstructed knees were compared to the contralateral knees at the 12 weeks time point. Finally, the ACL-reconstructed knees were compared to the contralateral knees at the two years time point.

To compare the tibiofemoral contact pattern of the injured and healthy knees longitudinally two analyses were performed. For this analysis the tibiofemoral contact point data at seven intervals from 0° to 90° was collapsed to a single mean of tibiofemoral contact. The mean contact of ACL-injured knees were compared at the preoperative, 12 weeks and 2 years time points. The contralateral knees were compared at preoperative, 12 weeks and 2 years time points. The data recorded for 12 healthy control subjects reported in Chapter 4 (Scarvell et al. 2003a) were included for analysis of changes in the healthy knee over time. Performing separate analyses enabled the effects of time and surgery to be examined independently, but increased the likelihood of type 1 error. If differences in kinematics were found, a Bonferroni comparison was used for post hoc analysis. The

significance level was set at $p = 0.05$. Data analysis was performed on Statistical Package for the Social Sciences version 11.5.

Results

Surgical outcomes

The outcome of knee reconstruction surgery was measured using the passive anterior laxity of the knee and the Cincinnati knee rating scale. KT 1000 showed a side-to-side difference in passive anterior laxity of 5.1 ± 2.6 mm (mean \pm SD) preoperatively, 2.5 ± 2.2 mm at 12 weeks and 2.1 ± 2.3 mm at 2 years (Figure 7.1). Preoperatively there were 18 subjects with ≥ 3 mm anterior laxity on manual maximum anterior drawer, at 12 weeks there were 8 subjects with ≥ 3 mm side-to-side difference and at 2 years postoperatively there were 6 subjects with ≥ 3 mm side-to-side difference.

The preoperative Cincinnati scores averaged 57 ± 11 out of 100, with 8 knees rated as “poor” and 12 as “fair” ($n = 20$). No subjects rated “good” or “excellent”. At 12 weeks postoperatively the mean score was 76 ± 10 , with 4 knees rated as “poor”, 12 as rated “fair”, 4 rated as “good” ($n = 20$). No subjects rated “excellent”. At 2 years postoperatively the mean Cincinnati score was 90 ± 11 . At 2 years, the surgical outcome defined by the Cincinnati rating scale rated 5 subjects as “fair”, 5 subjects as “good” and 8 subjects as “excellent” ($n = 18$). The reasons for 5 subjects being rated as “fair” at 2 years included passive anterior laxity measures of ≥ 5 mm side-to-side difference ($n = 2$), restricted activity levels ($n = 2$) and medial compartment narrowing on x-ray ($n = 1$).

Passive anterior laxity

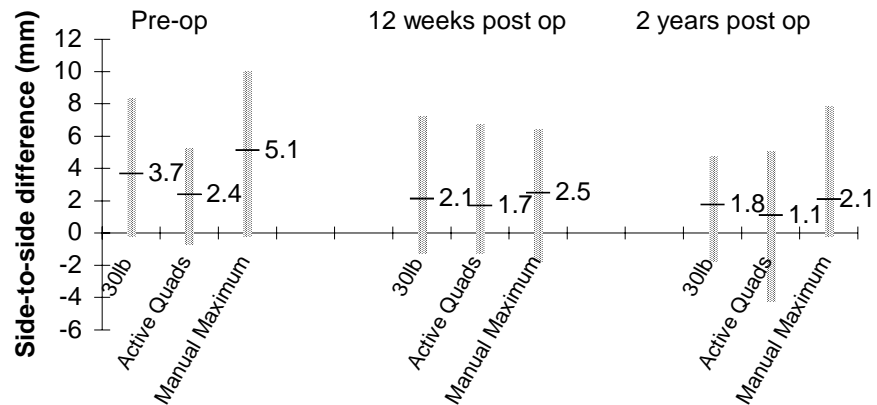


Figure 7.1: Passive anterior laxity measurements recorded preoperatively, at 12 weeks and 2 years postoperatively (mean and range). Side-to-side differences were measured with a KT1000 using a 30lb force, active quadriceps muscle contraction and a maximum manual anterior force.

For the self-reported condition of their knee 2 years postoperatively, 12 subjects rated the knee as “excellent” (9 or 10/10), 4 as “very good” (8 - 6/10) and 2 as “good” (5/10). No subjects rated the knee less than 5/10. Preoperatively 58% were participating in sports at least once per week. At 2 years, 69% of subjects were participating in sports at least once per week. There were no professional players in this group, however, those playing competitive amateur sports had all returned to their preferred sport.

Preoperative tibiofemoral contact patterns in healthy contralateral knees

The tibiofemoral contact pattern of the healthy contralateral knees differed between the medial and lateral compartments ($F_{(1,156)} = 321.6, p < 0.001$). At 0° flexion, the medial femoral condyle contacted the anterior tibial plateau 33 ± 3.9 mm from the posterior tibial cortex reference point. From 0° to 30°, the femoral condyle rolled back on the tibial

plateau so that at 30° it was 23.3 ± 3.3 mm from the posterior tibial cortex. Between 45° and 90° posterior movement was minimal (0.4mm) remaining centred on the tibial plateau.

The lateral contact pattern showed more posterior excursion than the medial contact pattern. The femur did not contact the tibial plateau as far anteriorly as the medial condyle, the contact point being 25.8 ± 3.1 mm from the posterior tibial cortex. Between 0° and 90° the femur rolls back steadily across the tibial plateau. At 30° knee flexion the femur had rolled back 7mm and was 18.8 ± 2.0 mm from the posterior tibial cortex and at 90° it was 14.4 ± 2.7 mm from the posterior tibial cortex.

Preoperative tibiofemoral contact patterns in ACL-injured knees

The ACL-injured knee had a similar contact pattern to the healthy contralateral knee, but it occurred more posteriorly on the tibial plateau ($F_{(1,152)} = 6.2$, $p = 0.014$) (Figure 7.2). The medial compartment was similar to the healthy contralateral knee; at 0° knee flexion the femur was positioned anteriorly on the tibial plateau 32.5 ± 5.0 mm from the posterior tibial cortex, and rolled back to the centre of the tibial plateau to 22.8 ± 2.9 mm from the posterior tibial cortex at 30° knee flexion and rolled back a further 2.7mm between 30° and 90° knee flexion, so that at 90° flexion it was positioned 19.1 ± 2.3 mm from the posterior tibial cortex.

The lateral condyle rolled further back on the tibial plateau than did the medial condyle. At 0° the femoral condyle was positioned 24.7 ± 4.3 mm from the posterior tibial cortex, then rolled posteriorly to 18.2 ± 2.3 mm at 30° knee flexion. It continued rolling back to finish 12.5 ± 2.8 mm from the posterior tibial cortex at 90°.

In ACL-injured and healthy contralateral knees, the medial and lateral compartments of the knee had different contact patterns, demonstrating longitudinal rotation of the knee ($F_{(1,136)} = 315.42$, $p < 0.001$). The effect of ACL injury on the contact pattern was not significantly more pronounced in the medial or lateral compartments ($F_{(1,152)} = 1.2$, $p = 0.27$). The mean difference in the tibiofemoral contact points between the ACL-injured knee and the healthy contralateral knee was 0.5mm for the medial compartment and 1.3mm for the lateral compartment.

The loaded condition did not produce a different tibiofemoral contact pattern to the unloaded condition, in either the healthy contralateral or ACL-injured knees ($F_{(1,136)} = 0.76$, $p = 0.386$).

Tibiofemoral contact patterns in knees at 12 weeks after reconstruction

Twelve weeks after knee reconstruction surgery, the tibiofemoral contact pattern in the ACL-reconstructed knees was no longer different from the healthy knees ($F_{(1,152)} = 2.5$, $p = 0.117$). The medial compartment was different from the lateral compartment ($F_{(1,152)} = 122.6$, $p < 0.001$). There was no difference in the tibiofemoral contact pattern of the reconstructed knees between the loaded and unloaded conditions at 12 weeks post surgery ($F_{(1,152)} = 1.3$, $p = 0.256$) (Figure 7.3).

Tibiofemoral contact patterns 2 years after reconstruction

Two years after ACL reconstruction, the tibiofemoral contact pattern in the ACL-reconstructed knees was the same as that of the healthy contralateral knees ($F_{(1,136)} = 0.013$, $p = 0.909$) (Figure 7.4). The medial compartment was different from the lateral

compartment ($F_{(1,136)} = 122.6$), $p < 0.001$). The loaded and unloaded knees' tibiofemoral contact patterns were not different ($F_{(1,136)} = 1.3$, $p = 0.256$).

Changes in tibiofemoral contact patterns to healthy knees over time

There was a significant difference in the contact pattern of the healthy contralateral knees at the three time points, pre-operatively, at 12 weeks and 2 years post knee reconstruction ($F_{(1,204)} = 11.5$, $p < 0.001$). Post hoc tests showed the difference to be between 12 weeks and 2 years in the healthy contralateral knees ($p = 0.003$). There was no significant difference between the tibiofemoral contact patterns preoperatively and 12 weeks postoperatively ($p = 0.559$). The lateral compartment was most affected by the changes over time ($F_{(2,204)} = 42.5$, $p = 0.021$).

The preoperative contact pattern in the lateral compartment of healthy contralateral knees was similar to the contact pattern at 2 years at 0° and 15° knee flexion. However, from 45° to 90° knee flexion the lateral femoral condyle did not roll back as far on the tibial plateau as it had at preoperative testing. Preoperatively, the healthy knee contact position at 45° knee flexion was 16.0 ± 2.0 and at 2 years was 18.5 ± 2.3 mm from the posterior tibial cortex reference point. At 90° knee flexion the preoperative position was 14.4 ± 2.8 mm and at 2 years was 16.8 ± 2.3 mm from the posterior tibial cortex. At 2 years the lateral femoral condyle did not roll back as far on the tibial plateau as it had preoperatively, but the rollback of the medial femoral condyle was unchanged. This indicates that the knees had lost some of the longitudinal rotation evident during knee flexion at the preoperative measurement.

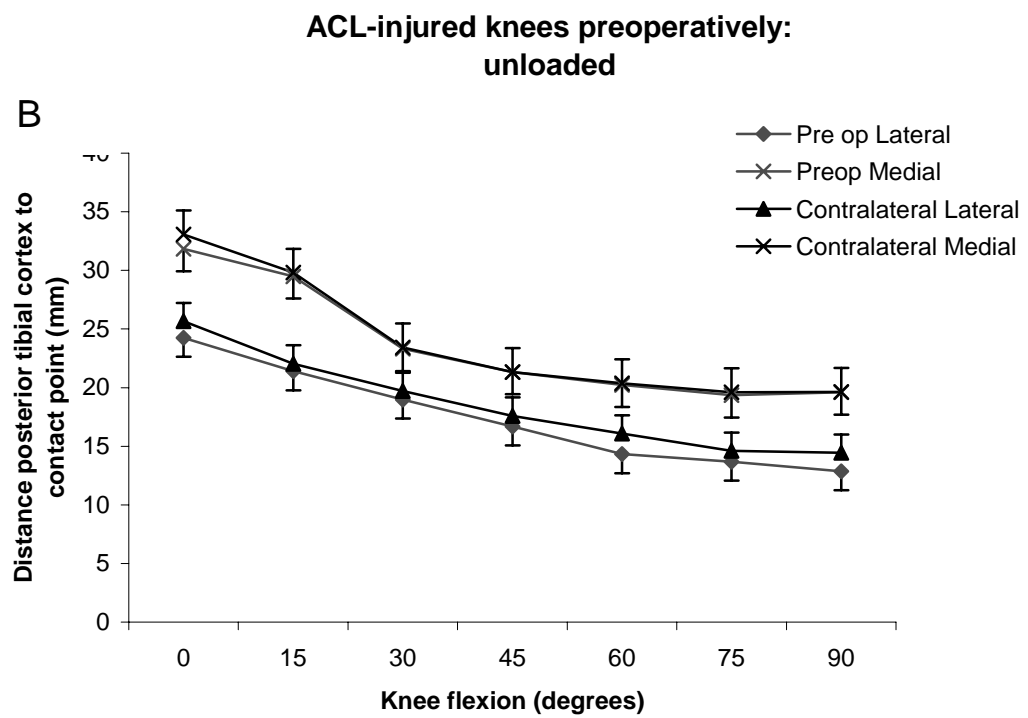
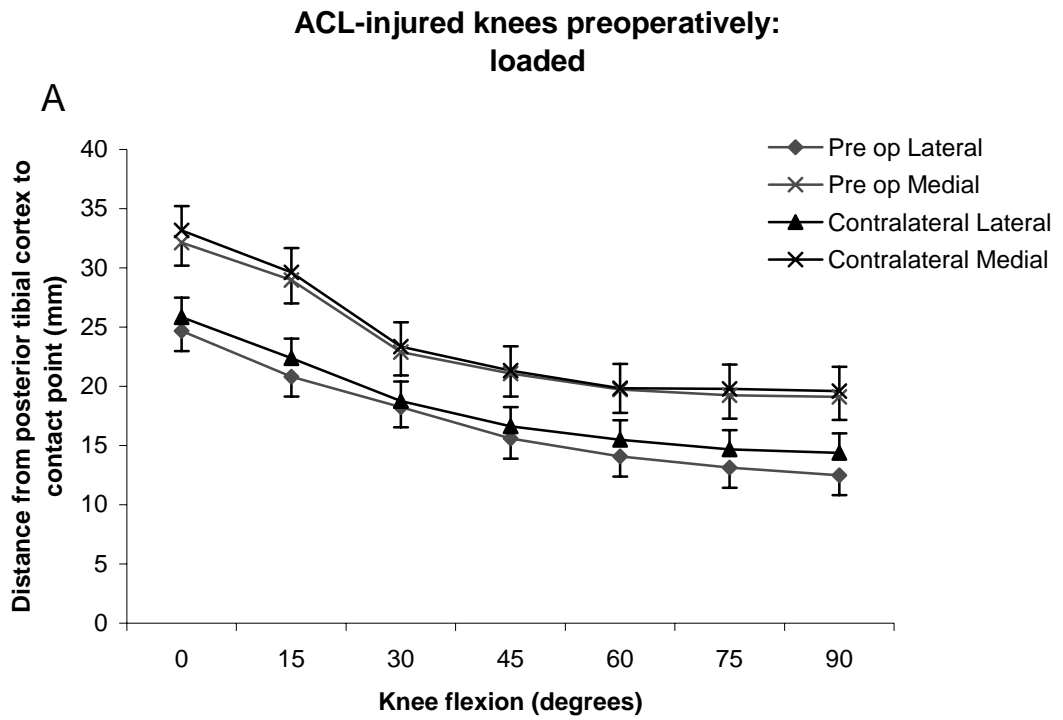


Figure 7.2: Tibiofemoral contact points in the ACL-injured and healthy contralateral knees preoperatively (Pre op) when A) loaded and B) unloaded (mean \pm SE).

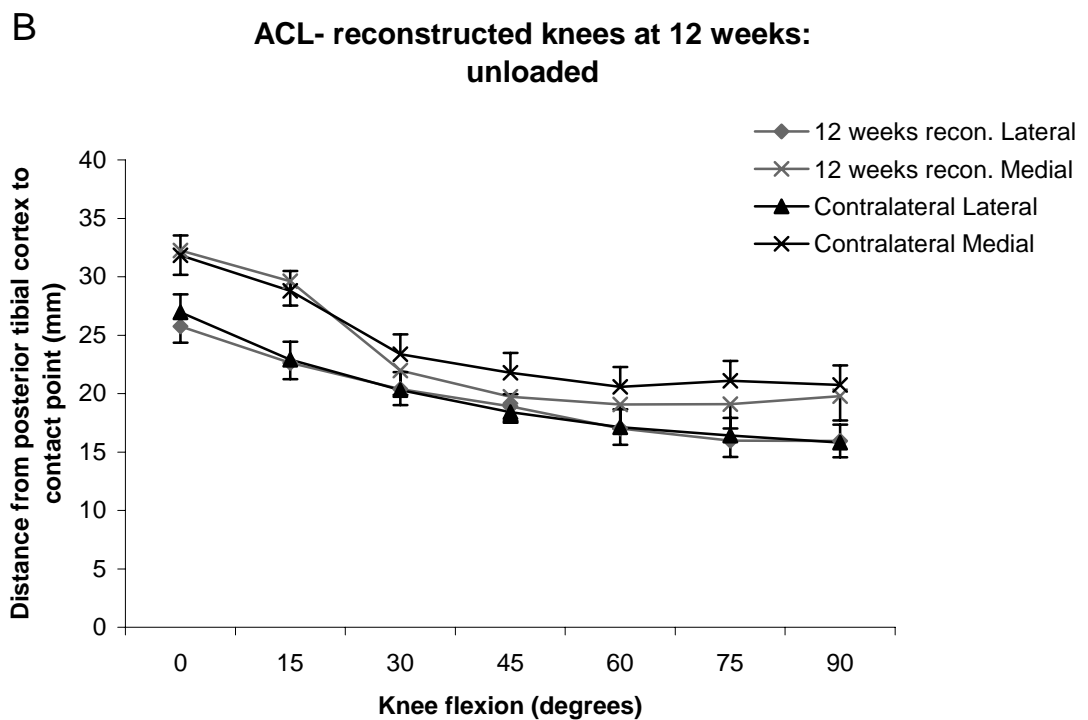
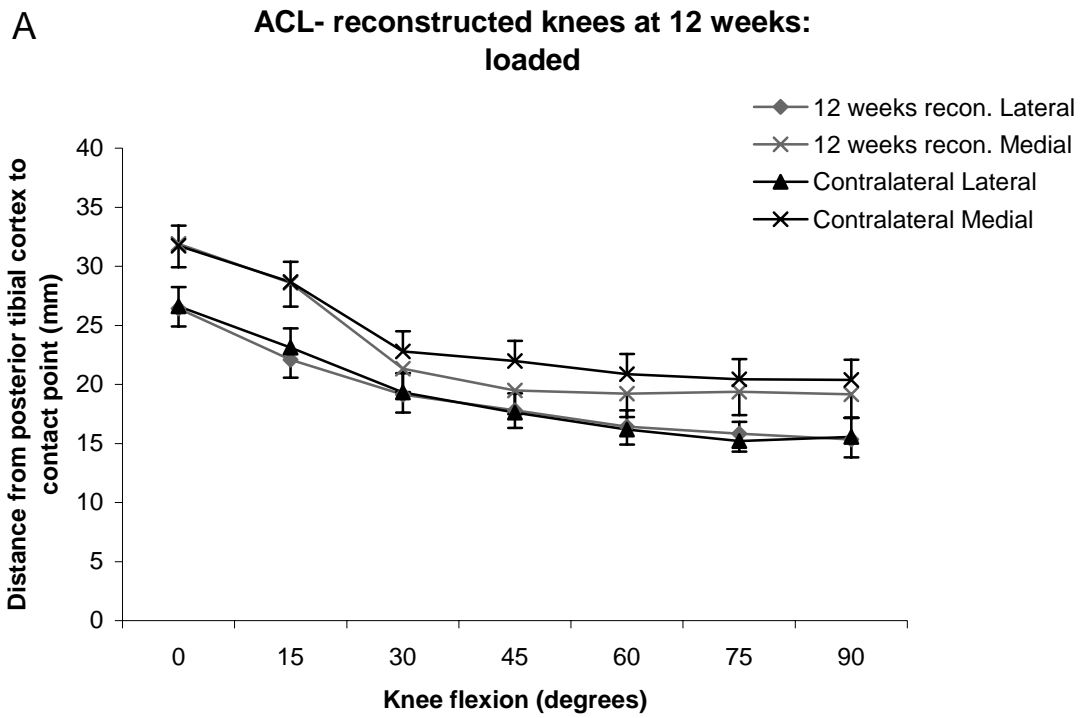


Figure 7.3: Tibiofemoral contact patterns in ACL-reconstructed and healthy contralateral knees 12 weeks after reconstruction (12 weeks recon.) (mean \pm SE) when A) loaded and B) unloaded. The difference between the ACL-reconstructed and healthy contralateral knees was not significant.

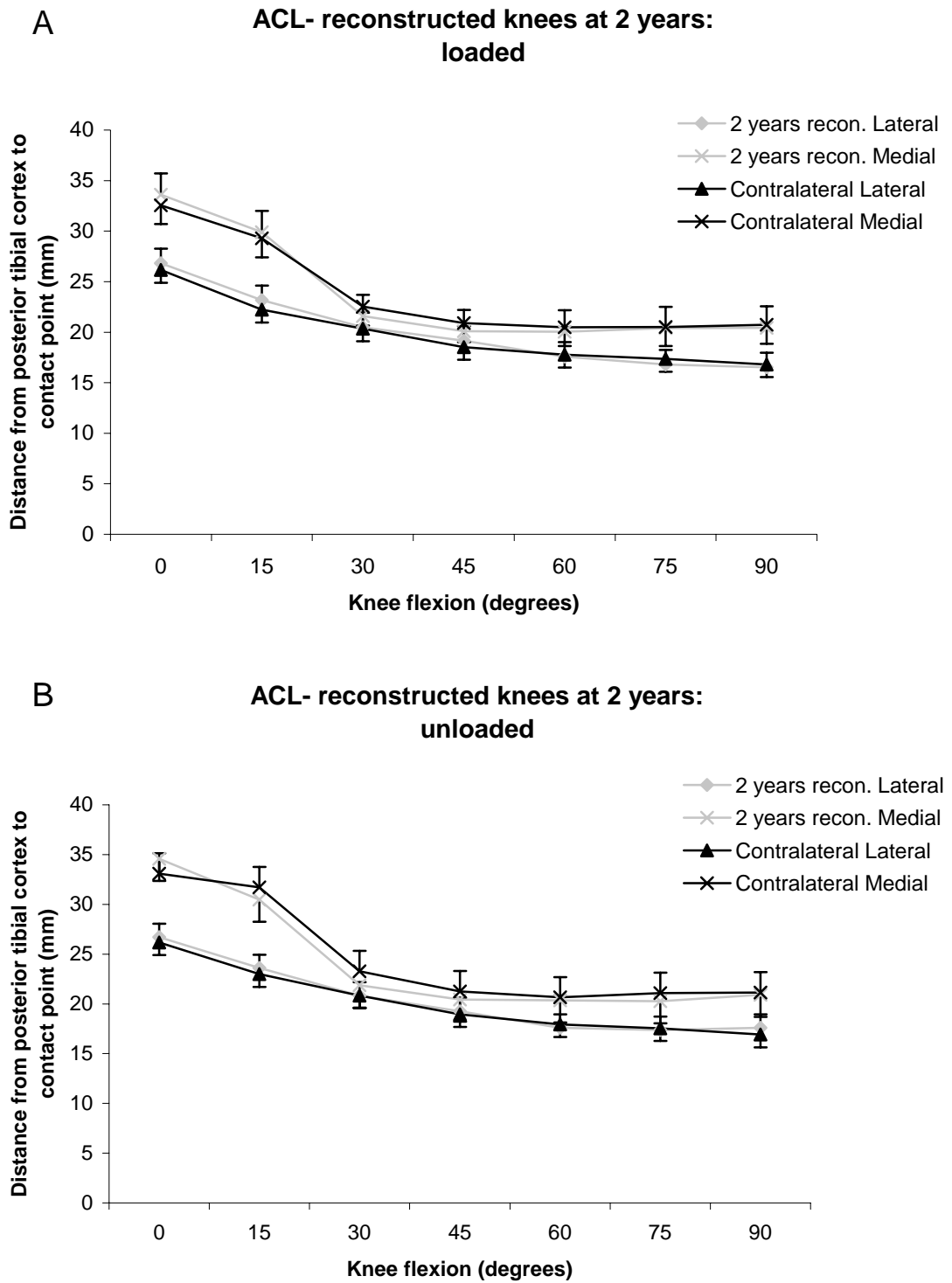


Figure 7.4: Tibiofemoral contact patterns in the ACL-reconstructed knees (2 years recon.) were the same as the healthy contralateral knees at 2 years (mean \pm SE) when A) loaded and B) unloaded.

Changes in tibiofemoral contact patterns to reconstructed knees over time

Changes over time also occurred in the reconstructed knees. Post hoc analysis showed difference in the contact pattern preoperatively and at 2 years postoperatively in the ACL-reconstructed knees ($p < 0.01$). The medial compartment contact pattern was unchanged but the lateral compartment progressively lost femoral roll back over the two years of the study. Preoperatively the femur rolled back to 12.5 ± 2.8 mm from the posterior tibial cortex at 90° knee flexion and at 2 years the femur rolled back to 16.5 ± 2.5 mm from the posterior tibial cortex.

Discussion

To examine whether the knee reconstruction surgery restored the kinematics of the operated knees, it is first necessary to know that the surgery was successful, ie. outcomes were similar to the international benchmarks. The generally accepted measure of surgical outcome is restoration of passive anterior laxity values to within the normal range, that is a side-to-side difference of ≤ 3 mm with an applied manual maximum anterior force (Daniel et al. 1985). Nevertheless, passive laxity is reported in a variety of different ways. Pinczewski et al (1997) reported that 75% of subjects had anterior laxity of ≤ 3 mm after reconstruction by hamstrings graft. O'Neill (2001) reported 75% of subjects with hamstrings graft reconstruction had ≤ 2 mm side-to-side difference. Shaieb et al (2002) reported 45% subjects had a side-to-side difference of ≤ 3 mm. Our result of 14/20 (70%) subjects with ≤ 3 mm side-to-side difference on maximum manual testing is consistent with international benchmarks for successful outcomes in hamstrings reconstruction.

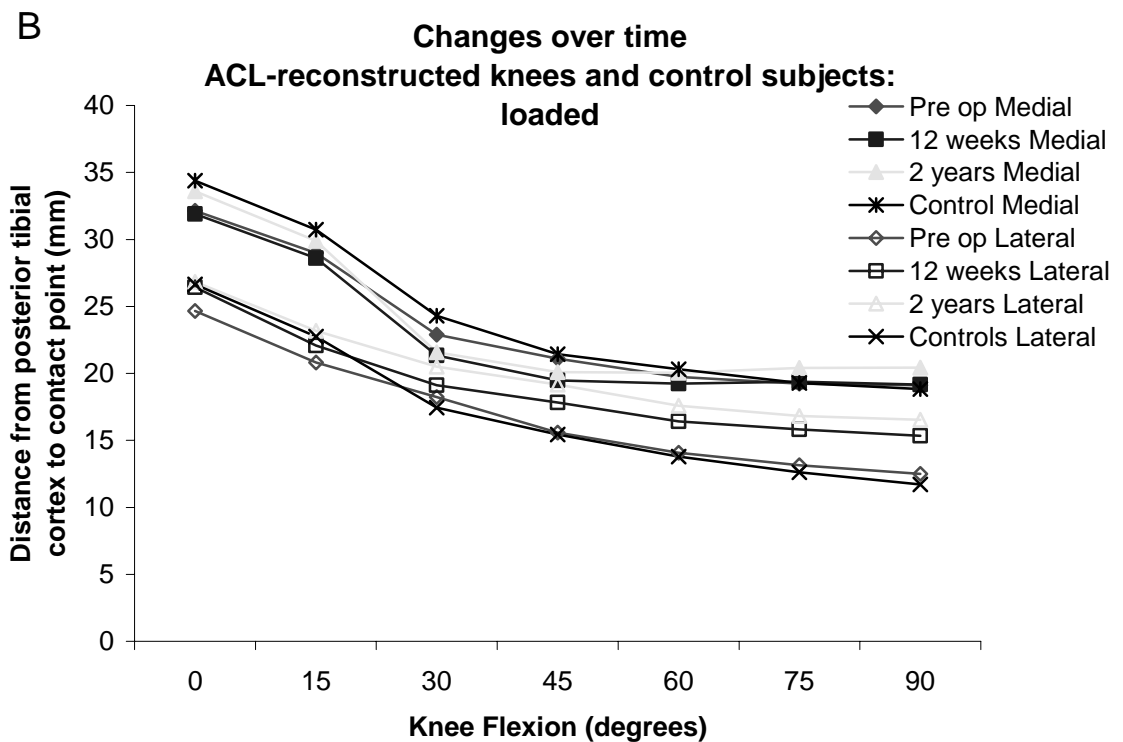
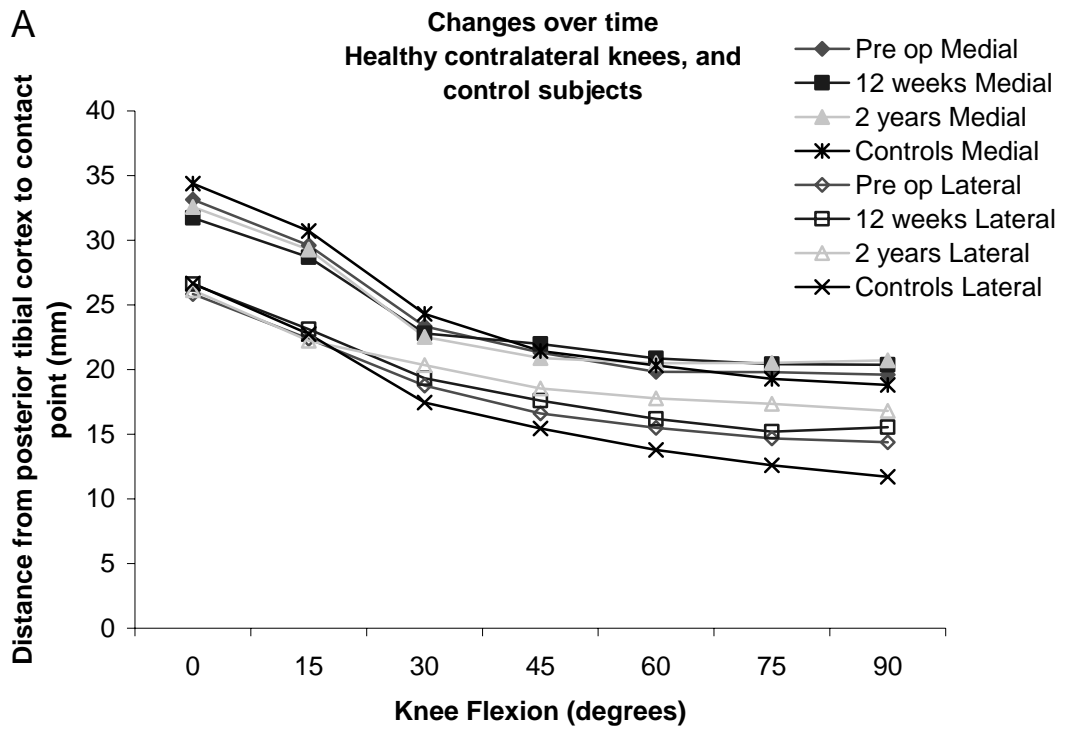


Figure 7.5: Changes over time in A) the healthy contralateral knees and B) ACL-reconstructed knees (mean \pm SE). This figure compares the healthy control subjects from Chapter 4 with the ACL-reconstructed subjects.

The Cincinnati scores for this study compare favourably with similar groups reported. The Cincinnati scores in our study were 76 ± 10 preoperatively and 90 ± 11 at 2 year follow up. This compares favourably with a similar group of ACL-reconstructed subjects ($n = 250$) who had 56 ± 9 preoperatively and 89 ± 11 at 2 year follow up (Barber-Westin et al. 1999); a group of patients with patella tendon reconstructions ($n = 44$) of whom 32 scored over 86/100 (Hrubesch et al. 2000); a group with ACL and high tibial osteotomy who scored 63 (mean) preoperatively and 82 at follow up (Noyes et al. 2000) and a group of subjects with chronic ACL deficiency (mean 7 years since injury) and consequent articular cartilage damage who scored 56 (mean) preoperatively and 86 at follow up (Noyes and Barber-Westin 1997). Our study included 6 subjects with side-to-side difference in passive anterior laxity $> 3\text{mm}$ which indicates the knee reconstruction was unsuccessful in restoring passive stability, according to Daniel's criteria (1985), however, the functional recovery of these subjects was very good, consequently they scored highly on the Cincinnati score, a measure of both functional and instrumented outcomes.

Kinematics results

Preoperatively the ACL-injured knees had a more posterior tibiofemoral contact pattern than the healthy contralateral knees. This shows that anterior laxity of the knee reported in vitro (Butler et al. 1980, Sakane et al. 1999) and measured in vivo by passive arthrometry (Daniel et al. 1985), is also present in the activity of a leg press and expressed in the altered contact pattern. Altered kinematics in the sagittal plane have also been described during walking (Dennis et al. 1996) and stepping up activities (Vergis et al. 1997 and Brandsson et al. 2001). It was important to confirm a difference in the tibiofemoral

contact pattern pre-operatively in order to provide a baseline from which to measure the effect of reconstruction surgery in normalising the knee kinematics.

Rotary instability of the ACL-injured knees, as reported *in vitro* (Sakane et al. 1997) and *in vivo* (Rudolf et al. 2001), could be expected to produce a difference in the medial/lateral contact patterns. The difference between the medial and lateral compartment contact patterns is evidence of the longitudinal rotation of the knee during flexion (Hill et al. 1999; Scarvell et al. 2003a), therefore, changes to the medial and lateral contact patterns indicate rotary aberrations. In the present study the difference between the healthy contralateral knee and injured knee appeared to be more pronounced in the lateral compartment than the medial compartments of the ACL-injured knees, though this failed to reach significance (Chapter 5; Scarvell et al. 2003c). Examples of the dynamic rotary instability have been reported in other activities, including a step up activity (Brandsson et al. 2001) and during swing phase of gait in ACL-injured subjects (Georgoulis et al. 2003), and this was reported as internal tibial rotation. As the tibia internally rotates the femur moves posteriorly on the tibial plateau, accordingly, our finding of a posterior tibiofemoral contact pattern in the lateral compartment could be a description of the same internal rotation.

Twelve weeks after ACL reconstruction the tibiofemoral contact pattern of the operated knee was not different from that of the contralateral healthy knee. There are no other studies that have examined kinematics at 12 weeks, however, gait analysis at 3 weeks after knee reconstruction has shown decreased flexion/extension range of motion during swing phase and decreased knee flexion during stance phase (De Vita et al. 1998), which had normalised by 6 months post surgery. Aberrant kinematics at 3/52 post surgery may

be a result of the acute healing process. In our study the kinematics of the reconstructed knee were no different to the contralateral knee 12 weeks post surgery.

The tibiofemoral contact pattern of the reconstructed knees at 2 years was the same as the healthy contralateral knees. This indicated that the knee kinematics, as assessed using this method, were restored to that of the contralateral knee by reconstruction surgery. The established MRI method we used demonstrated the difference in tibiofemoral contact pattern between injured and healthy contralateral knees preoperatively and this difference was no longer present at 2 years. The medial/lateral asymmetry of the knee was still evident in the difference in tibiofemoral contact patterns of the two compartments, demonstrating that longitudinal rotation occurred during knee flexion. These aspects of knee kinematics are consistent with the patterns of healthy control subjects (Chapter 4).

Other studies have not found that knee kinematics were restored by reconstruction (Brandsson et al. 2002; Beard et al. 2001). In an RSA study of 9 subjects 12 months following bone-patella tendon-bone reconstruction the tibial rotation and femoral translation still exhibited the same abnormal characteristics that had been present preoperatively; that is, the knees exhibited increased internal rotation at 55° and 0° flexion compared to the contralateral knee. Postoperatively, average internal rotation had decreased, however the change was not statistically significant. Brandsson et al (2002) concluded that knee reconstruction did not correct the aberrant knee kinematics exhibited preoperatively.

Several studies have reported normal kinematics after knee reconstruction (Bulgheroni et al. 1997; De Vita et al. 1998; Georgoulis et al. 2003). Kinematics recorded in these

studies included knee flexion /extension and longitudinal rotation. A cross-sectional study by Bulgerhoni et al (1997) compared healthy controls, matched for age and sex to ACL-injured and reconstructed subjects, finding that at 17 months, that kinematics of injured knees were different from healthy controls and reconstructed knees at were the same as healthy controls. These findings were supported by Georgoulis et al (2003), in a similar cross sectional study.

Knee kinematics, measured by anterior translation of the tibial tubercle during gait analysis, were not corrected by reconstruction surgery (Beard et al. 2001). Preoperatively there was no difference between the healthy and injured knee, but at 6 months postoperatively there was significantly more anterior tibial translation in the reconstructed knees. Of the 11 subjects, 6 had semitendinosus autografts and 5 had bone-patella tendon-bone autografts. The increase in tibial translation did not correlate with clinical outcomes of surgery, in this case the Lachman's test. There are several possible reasons for the increase in tibial translation postoperatively. At 6 months postoperatively the graft tissue may still be undergoing revascularisation and new collagen synthesis, and the anterior laxity of the knee consequently increased, though this should have been reflected in the Lachman's test results. Six months following surgery may be too soon to reflect the true outcome of surgery, because rehabilitation is still in progress in most cases and muscle strengthening and sensorimotor re-education is not likely to be complete (Neitzel et al. 2002). The finding of increased anterior tibial translation during gait in reconstructed knees is important because it may have implications for the choice of knee reconstruction for the long-term protection of the knee from degenerative sequellae and warrants further investigation.

Changes to kinematics over time

Changes to the healthy knee over the two-year period of the study are interesting. No other studies have examined kinematics of the healthy contralateral knees in the postoperative analysis. There is a suggestion that the kinematics of healthy contralateral knees of ACL-injured subjects are not the same as normal control subjects, implying that ACL-injured subjects have some intrinsic kinematic anomaly (Andriacchi and Birac 1992). However, this is the first reported longitudinal study of the healthy knee of ACL-deficient or reconstructed subjects with which to examine the long term change to the healthy knee.

The loss of lateral femoral condyle roll back in the lateral compartment of healthy contralateral knees over the 2 year period appears similar to the loss of roll back in the lateral compartment of knees with early osteoarthritis (Chapter 10). However, there were no symptoms of osteoarthritis or evidence on MRI in the healthy contralateral knees to suggest that this might be the case. Further study of the healthy contralateral knees over the long-term may clarify whether these changes are indeed indicative of early osteoarthritis.

Conclusion

Knee reconstruction by hamstrings autograft restored the tibiofemoral contact pattern to that of the healthy contralateral knee. The medial/lateral asymmetry of the knee indicated that longitudinal rotation occurred during the flexion movement. Loading does not alter the contact pattern of the knee. These kinematic characteristics are consistent with those

of the healthy knee. However, there appear to be changes to the kinematics of the ACL-reconstructed and healthy contralateral knees which may indicate that long-term changes to the kinematics of the knee occur as a result of ACL injury and subsequent surgery.

CHAPTER 8

KINEMATIC CONSEQUENCES OF CHRONIC ACL INJURY

Abstract

Late degeneration of the anterior cruciate ligament (ACL) deficient knee may be due in part to repeat injury, but also to aberrant kinematics altering the wear pattern at the tibiofemoral interface. Twenty-three subjects with a history of > 10 years (18 ± 8 years; mean \pm SD) ACL deficiency without knee reconstruction performed a closed-chain leg-press resisting a 150 N load. MRI scans were performed at 15° intervals from 0° to 90° knee flexion. The ACL-deficient knees had a posterior tibiofemoral contact pattern on the tibial plateau compared to the healthy knees ($p = 0.003$). The difference was most evident in the medial compartment ($p < 0.01$) and at 0° and 15° knee flexion. Articular cartilage damage in the medial compartment was related to the variation of the tibiofemoral contact pattern ($r = -0.53$). Articular cartilage damage was not related to time since injury. The kinematic consequences of chronic ACL injury may in part be responsible for the pattern of degenerative change in the knee, especially in the medial compartment

Introduction

It has long been recognised that anterior cruciate ligament (ACL) injury initiates a cascade of events frequently terminating in osteoarthritis of the injured knee (McDaniel and Dameron 1980; Clatworthy and Amendola 1999; Gillquist and Messner 1999). Factors responsible for degeneration of the knee may include chondral damage at the time of the original injury, repeated episodes of trauma due to instability of the knee and changes to the kinematics of the knee during routine activities. However, although several studies have examined the kinematics of the acute ACL-deficient knee (Vergis et al. 1997; Dennis et al. 1998; Vergis and Gillquist 1998; Brandsson et al. 2001), there is little known regarding the kinematics of chronic ACL-deficient knees (Wexler, 1998).

The cause of knee degeneration may be identified by its nature and location. Chondral impact injuries in association with ACL injury appear frequently as a kissing injury in the lateral compartment from a valgus blow to the knee (Myers et al. 2001). In animal models chondral impact injury has been used to precipitate degeneration of the chondral surface (Thompson et al. 1991a). Bone bruises visible on magnetic resonance imaging (MRI) scan are evidence that impact injuries in conjunction with ACL injuries may occur with sufficient force to cause bleeding in the subchondral bone (Johnson et al. 1998; Lahm et al. 1998; Johnson et al. 2000). It is not yet clear whether these bone bruises underlie areas of future chondral damage in humans (Stein and Fischer 1995). It may be that lateral compartment degeneration is due to chondral injury at the time of the original injury (Myers et al. 2001), but that medial

compartment degeneration is the result of other factors such as aberrant kinematics or subsequent knee injury (McDaniel and Dameron 1980; Finsterbush et al. 1990).

In the medial compartment of chronic ACL-deficient knees, degeneration of the menisci has been described as a major risk factor for osteoarthritis (Segawa et al. 2001). The medial meniscus has been shown *in vitro* to act as a secondary stabiliser of the knee during application of an anterior tibial force, resulting in additional load and shear forces on the meniscus (Allen et al. 2000). These increased loads may lead to degenerative failure and render the meniscus incapable of fulfilling the roles of shock absorption, cartilage nutrition and load distribution which protect the chondral surface (Keene et al. 1993; Bellabarba et al. 1997; Allen et al. 2000). A chronic ACL-deficient knee with medial meniscus incompetency is assured of pre-osteoarthritic change 27 years after injury and has an 80% risk of medial compartment osteoarthritis (Dejour et al. 1994).

Degeneration of the unstable ACL-deficient knee may also be due to aberrant kinematics altering loads and shear forces at the tibiofemoral interface. Even in routine daily activities such as stepping up, walking and lunging, the kinematics of the ACL-deficient knee are changed (Karrholm et al. 1988a; Karrholm et al. 1988b; Friden et al. 1993; Vergis and Gillquist 1998; Brandsson et al. 2001; Georgoulis et al. 2003). Altered kinematic patterns may change the area of loading and magnitude of shear forces at the tibiofemoral (TF) contact interface, enough to produce a repetitive micro trauma to the knee (Lane et al. 1994; Anderson and Dyhre-Poulsen 1997; Sakane et al. 1999; Allen et al. 2000). While ACL deficiency has been linked to patterns of tibial wear (Frankel et al. 1971; Harman et al. 1998),

it has not been shown specifically that TF contact patterns are related to areas of wear at the articular surface.

Studies of kinematics in the acute phase after ACL injury have described changes in the sagittal translation and longitudinal rotation of the injured knee during flexion (Karrholm et al. 1988a; Karrholm et al. 1988b; Friden et al. 1993; Vergis and Gillquist 1998; Brandsson et al. 2001; Georgoulis et al. 2003). A gait analysis study describes sagittal translation associated with time since injury in chronic ACL-deficient knees (Wexler et al. 1998). The tibiofemoral contact pattern has not been studied. Measurement of knee kinematics by tibiofemoral contact mapping is particularly suitable in the case of chronic ACL deficiency, as events at the tibiofemoral contact surface have relevance to cartilage wear patterns and shearing.

The aim of this study was to compare the kinematics of chronic ACL-deficient knees with healthy contralateral knees, using magnetic resonance imaging (MRI). The relationship between kinematic changes, as reflected by the TF contact pattern, time-since-injury, articular cartilage damage and meniscal damage were explored.

Method

Subjects

Twenty-five subjects were recruited with a history of ACL injury at least 10 years previously. Two subjects were excluded following diagnostic MRI, one because an ACL tear was not visible and one with a concomitant posterior cruciate ligament tear. The remaining 23 subjects

were aged between 31 and 67 years (45 ± 9.8 years) and 11 were male, 12 were female (Table 8.1). The injury was sustained between 10 and 35 years previously (17.8 ± 1.5 years). Twenty-one subjects were injured during sports and 2 in low level falls. Four were injured while skiing, 6 while playing various codes of football, 2 during netball, 3 during basketball and one each while dancing, wrestling, tobogganing, playing tennis, gymnastics and hockey. Six injuries were classified as contact injuries, in that the knee had been struck by an object or another person. The remaining 17 were non-contact injuries. Twelve subjects had arthroscopic reconstruction of the ACL at the conclusion of the study. ACL tear was confirmed by MRI scan for every subject.

Subjects were excluded if there were any contraindications to MRI, if they may have been pregnant or they were over 180cm tall (to permit knee flexion in the MRI tunnel). Subjects were also excluded if history of injury or symptoms were present in the contralateral knee, so that the uninjured knee could act as a matched control. All subjects provided informed consent. Ethics approval for the study was obtained from the Australian Capital Territory Department of Health and University of Sydney Ethics Committees.

Table 8.1: Demographic and injury characteristics of subjects. All subjects had chronic ACL deficiency of at least ten years standing.

	Age	Sex	Injured knee	Time since Injury (yrs)	Injuring sport	Mechanism of injury	Arthroscopy following study	Total Cincinnati knee score /100	Symptoms /40	Sports Participation	Self-reported grade/10	KT1000 Side-to-side difference
1	55	F	R	18	Netball	Non contact	No	61	26	75	8	8.0 mm
2	49	F	R	35	Running fall	Non contact	No	74	30	75	9	5.5 mm
3	33	M	L	14	AFL football	Contact	No	74	32	85	8	2.0 mm
4	34	F	L	11	Netball	Non contact	Yes	64	28	55	4	3.5 mm
5	33	M	R	11	Rugby league	Contact	Yes	51	16	55	2	7.0 mm
6	41	M	L	19	Dance /landing	Non contact	Yes	51	14	95	2	4.0 mm
7	47	F	R	26	Tobogganing	Non contact	Yes	59	20	75	5	0.5 mm
8	42	F	R	17	Hockey	Non contact	Yes	81	16	95	4	0.0 mm
9	37	M	L	22	Jump / landing	Non contact	Yes	64	28	55	8	4.5 mm
10	38	M	L	24	Wrestling	Contact	Yes	46	16	85	2	3.5 mm
11	39	M	L	18	Basketball	Non contact	Yes	52	22	80	3	7.0 mm
12	31	F	L	16	Gymnastics	Non contact	Yes	63	34	100	4	4.0 mm
13	62	F	R	15	Tennis	Non contact	Yes	47	12	40	4	2.0 mm
14	67	M	L	16	Skiing	Non contact	No	80	39	95	9	11.0 mm
15	38	M	R	23	Rugby	Contact	Yes	55	20	40	3	4.0 mm
16	49	M	L	18	AFL football	Contact	No	54	28	75	6	3.0 mm
17	53	M	R	20	Touch football	Non contact	No	56	34	80	6	5.5 mm
18	45	M	L	21	Rugby league	Non contact	No	47	20	55	2	10.6 mm
19	50	F	L	19	Basketball	Non contact	Yes	66	28	75	7	5.3 mm
20	46	F	R	10	Skiing	Non contact	No	60	22	90	6	6.0 mm
21	36	F	R	17	Basketball	Contact	No	76	32	80	7	3.8 mm
22	59	F	R	10	Skiing	Non contact	No	44	7	90	6	4.3 mm
23	46	F	R	10	Skiing	Non contact	No	74	24	60	6	1.5 mm

MRI procedure

The kinematics of the ACL-deficient knee was compared to the contralateral knee for each subject by taking an MRI of both knees while the subject performed a leg-press within the MRI field (Chapter 4; Scarvell et al. 2003a). Images were generated from 0° to 90° flexion at 15° intervals, with the legs relaxed (unloaded) and repeated with the legs resisting a 150 load. Elastic straps at the ankles and thighs maintained neutral tibial rotation and thigh adduction.

Imaging of both knees simultaneously was performed using a 1.5T Siemens Magnetron Vision (Erlangen, Germany). Spoiled gradient echo sequences were used to generate sagittal images in the same manner as described in Chapter 4. Reliability of this method of MR imaging of TF contact maps was previously tested in control subjects (ICC = 0.94; Scarvell et al. 2003a).

Tibiofemoral contact measurement

The position of the femoral condyle on the tibial plateau was recorded as the distance from the posterior tibial cortex to the point of contact of the tibia with the femur on both the medial and lateral condyles (Chapter 4; Scarvell et al. 2003a). Where contact occurred over a wide area, the area centroid was used. To account for variation in the size of subjects, the measurements of the tibial cortex to femoral contact point were normalised to mean tibial dimensions.

Clinical variables for knee injury

The passive anterior laxity of both knees, the functional level and the current level of sports participation of the subjects were recorded. The KT 1000 arthrometer was used to measure passive anterior laxity by quantifying the anterior displacement produced by the Lachman's manoeuvre (Daniel et al. 1985). Anterior tibial displacement was measured in both knees five times: at 15lb, 20lb, 30lb of force during anterior drawer, with a maximum manual force during anterior drawer and on active quadriceps muscle contraction (Chapter 5; Scarvell et al. 2003c). The passive laxity of the knee under a maximal manual anterior load demonstrated a side-to-side difference ranging from 0 to 11 mm (4.6 ± 2.8 mm, mean \pm SD). Four subjects had a side-to-side difference of less than 3 mm (Table 8.1), but all subjects had ACL tear confirmed by MRI. It is recognised that subjects with chronic ACL deficiency may guard the knee from displacement during testing by contraction of the hamstrings, despite careful application by the examiner.

The Cincinnati knee rating system (Barber-Westin et al. 1999) was used to measure the symptoms, functional level and details of the physical examination. The maximum Cincinnati knee score is 100 for normal knees with full function. Cincinnati scores averaged 60.2 ± 11 (range 44 to 81/100). The symptoms component of the score averaged 23 ± 8 (range 7 to 34/40). At this functional level a subject would be able to do light work or sports without symptoms, but no running, twisting or jumping. The subjects also rated the perceived condition of their knee on a visual analogue scale from 0 to 10, where 10 is normal, 5 is fair and 0 extremely poor. Subject self-rated scores were 5 ± 2 (range 2 to 9/10).

The level of sports participation was defined according to the frequency of participation and the type of sport: ranging from sport involving cuts, pivots and contact with other players, to swimming, cycling and walking. The highest score possible was 100, for sport of high demand played 4-7 times per week. A score of 20/100 indicated that activities of daily living were difficult and no sport participation and 0/100 indicated severe problems with activities of daily living, ie. on crutches and with full disability (Barber-Westin et al. 1999).

Details of joint damage

Joint damage in the ACL-deficient knees was measured from diagnostic MRI scan or at arthroscopy. Twelve subjects had knee surgery following participation in the study, enabling the area and depth of articular cartilage lesions to be documented at arthroscopy. Meniscal and ligament damage was also recorded. All 23 subjects had a diagnostic MRI. Damage was recorded using Noyes' system for recording articular cartilage damage out of 100, where 100 is no articular cartilage damage and 0 is full thickness lesions of at least 1.5cm diameter in all 3 compartments, ie. eburnated bone (Noyes and Stabler 1989).

The diagnostic MRI scans used were standard diagnostic sequences, including sagittal T1 and T2 weighted fast spin echo sequences, proton density, STIR, axial T2 and coronal T2 sequences. The images were reported by radiologists with particular expertise in MRI. For the 12 subjects who had both arthroscopic and MRI assessment of joint damage, the scores from the MRI and arthroscopic examinations were combined to give the worst case. Some partial thickness chondral damage may be missed or underestimated on an MRI scan, whereas arthroscopy is considered the gold standard for assessment of chondral and meniscal integrity

(Spiers et al. 1993; MacKenzie et al. 1996a; MacKenzie et al. 1996b; Carmichael et al. 1997; Lundberg et al. 1997; Potter et al. 1998; McCauley and Disler 2001). Therefore, damage reported by both MRI and arthroscopic examination was recorded as the worst scenario. For example, a partial depth lesion of 1cm diameter recorded at arthroscopy, but not visible on the MRI scan, was included in the combined score or where a lesion was recorded as 1.5cm diameter on MRI but 2.0cm at arthroscopy, was recorded as 2.0cm in the combined score.

Statistical analysis

Differences in the TF contact pattern between the loaded and unloaded conditions, the injured and healthy knee and between medial and lateral compartments of the knees were tested using repeated measures ANOVA, for knee flexion at 0°, 15°, 30°, 45°, 60°, 75° and 90°.

An association was sought between TF contact pattern in the healthy contralateral and chronic ACL-deficient knees and the damage evident in the chronic ACL-deficient knees, using a Pearson's r correlation. Relationships between sports participation and difference in kinematic pattern and joint wear were also examined using a Pearson's r.

Results

Kinematics of chronic ACL-deficient knees

The effect of loading on TF contact patterns in medial and lateral compartments

There was no difference in TF contact pattern between the relaxed condition and while performing a leg press against a 150N load, in either the injured or the healthy knees ($p = 0.247$). In both the healthy and ACL-deficient knees the medial TF contact pattern was different from the lateral pattern ($F_{(1,176)} = 196.4, p < 0.001$).

TF contact patterns in chronic ACL-deficient vs. healthy contralateral knees

The injured knees showed a different TF contact pattern to the healthy contralateral knee ($F_{(1,176)} = 9.2, p = 0.003$; Figure 8.1). The difference was more pronounced in the medial compartment. In the medial compartment with the knee loaded, the mean difference in contact points between the injured and healthy knees was 1.8 ± 3.2 mm ($p < 0.01$). This difference was largest at 0° and 15° knee flexion with differences of 2.9 mm and 2.8 mm respectively. In the lateral compartment, the mean difference between the healthy and injured knees was 0.24 ± 3.1 mm ($p = 0.19$), with the difference remaining negligible throughout knee flexion to 90° (Figure 8.2). A similar pattern was seen in the unloaded knees, the medial compartment of the injured knee demonstrating a more posterior contact area than the healthy contralateral knee ($p < 0.01$). This was less so in the lateral compartment ($p = 0.01$).

Tibiofemoral contact pattern of Chronic ACL-deficient knees

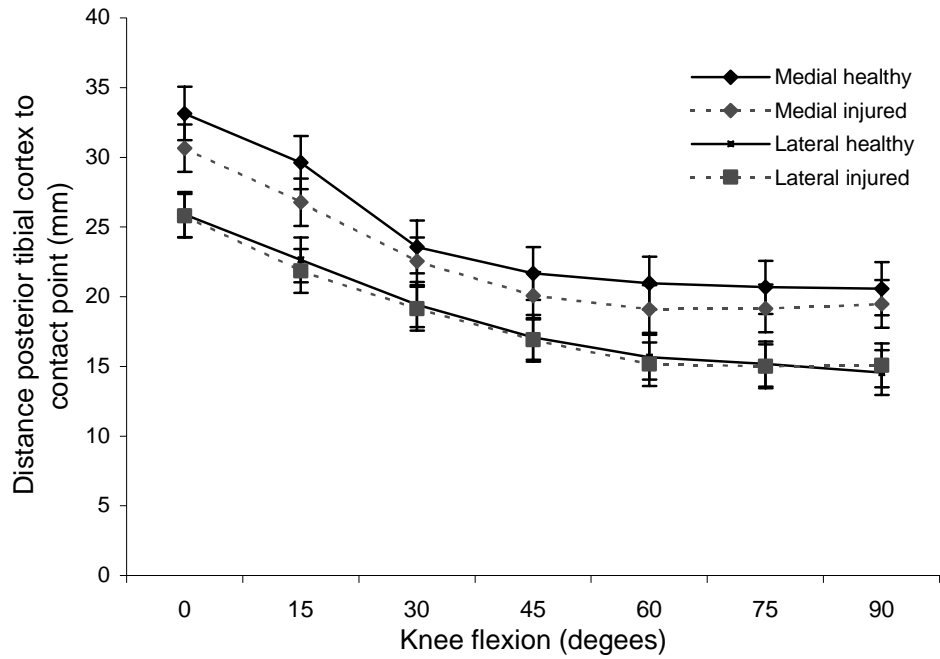


Figure 8.1: Tibiofemoral contact pattern for the loaded condition, comparing the healthy contralateral knees (black solid line) and chronic ACL-deficient knees (red broken line; mean \pm SE). The difference between healthy contralateral and chronic ACL-deficient knees was more pronounced in the medial compartment, with a more posterior position of contact on the tibial plateau.

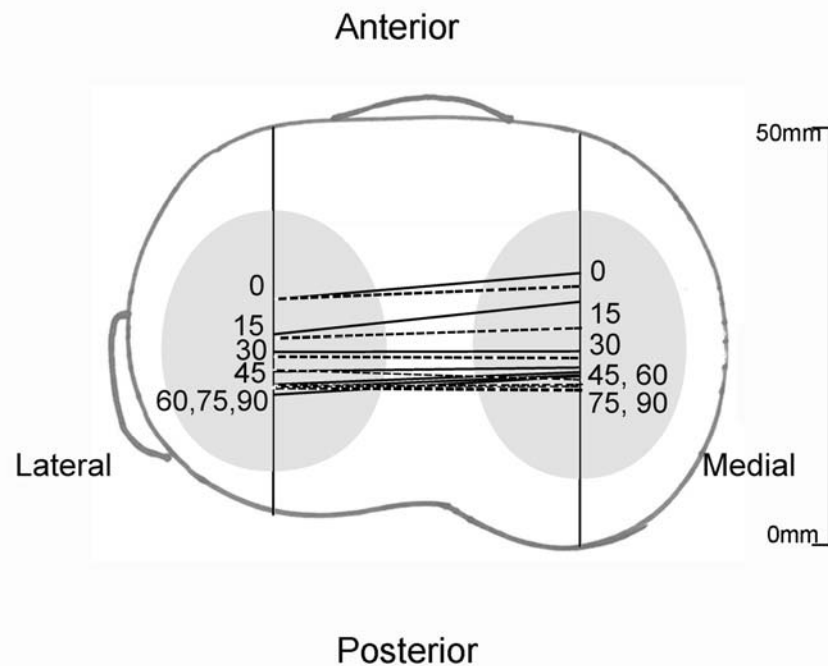


Figure 8.2: Diagram of the tibial plateau mapping tibiofemoral contact points between 0° and 90° of knee flexion for the loaded condition. The chronic ACL-deficient knees (broken line) showed a more posterior pattern of contact in the medial compartment than the healthy knees (solid line). In contrast, the lateral compartment showed a posterior contact pattern from 0° to 30°, but further into flexion, the TF contact was similar in chronic ACL-deficient and healthy contralateral knees.

Association between kinematic changes and joint damage

Damage to the articular cartilage in the chronic ACL-deficient knees reported from on MRI and arthroscopy was recorded as the combined joint damage scores of 72 ± 26 (range, 20 to 100). Five subjects had scores of 100, ie. no evidence of degenerative change. Sixteen subjects had degenerative changes in the medial compartment and 12 had degenerative changes in the lateral compartment. There was no significant difference between medial and lateral

compartments in the incidence or severity of degenerative change ($p = 0.788$). Medial meniscal damage (including tears, previous meniscectomy and degenerative tears) was identified in 16 subjects and lateral meniscal damage in 15 subjects.

Damage to the articular cartilage recorded at arthroscopy was associated with the side-to-side difference in TF contact points between the healthy contralateral and chronic ACL-deficient knees. In the medial compartment of the loaded knees ($n = 12$), the difference in contact pattern of the chronic ACL-deficient knees was moderately negatively correlated with the Noyes' scores for articular cartilage damage to the knees ($r = -0.45$). This indicates that the subjects with less joint damage (and higher Noyes' scores) had a tibiofemoral contact pattern similar to the healthy contralateral knee, while subjects with greater joint damage had a greater difference between the contact pattern of the ACL-deficient knee and the healthy contralateral knee.

Damage to the articular cartilage recorded from examination of diagnostic MRI scans combined with arthroscopic assessment was poorly correlated with the side-to-side difference in TF contact points between the healthy contralateral and chronic ACL-deficient knees ($r = -0.24$, $p = 0.26$; Figure 8.3). However, there were 3 outliers in this group of subjects. Three subjects had Noyes scores ≤ 40 /100, indicating marked osteoarthritic changes. For example, one subject with a Noyes score of 20 had a total knee replacement within 3 months of the study. With these 3 outliers excluded, there was a strong correlation between kinematic changes and knee joint damage ($r = -0.53$, $p = 0.01$). The 3 subjects with advanced osteoarthritis had a smaller magnitude of side-to-side difference in tibiofemoral contact pattern than the other chronic ACL-deficient subjects.

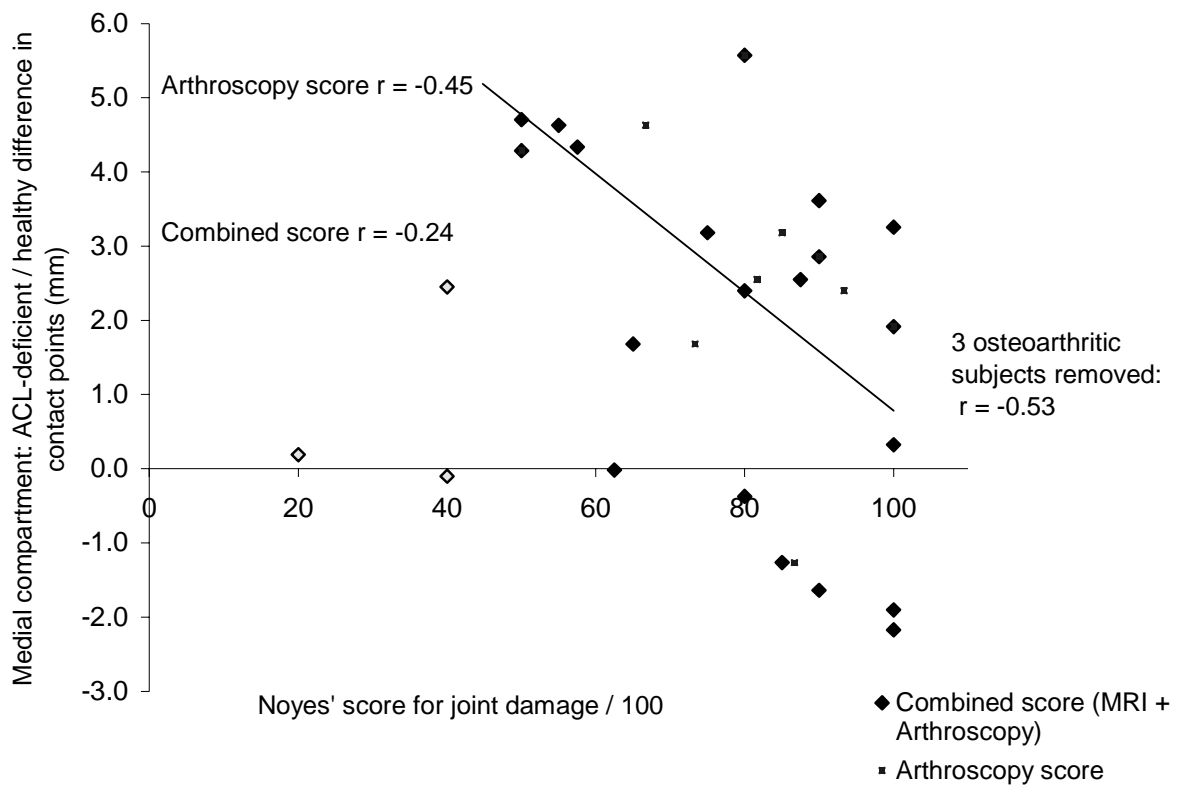


Figure 8.3: Correlation of knee kinematics in the medial compartment with articular cartilage damage in chronic ACL-deficient knees. The difference in contact pattern in the medial compartment between the chronic ACL-deficient and healthy contralateral knees was moderately correlated with joint damage recorded at arthroscopy ($r = -0.45$). There was a poor correlation with combined MRI and arthroscopy scores ($r = -0.24$). However, the association between joint damage scores and kinematic changes was stronger if 3 subjects with Noyes' scores ≤ 40 (hollow diamonds ◇) were excluded ($r = -0.53$).

Association between kinematic changes and clinical variables

Passive anterior laxity did not correlate with total Cincinnati knee scores, subjects' self-reported scores or with the component of the Cincinnati score that indicates severity of symptoms (pain, giving-way etc). There were moderate correlations for components of the Cincinnati knee score. The total Cincinnati score correlated with the symptoms score ($r = 0.63$) and with subject self-reported score ($r = 0.63$). Symptoms correlated with subjects' self-reported score ($r = 0.62$; Table 8.2).

Time since the original ACL injury did not correlate with subjects' symptoms ($r = -0.26$, $p = 0.458$), self-reported score ($r = 0.11$, $p = 0.770$) or total Cincinnati score ($r = -0.23$, $p = 0.519$). Time since injury was not correlated with degenerative damage to the knee, measured from MRI ($r = -0.373$, $p = 0.323$), arthroscopy ($r = -0.009$, $p = 0.981$) or total score for knee damage ($r = -0.16$, $p = 0.650$).

A higher Noyes score for joint damage from arthroscopy was strongly correlated with a lower sports participation level ($r = -0.60$, $p = 0.060$), that is, subjects with more joint damage currently played less sport.

Table 8.2: Correlations (Pearson's *r*) between knee joint damage, subjects' symptoms and sports participation. The Cincinnati score is a composite score including assessment of symptoms, self-report of condition of the knee and passive anterior laxity of the knee on a manual maximum anterior drawer, measured using a KT 1000 arthrometer.

Variable	Joint Damage		Time since injury	Sports participation	Cincinnati Score Components			
	MRI	Arthroscopy			Total Cincinnati score	Symptoms	Subjects' self-report	Passive laxity
Total Damage score	0.98**	0.88**	0.12	0.03	0.39	0.20	0.15	0.11
MRI score	x	0.58	0.12	0.10	0.39	0.22	0.08	0.10
Scope score		x	-0.01	-0.60*	-0.46	-0.35	-0.50	-0.01
Time since injury			x	-0.05	0.03	0.15	0.11	0.03
Sports participation				x	0.27	0.20	0.20	0.01
Cincinnati score					x	0.63**	0.63**	-0.14
Symptoms						x	0.62**	0.26
Subjects' self-report							x	0.09

Features of subjects with no joint damage

Five subjects (four female and one male) had no evidence of articular cartilage damage or meniscal damage. They were aged 46 ± 8 years. They had sustained the original injury 19 ± 10 years ago (range 10 to 35 years). Their current sports level was 66 ± 10 (range 55 to 75 out of a possible 100) ie. they ran, cycled or swam 1- 3 times per week, to 1-3 times per month. They

had all ceased sports requiring cutting, pivoting and running after the original ACL injury, but continued to keep fit in non-pivoting sports. The Cincinnati knee scores for this group were 66 ± 8 (range 61 to 74/100) i.e. “fair”. Their symptom levels ranged from 24 to 30 / 40, i.e. moderate work or sports involving running/ twisting or turning produced swelling, pain or giving way. Self-reported knee condition was 6.8 ± 2 out of 10 (range 4 (“fair”) to 9 (“almost normal”). The three subjects who were happy to continue with conservative management for the long term were health professionals. The two subjects who were not happy with the repeated episodes of instability and pain on activity subsequently had knee reconstruction surgery. The fourth subject with no joint damage was a chef and sustained the injury 10 years ago and the fifth was engaged in homeduties and sustained the original injury 11 years ago.

Discussion

Chronic ACL-deficient knees had an altered tibiofemoral contact pattern compared to healthy contralateral knees, particularly in the medial compartment of the knee. There are no other studies of tibiofemoral contact patterns in chronic ACL-deficient knees. In the recent ACL-injured knees, the tibiofemoral contact pattern was different from the healthy contralateral knees (Chapter 5; Scarvell et al. 2003c). However, the changes to the tibiofemoral contact pattern in the recent ACL-injured knees appeared to be greater in the lateral compartment, suggesting a shift in the axis of rotation towards the medial side of the knee or increased internal rotation of the tibia during flexion. Increased internal rotation of the ACL-injured

knee compared to the contralateral healthy knee was also noticed in subjects studied using roentgen stereophotogrammetric analysis (RSA) while stepping up onto a box (Brandsson et al. 2001). However, the present study is the first to describe altered tibiofemoral contact patterns in the medial compartment of chronic ACL-deficient knees in vivo.

Differences in contact pattern between the recent and chronic injuries could reflect long-term damage to the knee resulting from chronic ACL-deficiency. In the recently injured knees, the posterior TF contact pattern was more pronounced in the lateral compartment (Scarvell et al. 2003c) and in the chronic ACL-deficient knees the contact pattern changes were more pronounced in the medial compartment. One explanation for greater contact pattern changes in the lateral compartment of recent ACL-injured knees, is that in the absence of the ACL, the MCL becomes the primary restraint to both anterior motion and rotation of the tibia (Butler et al. 1980), together with a contribution from the medial meniscus. Thus, the long axis of rotation of the knee appeared to shift towards the MCL. The increased internal rotation of the knee is evidenced by the lateral femoral condyle contacting the tibial plateau in a more posterior position than in healthy knees. In chronic ACL-deficient knees, the secondary restraints to anterior and rotary tibial movement, the MCL and the medial meniscus, have probably assumed this role for many years (Butler et al. 1980; Allen et al. 2000) and it is possible that they have become attenuated. The TF contact pattern might have been more altered in the medial compartment than the lateral compartment, because the attenuation of the secondary restraints has reduced the structural stability of the medial compartment.

We did not find a correlation between time since injury and changes in knee kinematics. There is one other study of kinematics in chronic ACL-deficient knees, in which gait analysis

demonstrated increased anterior translation of the tibia in stance phase of walking and the magnitude of translation was associated with time since injury (Wexler et al. 1998). It was suggested that increased anterior translation indicated the attenuation of the secondary restraint structures of the joint. In our study the changes in kinematics were not associated with time since injury, but rather with articular cartilage damage.

The kinematic changes seen in the medial compartment contact pattern may produce more wear in the medial compartment, of both articular cartilage and medial meniscus. In the present study, there was an association between the extent of joint damage and the deviation of TF contact from that of the healthy knee in the medial compartment. This study cannot however, show causality. The pattern of damage in the medial compartment of the knee could be the result of increased load on secondary restraints in the ACL-deficient knee, causing shearing of the articular cartilage and trauma to the medial meniscus (Allen et al. 2000; Smith and Barrett 2001). One large meta-analysis found that the ratio of medial:lateral meniscal damage in chronic ACL-deficient knees was 70%:30% (Bellabarba et al. 1997). When the menisci are incompetent for load distribution, shock absorption or cartilage nutrition, the articular cartilage surface is subject to damage and the consequent degeneration is well described (Fairbank 1948; Tapper and Hoover 1969; Kettlekamp and Jacob 1972; Casscells 1978; Kurosawa et al. 1980). Thus, it appears that the changes over time occurring in the medial compartment are associated with altered medial compartment kinematics.

The role of shear forces in degeneration is poorly understood. There is controversy regarding the relative importance of joint load and shear forces at the joint surface in osteoarthritis. Some researchers deny that instability causes osteoarthritic changes (Burr et al. 1990), arguing

that the ACL-deficient knee does not develop osteoarthritic changes unless the menisci are damaged, i.e. an ACL injury in isolation does not initiate the osteoarthritic changes (Burr and Radin 1990; Radin et al. 1991). Others found using a dog model of ACL deficiency, osteoarthritic changes were present in all dogs at 34 weeks (Lopez 2003). Dejour et al (1994) concluded that osteophytes and superficial destruction of the cartilage are likely at 10 years after ACL injury and significant osteoarthrosis by 20 to 30 years. These rates are influenced by the damage to the menisci; that if both the ACL and medial meniscus are damaged, the rate of degeneration is higher than if only one structure is damaged. In order to identify the contribution of altered TF contact to the degenerative process independently of meniscal damage, a prospective longitudinal study recording details of meniscal damage and chondral damage is required. The incidence of chondral damage in the absence of meniscal pathology could then be measured.

The association between altered kinematics and wear in the medial compartment was much stronger after removal of 3 subjects with established osteoarthritic changes, indicating that the kinematic characteristics of osteoarthritic knees are different from chronic ACL-deficient knees. Indeed, the stages of knee injury and degeneration that have been studied in this thesis each have different characteristics. Recently injured knees, chronic ACL-deficient knees and knees with established osteoarthritic changes have distinctly different kinematic characteristics. In this study of chronic ACL-deficient knees, the medial compartment contact pattern is more posterior on the tibial plateau, but roll back of the lateral compartment is preserved. In knees with established osteoarthritis, reported in Chapter 10, the medial compartment contact pattern was more anterior on the tibial plateau; furthermore, the lateral compartment had lost roll back. Wada et al (1996) found that knee laxity was associated with

ACL deficiency in early osteoarthritis, but as the osteoarthritis progressed, knee laxity was no longer affected by ACL deficiency. While passive laxity does not correlate with the kinematics represented by the TF contact pattern (Chapter 5; Scarvell et al 2003c), the laxity may indicate that the ACL influences the osteoarthritic knee stability less as the disease progresses. The changes in TF contact patterns may indicate that the process of degeneration in ACL-deficient knees is marked by various stages, with different kinematic characteristics.

The correlation between kinematic changes and joint damage was much stronger using arthroscopic assessment of joint damage than MRI assessment. The sensitivity for detecting articular cartilage lesions with arthroscopy is better than MRI, so MRI scores underestimate the prevalence of articular cartilage damage. Arthroscopy has been considered the gold standard for assessment of articular cartilage damage, whereas MRI developed more recently for diagnosis of soft tissue injury and pathology. Early MRI sequences reported a sensitivity of 18% and specificity of 100% (Spiers et al. 1993). With the development of sequences and techniques better suited to articular cartilage imaging, sensitivity is now reported as 83-87% and specificity as 94-99% (Potter et al. 1998; Bredella et al. 2000; Murphy 2001). However, these techniques have been more successful for detection of focal cartilage defects occurring in conditions such as osteochondritis dissecans and less successful in assessment of chronic degenerative and generalised cartilage damage evident in osteoarthritis. In degenerative conditions the sensitivity of MRI is reported as 60% and specificity as 93.7% (Kawahara et al. 1998), but MRI is more sensitive to full thickness cartilage lesions. Sensitivity is greatly reduced for partial thickness lesions (Kawahara et al. 1998). The specificity of MRI for detection of cartilage lesions is higher than the sensitivity, indicating that lesions are more likely to be underestimated by MRI, than arthroscopy (Balkisoon 1996; Blackburn et al. 1996;

McCauley et al. 2001). With these factors taken into account the relationship between kinematic changes and articular cartilage damage may in fact be underestimated in this study.

We did not find a correlation between time since injury and joint damage. Myers et al. (2001) found in a retrospective study of 541 subjects, that chondral impact at the time of injury could account for lateral compartment damage, but that medial compartment changes, either to chondral cartilage or menisci, were proportional to time since injury. Bellabarba et al. (1997) also related medial compartment changes to time since injury. These retrospective studies support the association between chronicity of ACL deficiency and medial compartment degeneration and appear to give credence to the concept of disturbance in TF contact influencing degenerative change. The present study suggests that it is not time that influences degenerative change. It is altered kinematics.

Articular cartilage damage in chronic ACL-deficient knees is likely to be due to events at the tibiofemoral interface rather than secondary to meniscal impingement and attrition. The TF contact pattern of the ACL-deficient knees was approximately 2mm posterior to the pattern in healthy knees ($1.8 \pm 3\text{mm}$) in the medial compartment. The mobility of the medial meniscus is such that 2 mm of posterior femoral displacement should not cause meniscal impingement, because MRI studies have demonstrated 5.1mm posterior movement in vitro and 4mm in vivo during knee flexion (Thompson et al. 1991b; Vedi et al. 1999). The magnitude of the difference in TF contact pattern is too small to have impinged on the medial meniscus during this leg-press activity. The altered TF contact patterns and possibly the shift of rotation axis of the knee may be responsible for the long-term wear of the articular cartilage. It is likely that

the aberrant TF contact pattern influences degenerative changes in the knee independently of meniscal damage.

The level of sports activity was correlated with severity of degeneration, but this could be that, as a result of developing osteoarthritis, the subjects had reduced their level of participation. Several authors have recommended that ACL injury be treated conservatively if the patient is willing to cease competitive sports (Clatworthy et al. 1999; Gillquist et al. 1999). It may be that cutting and pivoting sports should be ceased as there is a higher risk of osteoarthritis once the menisci are damaged, especially in combination with ACL injury. However, there is insufficient evidence that swimming, cycling and other non-contact sports are associated with increased risk of osteoarthritis in ACL injury.

Conclusion

The TF contact pattern of chronic ACL-deficient knees was different from healthy knees. This difference was particularly evident in the medial compartment of the knee and particularly at 0° and 15° of knee flexion. These kinematic consequences of chronic ACL deficiency may in part be responsible for the pattern of degenerative change, but could be the result of degenerative change, particularly in the medial compartment of the knees.

CHAPTER 9

BONE MINERAL DENSITY CHANGES IN THE ANTERIOR CRUCIATE LIGAMENT INJURED KNEE.

Abstract

Testing of fixation devices in knee reconstruction has assumed that the injured knee has the bone mineral density (BMD) of the healthy adult knee. However, BMD in the injured knee is reduced due to inflammatory and biomechanical processes.

Dual energy x-ray absorptiometry (DEXA) was used to measure BMD in both knees of 20 adults with unilateral anterior cruciate ligament (ACL) injury (time since injury: 1-48 months). The test-re-test reliability of DEXA used for BMD measurement at the knee had a mean coefficient of variation of 18%. Femoral and tibial BMD was measured adjacent to the articular surface, and at the sites used for fixation in knee reconstruction in the tibia and femur.

BMD at the site of tibial fixation of the injured knees was $0.51 \pm 0.3\text{g/cm}^2$, which reflects a loss of density of 17% (+9 to -57%), $p=0.001$. The medial tibia recorded mean loss of 12% (+17% to -45%), $p= 0.005$, and the lateral tibia mean loss of 16% (+5% to -58%), $p = 0.001$. Femoral bone density loss was similar. Loss was correlated with time since injury, but not sex or age. At the site of tibial graft fixation 11/20 subjects had BMD below the critical value of 0.6g/cm^2 identified as the threshold for reliable graft fixation.

There is rapid and profound BMD loss in the ACL-injured knee.

Introduction

Bony fixation of the ACL graft is considered the weakest point of the quadrupled hamstrings tendon knee reconstruction in the early postoperative period. The autologous graft material itself is similar or stronger than the original ACL tissue (Brown et al. 1993; Fu et al. 1999; Hamner et al. 1999), and failure tends to occur instead at the graft/host bone interface (Steiner et al. 1994; Brown et al. 1996; Weiler et al. 2000). Early and accelerated rehabilitation trends have emphasised the need for reliable fixation in the initial weeks following surgery (Pena et al. 1996; Weiler et al. 1998; Brand et al. 2000).

Strength of the fixation is dependent on several factors, including type of fixation, and bone quality. Bone quality can be quantified by measurements of insertion torque of the interference screw and bone mineral density (BMD) of the regional cancellous bone (Pena et al. 1996; Brand et al. 2000). There is a demonstrated correlation between BMD and strength of fixation (Pena et al. 1996; Brand et al. 2000). However, much of the research in strength of graft/ bone fixation has been performed on elderly cadavers, and results adjusted to estimate fixation strength in young healthy adults (Brown et al. 1993; Steiner et al. 1994; Caborn et al. 1998). Calf femur and tibia sections, chosen for their resemblance to young bone have also been used (Butler et al. 1994; Brown et al. 1996; Weiler et al. 1998). Several studies have attempted to match the bony quality of cadaveric specimens to the age and physical characteristics of the population at risk of ACL injury (Rowden et al. 1997; Magen et al. 1999). Researchers have assumed that ACL-injured knees have the BMD of healthy adult knees and attempted to reproduce this bone quality in testing of fixation devices.

The assumption that ACL-injured knees have the BMD of healthy adult knees may be incorrect. For example, BMD in injured knees may be reduced due to inflammatory and biomechanical processes (Jarvinen et al. 1997). BMD of ACL-injured knees has been reported in only two studies, and was reported to have fallen following injury (Sievannen et al. 1994) and following surgery (Leppala et al. 1999). The study by Sievannen et al (1994) was a single case study, using a Norland DEXA scanner. The study by Leppala et al (1999), was a prospective study, comparing BMD in reconstructed knees with knees with a partial ACL tear, conservatively managed, so there was no pre-op measure of knees with a complete ACL tear. It appears, however, that extrapolation of data for graft fixation strength based on healthy BMD may overestimate the BMD of the injured knee (Jarvinen et al. 1997; Wohl et al. 2001). For correct estimation of the strength of fixation, it is important to know the BMD of the ACL-injured knee.

Dual energy x-ray absorptiometry (DEXA) provides a non-invasive and low radiation exposure method to quantify BMD *in vivo*. DEXA of the hip, wrist and lumbar spine is routinely used to assess BMD in patients at risk of osteoporosis with chronic illness (Mottet et al. 1996), post transplantation (Hamburg et al. 2000; Spira et al. 2000) and post menopause (Petley et al. 2000; Iki et al. 2001; Cure-Cure et al. 2002). Population data enables recorded BMD for a patient to be defined by the deviation from the young adult or age matched scores (Abrahamsen et al. 2001). DEXA is also used to evaluate the progression of osteoarthritis at the knee (Hurwitz et al. 1998; Andriacchi et al. 2000; Wada et al. 2001; Hulet et al. 2002), and bone remodelling processes after total knee arthroplasty (Petersen et al. 1996; Li et al. 2000; 2000; 2001). Forearm software is commonly used to analyse knee scans, as it accommodates for soft tissue, bone and air inclusion in the scan

field (Casez et al. 1994; Hurwitz et al. 1998; Leppala et al. 1999; Hurwitz et al. 2001), and has been shown to provide valid data.

The aim of this study was to examine changes in the BMD of the ACL-injured and contralateral healthy knee and in particular at the sites of fixation for knee reconstruction by autologus hamstrings graft.

Method

Twenty adults with ACL injury awaiting reconstruction were recruited and provided informed consent. Subjects were 6 male and 14 female, aged 19-38 years. Nine injuries were on the right side, and 11 on the left. Time since injury was 1-48 months (mean 8 months), and average time to reconstruction surgery was 10 weeks. Subjects were excluded if there was any history of injury or symptoms in the contralateral knee, tibia or femur, if they had past knee injury or reconstruction surgery or if they were pregnant. Passive anterior laxity was quantified by arthrometry using a KT 1000 (Medmetric, San Diego, California). ACL injury was confirmed at the time of surgery. This study was approved by the Human Research Ethics Committees of the University of Sydney, and the Australian Capital Territory Department of Health and Community Care.

A Lunar DEXA scanner (General Electric, Madison, USA) was used to measure BMD at the knee, using software designed for the forearm. The subjects were positioned in supine with the knees in extension, and neutral rotation (measured by alignment of the infrared beam of the scanner with the anterior tibial spine). Knee position was maintained by the

use of rice bags, rather than thermoplastic splinting (Murphy et al. 2001) as the splinting generated an artefact in the raw data collection. Each scan took nine minutes to complete.

Reliability of the BMD measurement was tested by repeating BMD measurements on 2 separate days, in six healthy control subjects. The coefficient of variation in BMD measurements was $18 \pm 30\%$ (range 3% to 78%). Reasons for this wide variation in measurement was examined by testing in one subject (the author JMS) against a range of factors considered to have influenced the ability of the DEXA scanner to sample and compare the tissues (muscle, bone and air). These factors included comparing thermoplastic splinting to rice bags for positioning, using rice bags against the knee as a tissue substitute and using windows of various widths and lengths. The Lunar DEXA machine was tested with a phantom and had a coefficient of variation of $< 2\%$. It was tested again with forearm BMD measurements using the forearm software, and had a coefficient of variation of $< 2\%$. Lunar GE representatives in Australia and in the USA determined that the forearm software required samples of all tissues, ie. bone, muscle and air in each scan for comparison of density, in order to determine BMD. If the sample of muscle tissue, for example, was insufficient, the BMD would be inaccurate. Personal communication with other research teams (Murphy et al.2001, and Li and Nilsson 2001) had also described problems with reliability of the technique, and these teams had since had ceased to Lunar GE DEXA scanners for BMD of the knee.

BMD was calculated at six regions of interest: centrally below the intercondylar eminence of the tibia, at the femur and tibia adjacent to the articular surface of the medial and lateral condyles, and at the lateral epicondyle of the femur (Figure 9.1). The regions measured below the intercondylar eminence of the tibia, and at the lateral epicondyle of the femur represent the sites of fixation of the ACL graft in reconstruction surgery. The intercondylar

eminence of the tibia is the site for the tibial tunnel and tibial interference screw. The lateral epicondyle is the site for femoral fixation using a femoral suspensory pin. To control for the confounding variables of age, sex, weight and medical history the injured knee was compared to the healthy contralateral knee.

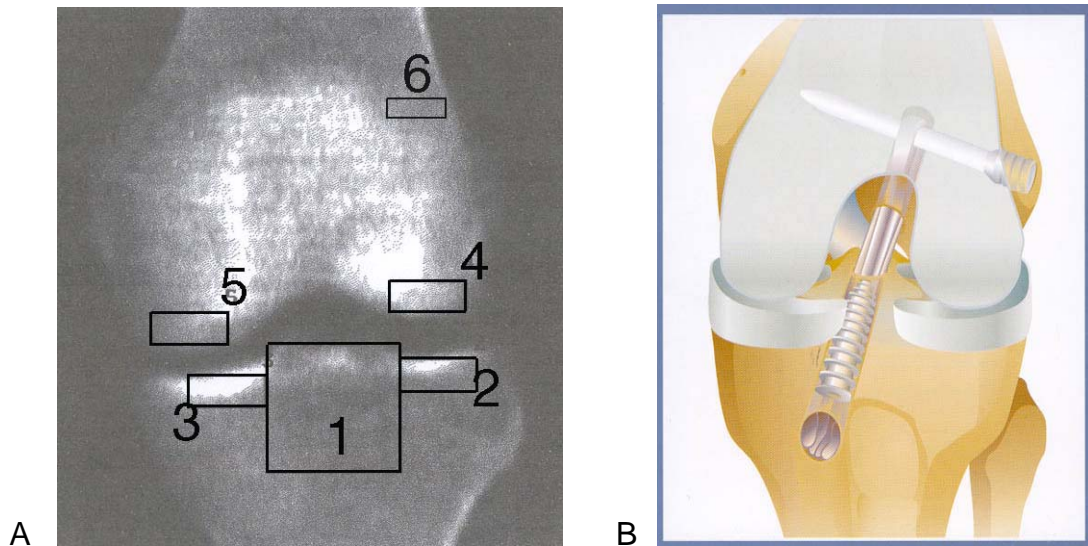


Figure 9.1: A: Image retrieved from a Lunar DEXA scanner used to measure bone mineral density at six regions of interest: 1. Central tibia, 2. Lateral tibial condyle, 3. Medial tibial condyle, 4. Lateral femoral condyle, 5. Medial femoral condyle, 6. Lateral femoral epicondyle.

B: Diagram of the knee showing the fixation system used at The Canberra Hospital for hamstrings autograph reconstruction of the ACL. The tibial interference screw is located at BMD measurement region 1 and the femoral suspensory pin is located at BMD region 6.

Statistical Analysis

A student's t-test was used to compare the BMD of the injured knee to the healthy knee for each of the six regions of interest. A Pearson's r correlation coefficient was used to examine the relationship of BMD and the variables of age, sex and time since injury, and side of injury.

Results

Significant BMD loss was demonstrated at all six regions of interest (Table 9.1). At the central tibia, below the intercondylar eminence, a mean loss of 17% was recorded in the BMD of the injured knees. The mean BMD for the healthy knee was $0.61 \pm 0.29\text{g/cm}^2$ (mean \pm SD) and for the injured knee was $0.51 \pm 0.27\text{g/cm}^2$. This demonstrates a significant bone loss ($p= 0.001$) at the site of tibial fixation of the tissue graft.

At the regions close to the articular surface on the tibia a mean bone loss was found of 16% ($p = 0.001$) at the lateral tibial condyle and 12% ($p = 0.007$) at the medial tibial condyle. At the regions close to the femoral articular surface similar BMD loss was seen. At the lateral femoral condyle there was a mean loss of 17% ($p = 0.005$), and loss of 21% ($p = 0.002$) at the medial femoral condyle. That is, the medial and lateral compartments of the knee both demonstrated a significant loss of BMD in the condylar bone.

At the femur a wide range of the bone density values was recorded, from extremely low bone density values, (for the injured knee 0.13 g/cm^2 on the lateral femoral condyle and 0.18 g/cm^2 at the medial femoral condyle) to high BMD values (1.43 g/cm^2 to 1.23 g/cm^2 respectively). Similarly, side-to-side differences varied widely at the femoral regions of interest, from a gain in BMD of 38% to a loss of 64% at the medial femoral condyle.

The lateral epicondyle of the femur demonstrated a mean loss of BMD of 24% in the injured knee, from $0.53 \pm 0.31 \text{ g/cm}^2$ in the healthy knee to $0.39 \pm 0.26 \text{ g/cm}^2$ in the injured knee. This region recorded the lowest individual bone density scores for the knee.

Table 9.1: Bone mineral density (g/cm^2 ; mean \pm SD) changes in the ACL-injured knee.

Region measured	Healthy knee BMD (g/cm^2)	ACL-injured knee BMD (g/cm^2)	% Loss BMD	Significance level
Central tibia	0.61 ± 0.29	0.51 ± 0.27	Mean 17% Range: +9 to -57%	$\rho = 0.001$
Lateral tibial condyle	0.91 ± 0.30	0.76 ± 0.28	Mean 16% Range: +5 to -57%	$\rho = 0.001$
Medial tibial condyle	1.18 ± 0.31	1.01 ± 0.25	Mean 12% Range: +17 to -45%	$\rho = 0.007$
Lateral femoral condyle	0.91 ± 0.38	0.75 ± 0.36	Mean 17% Range: +36 to -56%	$\rho = 0.005$
Medial femoral condyle	0.83 ± 0.31	0.65 ± 0.31	Mean 21% Range: +38 to -64%	$\rho = 0.002$
Lateral epicondyle femur	0.53 ± 0.31	0.39 ± 0.26	Mean 24% Range: +28 to -78%	$\rho = 0.001$

Bone mineral density changes in the ACL-injured knee

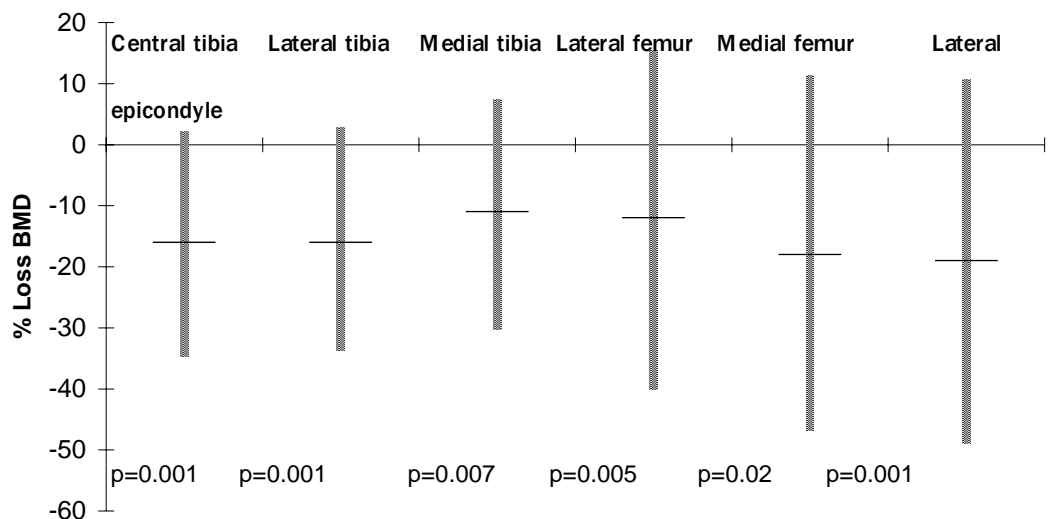


Figure 9.2: Difference in bone mineral density data between the ACL-injured and contralateral knee at the six regions of interest (mean \pm SD).

Case study: Of particular interest was a 27 year old carpenter with a recorded BMD of 0.05 g/cm^2 at the healthy lateral femoral condyle and 0.04 g/cm^2 at the injured lateral femoral condyle, and 0.20 g/cm^2 and 0.16 g/cm^2 at the healthy and injured central tibia. Because of these low recorded results, this subject was recalled for repeat scanning, which resulted in a variation of less than 2% from the original calculation. His lumbar spine and hip BMD were then tested. The lumbar spine was 0.7 standard deviations below the age-matched population, and the proximal femur was 0.2 standard deviations below the age-matched mean. This example illustrates the normal variation in BMD values within individuals.

BMD data were analysed for correlations with age, sex, right or left knee or time since injury. Time since injury was moderately correlated with BMD of the central tibia (Pearson's $r = 0.51$, $p = 0.022$; Figure 9.3). BMD loss appeared to persist in the injured knee for approximately two years before beginning to reverse (Figure 9.3). To more closely examine the effects of time since injury on BMD loss, and remove the influence of the chronic ACL-injured subjects, the data were extracted for those 16 subjects who injured the knee within the last 12 months. There was a moderately strong correlation at the central tibia (Pearson's $r = 0.60$, $p = 0.014$) and a trend at the medial tibial and femoral condyles (tibial condyle $r = 0.44$, $p = 0.09$; femoral condyle $r = 0.42$, $p = 0.011$). Thus, it appeared that the BMD of the tibia of the injured knee decreased with time since injury. The BMD at the medial tibial and femoral condyles followed the same pattern. The BMD of the lateral condyles did not appear to be related to time since injury. There were no correlations with age, sex, or side of injury.

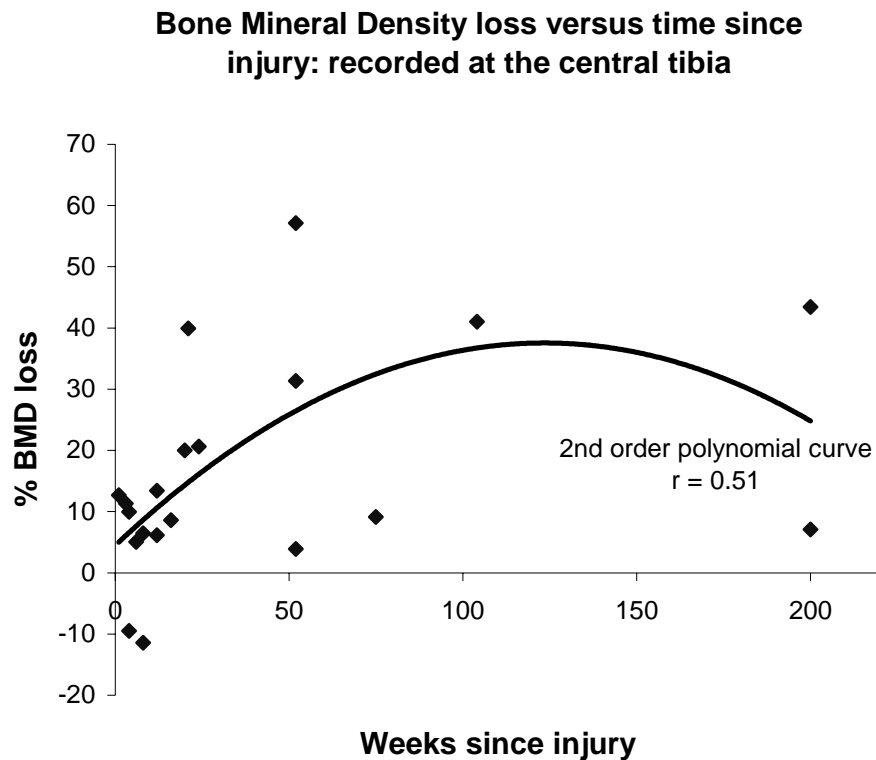


Figure 9.3: Analysis of the correlation between BMD at the central tibia (Region 1) and time since injury. BMD loss appeared rapidly after injury, and may persist for 2 years before recovering.

Discussion

Significant BMD loss in the ACL-injured knee at the graft fixation sites indicates that bone quality required for secure fixation may be compromised. At the centre of the tibia, 11 of the 20 subjects recorded BMD of less than 0.6 g/cm^2 in the injured knee, the level described by Brand et al (2000) as the critical BMD for reliable fixation. At the lateral femoral condyle BMD measurements in 13 / 20 subjects were below this critical value in the injured knee. Testing of the strength of fixation devices needs to be re-evaluated using

tissue specimens with BMD values more similar to those of the ACL-injured population, and should include the worst-case scenario. Meanwhile, it is important to recognise that currently established values for strength of fixation at the knee are based on young healthy bone stock and are therefore not necessarily applicable to the ACL-injured population. The values that were recorded from elderly cadaver studies (Steiner et al. 1994; Caborn et al. 1998; Brand et al. 2000) may be more relevant than previously assumed.

Our finding of decreased BMD in ACL-injured knees is consistent with previous studies. Sievannen et al (1994) reported a single case study of a 26-year-old student who injured the ACL while participating in a trial of strength training effects on BMD. This accidental event enabled longitudinal recording of preinjury and postoperative BMD, though not post injury BMD. BMD at the femur and tibia reached their lowest point at 15 weeks post surgery (18.5% at the distal femur, and 17.4% at the proximal tibia). Twelve months later muscle performance was completely recovered but BMD values were still below the subject's baseline (9.6% at the distal femur and 13.9% at the proximal tibia). This accidental finding was followed by a prospective study of 21 patients. BMD fell 21% at the femur and 14% at the proximal tibia 12 months after bone-patella tendon-bone reconstruction for ACL injury (Leppala et al. 1999). However, this study did not report the preoperative BMD of patients with a complete ACL tear. Though these studies both record postoperative rather than post injury BMD loss, and postoperative BMD loss could be expected to be greater due to the additional inflammation in the knee due to surgery, the results are of similar magnitude to those reported here.

Time from injury correlated well with BMD loss at the central tibia, but it was not possible to predict the optimal time for knee reconstruction surgery based on the BMD changes. It

is apparent however, that BMD falls rapidly in the first weeks post injury and may remain low for 2 years. This study was a cross sectional cohort study, and as such, conclusions about BMD changes over time are speculative. It would be necessary to perform a longitudinal study of ACL-injured subjects in order to confirm these findings.

It would be useful to isolate the risk factors for BMD loss. Age, sex, or side of injury were not identified in this study as risk factors for post injury osteopaenia at the knee. It may be possible that bone turnover is slower in older people, or slower in older women. There is some evidence that it is more difficult to increase bone density in older women (Frost, 2000). In our study the sample may have been too small, or too homogeneous to demonstrate the effects of age and sex on bone density loss.

In this study of ACL-injured knees a wide range of values for side-to-side difference in BMD was recorded from 86% increase to 79% decrease with an overall mean loss of 15%. Likewise, in the test-retest reliability study the coefficient of variation for the measurement varied widely from 78% to 3%. The wide range of BDM values may reflect heterogeneity of the subject group in terms of confounding joint trauma, bone bruising, and mechanism of injury. It is also possible, however, that the wide range in BMD values resulted from the unsolved reliability issues with this method of BMD measurement at the knee. The problems of reliability originate from the software used to compare the X-ray absorption of sampled tissues. However, the problems with recorded BMD were random, so while the standard deviation was increased, the population means were maintained. The significant level of BMD loss that occurred in the ACL-injured knees was clear despite the large standard deviation of the population sampled.

The role of BMD in graft fixation failure requires further detailed examination *in vivo*. *In vitro* studies have demonstrated failure of fixation, and graft slippage around the fixation as the common reason for failure, rather than midsubstance rupture of the graft (Pena et al. 1996; Weiler et al. 1998; Brand et al. 2000; Weiler et al. 2000). No clinical studies have separated fixation failure from graft tissue failure as a reason for failure of reconstruction surgery.

Accelerated rehabilitation programs demand adequate fixation strength in the postoperative period. A significant frequency of graft failure in the early postoperative period has been reported (Salmon et al. 2002), and this may in fact be due to fixation failure in this critical period. Planning for rehabilitation and progression of weight bearing, both post injury and postoperatively must take the BMD loss into account. Perhaps early weight bearing and strength training will be able to minimise the BMD loss at the critical postoperative period before graft incorporation.

Conclusion

BMD was found to be significantly lower in the ACL-injured knees than the healthy contralateral knees, and potentially posed a threat to secure fixation. BMD was reduced in subjects as early as three weeks following injury, and was also reduced in subjects twelve months following injury. Despite problems with the reliability of Lunar (GE) DEXA scanning and software at the knee, it is apparent the loss of BMD in ACL-injured knees is rapid and profound.

Compromise in fixation is an important consideration in accelerated rehabilitation programs following ACL reconstruction. BMD at fixation sites is critical to choice and integrity of fixation, and planning of rehabilitation schedules. In order to make an informed decision about surgical management, an assessment of BMD is recommended as routine workup for reconstruction surgery. It is a simple, inexpensive, and low risk procedure, and would enable choice of fixation appropriate to the patients' bone quality. Development of a reliable system for measurement of BMD at the knee is required. Current systems developed by Lunar GE for measurement of BMD at the lumbar spine and forearm have limited application for use at the knee.

CHAPTER 10

KINEMATICS OF OSTEOARTHRITIC KNEES

Abstract

Osteoarthritis of the knee is a widespread problem, yet little is known about the kinematics of osteoarthritic knees or the tibiofemoral (TF) contact pattern.

Fourteen subjects with symptomatic osteoarthritis in one knee and no pain or injury in the contralateral knee performed a supine leg-press from 0° to 90° flexion against a 150N load. The tibiofemoral contact pattern was recorded for both knees using MRI. Bone mineral density (BMD) was recorded using dual energy x-ray absorptiometry close to the subchondral bone. Pain and disability were recorded using a WOMAC questionnaire.

Severity of osteoarthritis in the knees ranged from grade 2 to 4 (8 subjects had grade 4) in the symptomatic knee and from 0 to 3 (8 subjects had grade 0 or 1) in the contralateral knee. TF contact in the lateral compartment of symptomatic knees was more anterior on the tibial plateau than healthy knees ($p < 0.01$) and this was associated with severity of osteoarthritis ($p < 0.01$). TF contact in the medial compartment was also more anterior on the tibial plateau and this was more pronounced at grade 3 than grade 4 osteoarthritis ($p = 0.014$). Abnormality in lateral compartment tibiofemoral contact patterns was correlated with disability (WOMAC score; $r = 0.54$, $p = 0.047$). There was no difference in BMD between the osteoarthritic and contralateral knees, but the BMD was correlated with pain ($r = 0.63$, $p < 0.01$) and physical function ($r = 0.63$, $p = 0.03$) of the WOMAC score, ie. as function decreased, bone density increased.

Loss of tibiofemoral roll back and longitudinal rotation in osteoarthritic knees is associated with loss of range of motion and patterns of wear in the knee.

Introduction

Osteoarthritis of the knee is a widespread problem, yet very little is known about the kinematics of the osteoarthritic knee. In Australia, about 12% of the population and 34% of people over 50 years of age, suffer from osteoarthritis (Chapman and Feller 2003). The knee is commonly affected (Segal et al. 2002). The incidence of total knee arthroplasty (TKA) in Australia has risen from 56.4 per 100 000 in 1994 to 76.8 per 100 000 population in 1998 (Wells et al. 2002). Internationally the demand for TKA is also increasing, with the Swedish Arthroplasty Register reporting a five-fold increase in TKA performed annually since 1976 (Robertsson et al. 2000). The social and economic impact of knee osteoarthritis to the community is expected to increase as the general population age rises, both in terms of health costs and lost quality of life (Hurwitz et al. 2001). However, research into the kinematics of osteoarthritic knees is very limited.

Kinematic studies of the knee have shown that some characteristics of normal knee motion are lost in osteoarthritic knees. Osteoarthritic knees lose some of the longitudinal rotation that normally occurs with terminal extension (Nagao et al. 1998). In healthy knees the longitudinal rotation of the knee during knee flexion has been described in vitro (Shaw and Murray 1974; Blankevoort et al. 1988; Hollister et al. 1993; Pinskerova et al. 2000) and in vivo (Todo et al. 1999; Hill et al. 2000b; Iwaki et al. 2000). Koga (1998) found variation from normal of the longitudinal rotation axis to be associated with the severity of osteoarthritis in the knee. The loss of normal longitudinal rotation during flexion in osteoarthritic knees could be either due to contracture and thickening of synovial and capsular tissues that limits joint mobility or due to attrition of the anterior cruciate ligament

(ACL) (Allain et al. 2001) and this has a role in controlling longitudinal rotation (Lane et al. 1994).

Gait analysis studies have demonstrated an increased adduction moment in osteoarthritic compared to healthy knees (Sharma et al. 1998; Andriacchi et al. 2000). Varus thrust was initially described during stance phase of gait as weight was transferred onto the medial compartment of the knee. In the knee with medial compartment osteoarthritis, the medial joint space width was reduced, causing varus angulation, with a resultant adduction moment as weight was applied (Maquet 1976). This concept has been further developed with the calculation of directional forces recorded by a force platform during gait analysis (Andriacchi et al. 2000). The adduction moment of osteoarthritic knees not only characterises the unusual kinematics of osteoarthritic knees (Andriacchi et al. 2000), but also is associated with severity of disease (Sharma et al. 1998; Sharma et al. 2001), is correlated with subchondral bony sclerosis (Hurwitz et al. 1998; Sharma et al. 1998; Hurwitz et al. 1999; Hurwitz et al. 2000; Hurwitz et al. 2001; Baliunas et al. 2002) and is an indicator of disease progression (Sharma et al. 1998; Baliunas et al. 2002).

The relationship between the knee kinematics described by tibiofemoral contact and the process of osteoarthritis has not been examined. There is no published data on the tibiofemoral contact pattern in osteoarthritic knees. The contact area of the knee in extension has been described in a cadaver study, using silicon extrusion to demonstrate the area of contact (Fukubayashi and Kurosawa 1980). The contact area of the knee in extension was wider than the normal knee and the menisci made little contribution to the load sharing of the knee. This is consistent with the flattened shape of the condyles (Fairbank 1948; Kawahara et al. 2001) and pattern of degeneration of the menisci that

characterise the osteoarthritic knee (Kawahara et al. 2001). However, the study by Fukubayashi and Kurosawa (1980) was limited to knee extension in vitro and does not inform us about the contact pattern of osteoarthritic knees during active motion.

Magnetic Resonance Imaging (MRI) has been used to examine active motion of healthy knees, but not the motion of osteoarthritic knees (Todo et al. 1999; Hill et al. 2000a; Hill et al. 2000b; Nakagawa et al. 2000). In healthy knees, recording the motion of the flexion facet centre of the femoral condyle has demonstrated that longitudinal rotation of the knee occurs during knee flexion from 0° to 120° through an axis occurring through the medial compartment of the knee (Todo et al. 1999; Hill et al. 2000a). Thus MRI has been able to consistently and accurately describe kinematics of healthy knees, but this technique has not yet been used to describe the tibiofemoral contact pattern of osteoarthritic knees.

Osteoarthritis is assessable by MRI because changes to bone, soft tissue and cartilage structures are all visible (Zanetti et al. 2000; McCauley and Disler 2001) and areas of damage to the articular cartilage can be recorded reliably (Lundberg et al. 1997; Potter et al. 1998; Duchateau and Vande Berg 2002). Bone features that are visible on MRI include osteophytes, subchondral sclerosis, subchondral haematoma and cysts (Zanetti et al. 2000). MRI can be used to also assess ligament integrity. As many as 50% of osteoarthritic knees may have a complete ACL tear, with an additional 25% having a partial ACL tear (Wada et al. 1996; Allain et al. 2001). In the present study, MRI was used to assess the articular cartilage, ligament and meniscal damage present in the osteoarthritic knees.

Increase in bone mineral density (BMD) at the distal femur and proximal tibia has been used as a very early indicator of osteoarthritis (Buckland-Wright et al. 2000; Messner et al.

2000) and has been associated with disease progression (Madsen et al. 1994; Wada et al. 2001). The medial compartment has higher BMD than the lateral compartment in healthy knees, and in osteoarthritic knees the ratio is higher due to the sclerosis of the subchondral bone (Madsen et al. 1994; Hurwitz et al. 1998). Dual energy x-ray absorptiometry (DEXA) provides a reliable and accessible means to measure BMD at the knee (Petersen 2000), with low radiation exposure to subjects (Ott 1998). It has been assessed for accuracy and precision at the distal femur and proximal tibia (Sievanen et al. 1992; Petersen 2000; Murphy et al. 2001; Nilsson 2001). DEXA has been shown to be sensitive to changes in BMD at the knee due to high tibial osteotomy (Akamatsu et al. 1997), knee injury and reconstruction (Sievanen and Kannus 1994; Leppala et al. 1999). DEXA has also been used to assess progression and severity of knee osteoarthritis (Hurwitz et al. 1998; Andriacchi et al. 2000; Hurwitz et al. 2001; Wada et al. 2001; Hulet et al. 2002).

The aim of this study was to map, for the first time, the tibiofemoral contact pattern in osteoarthritic knees between 0° and 90° knee flexion and describe the differences between the contralateral asymptomatic knees and healthy knees. The associations between differences in the tibiofemoral contact pattern and severity of osteoarthritis were also examined.

Method

Subjects

Fourteen subjects with symptomatic osteoarthritis in one knee and no symptoms or history of osteoarthritis in the contralateral knee (Table 10.1), were recruited to participate and provided voluntary informed consent. Three subjects were male and eleven were female,

which reflected the population distribution of gender in osteoarthritis (Segal et al. 2002). Subjects were aged 54 to 81 years (65 ± 9.1 years; mean \pm SD). Ten subjects had primary idiopathic arthritis and four had osteoarthritis secondary to injury. Three subjects had sustained anterior cruciate ligament injuries 16, 21 and 23 years ago, including one subject who had an extraarticular knee reconstruction 12 years ago. One subject had osteoarthritis secondary to patella fracture through the articular surface. Eight had osteoarthritis in the right knee and six in the left. Subjects were excluded if they had any symptoms in the contralateral knee, including pain, stiffness or swelling indicative of osteoarthritis or any injury to the ligaments, menisci or bones of the contralateral knee. Subjects were excluded if they had contraindications to MRI. The principle contraindications to MRI that were encountered were ferrous metal implants (including pacemakers) and claustrophobia. Pregnancy was also a contraindication to MRI and DEXA in this study, but the female subjects recruited were postmenopausal.

Twelve healthy subjects were used as controls for the comparison of knee kinematics. These subjects are described in Chapter 4. These healthy subjects were aged 20 to 50 years, 7 were male and 5 were female. None had any symptoms or history of injury in either knee.

The study was approved by the University of Sydney and Australian Capital Territory Health Department Human Research Ethics Committees.

Table 10.1: Details of the osteoarthritic subjects. Details of joint damage were recorded from MRI and operation reports.

	Age	Sex	Side	OA Type	Kellgren Lawrence Grade		Ligament Status				Compartment most affected	Meniscal damage	
					Symptomatic	Asymptomatic	ACL	PCL	MCL	LCL		Medial	Lateral
1	63	F	R	2	4	3	absent	✓	✓	✓	both	macerated	tear
2	75	F	R	1	4	3	✓	✓	✓	✓	both	macerated	✓
3	74	F	R	1	4	1	absent	✓	✓	✓	medial	macerated	macerated
4	54	F	L	2	3	0	absent	✓	old tear	✓	both	d.tear	d.tear
5	65	F	L	1	4	1	✓	✓	✓	✓	medial	macerated	✓
6	77	M	R	1	4	0	✓	✓	✓	✓	both	macerated	macerated
7	54	F	R	1	4	2	✓	✓	✓	✓	medial	PAM.	✓
8	58	F	R	1	3	0	✓	✓	✓	tear	medial	✓	tear
9	61	F	L	1	2	2	✓	✓	✓	✓	lateral P/F	✓	✓
10	58	F	L	1	4	2	absent	✓	✓	✓	both	✓	macerated
11	71	M	R	2	3	0	absent	✓	✓	✓	both	macerated	macerated
12	70	F	L	1	4	2	v frayed	✓	✓	✓	medial	macerated	✓
13	80	F	L	1	3	1	✓	✓	✓	✓	medial	d. tear	✓
14	54	M	R	2	3	0	absent	d.tear	d.tear	✓	both	✓	macerated

“✓” indicates normal appearance

“d.tear” indicates degenerative tear, seen at surgery or abnormal signal intensity indicative of a tear

“macerated” indicates severe late stage degeneration with extensive tissue damage

“PAM” previous arthroscopic meniscectomy

Kinematic MRI

Kinematic MRI scans were performed using the technique described in Chapter 4 (Scarvell 2003c). Subjects performed a leg-press in supine. Images were taken of both knees at seven 15° intervals from full knee extension to 90° flexion. Subjects were scanned at each knee flexion angle twice, once while relaxed (unloaded) and again pressing down against a footplate weighted with a 150N load (loaded). Sixteen slices were generated (eight through each knee) approx 10 mm apart (TR = 160.0, TE = 2.3/1, TA = 00:46), with a 256 x 256 matrix using a spoiled gradient echo sequence. The digital images were analysed using Osiris[®] software (Université de Genève, Switzerland).



Figure 10.1: Sagittal MRI scan of the medial compartment of an osteoarthritic knee at 90° knee flexion. The medial compartment contact point was recorded as the distance from the posterior tibial cortex to the tibiofemoral contact.

The tibiofemoral contact point was measured as described in Chapter 4. The distance was measured from the posterior tibial cortex to the point at which the femoral condyle contacted the tibial plateau (Figure 10.1).

Range of motion, measured by a goniometer in sitting, was restricted in the symptomatic osteoarthritic knee of all subjects, with none recording over 135° knee flexion. Knee flexion was $107 \pm 9^\circ$ (mean \pm SD; range 83° to 135°). Extension was $2 \pm 3.8^\circ$ (range -5° to 10° flexion). Six subjects were unable to extend the knee to 0°. During the procedure for kinematic MRI, the measure recorded as the 0° position is the limit of knee extension for each subject.

Details of osteoarthritis and damage in the knee joint

The symptomatic and contralateral knees of subjects were graded for severity of osteoarthritis according to the criteria described by Kellgren and Lawrence (1957) (Table 10.2).

Table 10.2: Definitions of the Kellgren Lawrence grades for knee osteoarthritis.

Grade	Summary	Definition
Grade 0	None	Normal appearance
Grade 1	Doubtful	Possible osteophytic lipping
Grade 2	Minimal	Definite osteophytes and possible joint space narrowing
Grade 3	Moderate	Moderate or multiple osteophytes, definite joint space narrowing and some sclerosis and possible bony attrition
Grade 4	Severe	Large osteophytes, marked joint space narrowing, severe sclerosis and definite bony attrition.

Diagnostic MRI scans of the symptomatic osteoarthritic knee were used to record details of articular cartilage damage, meniscal damage and ligament integrity in the knee joint. Six sequences for examination of the knee joint were used: proton density sequences were used to image cartilage defects, STIR sequences to image articular cartilage, T2 sagittal, coronal and axial sequences to assess bony structures and soft tissue structures around the knee and T1 sequences to examine soft tissue and ligamentous structures, because fat appears as high signal intensity. These six sequences were used to generate slices 4mm apart. A radiologist experienced in MRI techniques and image interpretation for musculoskeletal conditions reported the images.

For subjects with total knee replacement, details of ligament integrity, articular cartilage damage and meniscal damage were obtained from operating theatre records. Details of joint damage are reported in Table 10.1.

A self-administered functional disability score was used to record the limitations to activities of daily living and experience of pain. The Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC) was used. The WOMAC records self-reported pain, stiffness, physical function, social function and emotional function on a five-point Likert scale (Bellamy et al. 1988; Bellamy 1989). The scores for pain, stiffness and physical function have been well validated for construct reliability in subjects with knee osteoarthritis (Bellamy 1989). The scores for pain were found to be the most responsive to change and also the most important to subjects (Bellamy 2003). Prior to generating a total WOMAC score, it is recommended that the scores for pain, stiffness and physical function be normalised as a percentage, then weighted according to their relative importance to the subject, prior to totalling. The relative importance assigned to the scores was pain 0.42,

stiffness 0.21 and physical function 0.37 (Bellamy 2003). Emotional and social scores are not recommended to be included in the total WOMAC score.

Cincinnati knee score (Bellamy et al. 1988; Bellamy 1989; Barber-Westin et al. 1999) was also completed for each subject. The Cincinnati knee score was designed to measure severity of knee injuries and postoperative outcomes and therefore tends to have floor effects in groups of more severely disabled and elderly individuals. However, in this thesis the Cincinnati knee score has been used throughout to record the symptoms, examination findings and radiological findings in acute and chronic ACL-injured subject groups and gives an indication of the severity of dysfunction in osteoarthritic subjects also.

Subchondral bone density measurement

BMD of the subchondral bone regions of the tibia and femur were measured using dual energy x-ray absorptiometry, as described in Chapter 9. A Lunar DEXA scanner (General Electric, Madison, USA) was used throughout, using software designed for the forearm, because the forearm software also allowed for inclusion of regions of air in the scan field. The size of the soft tissue sample was small around the knee, so the inclusion of rice bags beside the knee provided a soft tissue sample substitute. The subjects were positioned in supine with the knees in extension and neutral rotation measured by alignment of the infrared beam of the scanner with the anterior tibial spine and the patella centred. Knee position was maintained by the use of rice bags at the ankle and beside the knee to control rotation. Each scan took 9 mins to complete.

BMD was calculated at five regions of interest; centrally below the intercondylar eminence of the tibia, adjacent to the articular surface of the medial and lateral tibial condyles and

adjacent to the articular surface of the medial and lateral femoral condyles (Figure 10.2). The region of interest at the intercondylar eminence of the tibia was chosen to provide a baseline measure, consistent with the regions used in Chapters 8 and 9. The regions of interest adjacent to the articular surface of the knee joint were chosen to measure changes to subchondral bone, for example subchondral sclerosis.

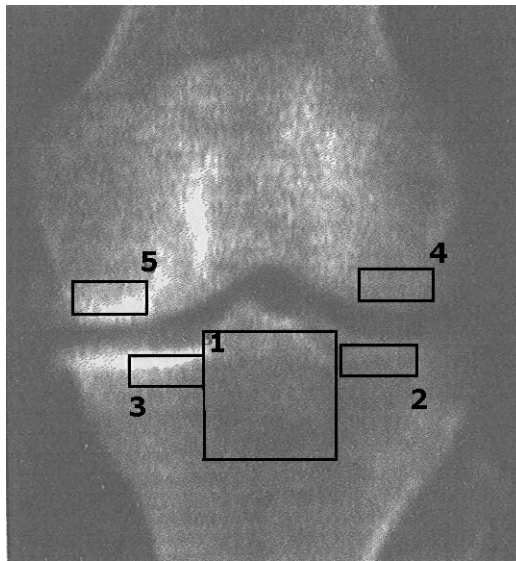


Figure 10.2: Bone mineral density was measured using dual energy x-ray absorptiometry at 1) the central tibia, 2) the lateral tibial plateau, 3) the medial tibial plateau, 4) the lateral femoral condyle and 5) the medial femoral condyle close to the subchondral bone of the knee.

Statistical Analysis

The tibiofemoral contact pattern of the symptomatic osteoarthritic knees was compared to the contralateral knee and to the knees of 12 healthy subjects (reported in Chapter 4), using repeated measures ANOVA. Differences between tibiofemoral contact patterns were compared for the medial and lateral compartments of the knee, for the loaded and unloaded conditions and for differences between osteoarthritic and contralateral knees of both osteoarthritic subjects and healthy subjects.

Associations between differences in the tibiofemoral contact pattern in the osteoarthritic knee and contralateral knee and Kellgren Lawrence grade, type of osteoarthritis and differences in BMD were tested for correlation using a Pearson's correlation co-efficient.

Results

Severity of disease in the osteoarthritic knees

For the osteoarthritic knees, no subject had grade 0 or grade 1 osteoarthritis on the Kellgren Lawrence scale. One subject had grade 2 osteoarthritis, 5 subjects had grade 3 osteoarthritis and 8 subjects had grade 4 osteoarthritis. MRI reports described severe tricompartmental osteoarthritis with full-thickness cartilage wear in 7 subjects. Six subjects had full thickness cartilage wear in the medial compartment only, with less severe osteoarthritis in the lateral and patellofemoral compartments. No subject had predominantly lateral compartment osteoarthritis. One subject had patellofemoral osteoarthritis, with minor changes only in the tibiofemoral compartments. In terms of meniscal damage, 10 subjects had medial meniscal damage, including one with a previous meniscectomy, 2 with degenerative tears and 7 with macerated menisci. Eight subjects had lateral meniscus damage, including 3 with tears and 5 with macerated menisci. Five subjects had damage to both menisci. Eight subjects had ligament damage to the knee including 7 with ACL deficiency defined at surgery. One ACL-deficient subject had concomitant PCL degeneration and degenerative MCL and LCL tears. Another ACL-deficient subject had a concomitant old MCL tear. One subject had an isolated LCL tear seen at surgery (Table 10.1).

Severity of disease in the asymptomatic knee

In 7 of the 14 subjects, the asymptomatic knees showed the appearance of osteoarthritis on x-ray. Five subjects had grade 0 (normal) and three had grade 1 (doubtful) radiographs on the Kellgren Lawrence scale. However, 4 subjects had grade 2 (minimal) and 3 subjects had grade 3 (moderate) osteoarthritis on x-ray. These 7 subjects with evidence of osteoarthritis on x-ray but no symptoms, included 6 with primary idiopathic osteoarthritis and one with osteoarthritis secondary to ACL injury in the symptomatic knee.

Self-report questionnaire results

Pain was reported on the WOMAC questionnaire as 9 ± 3.5 (range, 3 to 15; worst possible, 20). Subjects' reported experience of stiffness was 4 ± 2.2 (range, 0 to 6; worst possible, 8), including two subjects who experienced no stiffness. Physical function was reported as 33 ± 12.1 (range, 15 to 58; worst possible, 68). Social function was reported as 13 ± 6.9 (range, 0 to 24; worst possible, 28). Emotional function was reported as 12 ± 9.2 (range, 1 to 27; worst possible, 40). The total WOMAC score was $50 \pm 14\%$ (range, 28% to 75%). There was a very high correlation between components of the WOMAC scores, in particular the association between pain and other components of the WOMAC score (Table 10.3).

Table 10.3: Correlations (Pearson's *r*) among aspects of the WOMAC self-report questionnaire. Scores for pain, stiffness physical and social function were highly correlated. Emotional function was correlated to pain, but not the other components.

Variables	Pain	Stiffness	Physical function
Stiffness	0.78	-	
Physical function	0.69	0.70	-
Social function	0.82	0.76	0.83
Emotional function	0.61	0.40	0.34

Tibiofemoral contact patterns

The symptomatic and contralateral asymptomatic knees of osteoarthritic subjects did not have a significantly different tibiofemoral contact pattern ($F_{(1,104)} = 1.475$, $p = 0.227$) (Figure 10.5). However, the knees of osteoarthritic subjects had a different tibiofemoral contact pattern to that of healthy control subjects ($F_{(1,1884)} = 19.3$, $p < 0.001$). When post hoc analysis was employed to examine the difference, it was found that the symptomatic osteoarthritic knee had a different TF contact pattern to the healthy control subjects ($p < 0.001$) and the asymptomatic contralateral knee of the osteoarthritic subject was also different to the healthy control subjects ($p < 0.001$) (Figure 10.5).

In the medial compartment of the knee of healthy subjects the tibiofemoral contact pattern in knee extension began anteriorly on the tibial plateau (mean distance from the posterior tibial cortex 34 ± 4 mm (Figure 10.6). The femur then rolled back on the tibial plateau to 30° knee flexion (to be 21 ± 3 mm from the posterior tibial cortex). For the remainder of knee flexion to 90° , the healthy knee moved little, moving back to 19 ± 2 mm from the posterior tibial cortex at 90° knee flexion. In this respect the healthy medial condyle could be described as predominantly rolling between 0° and 30° and gliding between 45° and 90° . The medial compartment of the symptomatic osteoarthritic knees had similar contact in knee extension, but the femur did not move back on the tibial plateau as far, remaining 26 ± 5 mm from the posterior tibial cortex, ie. a difference of 5mm more anteriorly than the healthy knee contact position (Figures 10.3 and 10.4).

In the lateral compartment of the symptomatic osteoarthritic knees the tibiofemoral contact did not move posteriorly during flexion as far as healthy control subjects. In the healthy knees the femur moved from an anterior position on the tibial plateau in knee extension (27 ± 4 mm from the posterior tibial cortex reference point), to a posterior position at 90° knee flexion (11 ± 2 mm from the posterior tibial cortex), fairly steadily. The femoral contact pattern of the osteoarthritic knees did not move back as far on the tibial plateau: in knee extension the contact point was 29 ± 5 mm from the posterior tibial cortex and at 90° knee flexion it had only rolled back to 19 ± 4 mm from the posterior tibial cortex. Thus, there was less roll back in both the medial and lateral compartments of the symptomatic osteoarthritic knees (Figures 10.5 and 10.6).

The tibiofemoral contact pattern of the osteoarthritic knees when loaded was the same as the contact pattern when unloaded ($F_{(1,10\ 4)} = 0.095$, $p < 0.759$). There was no interaction between the symptomatic knee and asymptomatic knee of osteoarthritic subjects when the knee was loaded or unloaded ($F_{(1,10\ 4)} = 1.184$, $p < 0.909$).

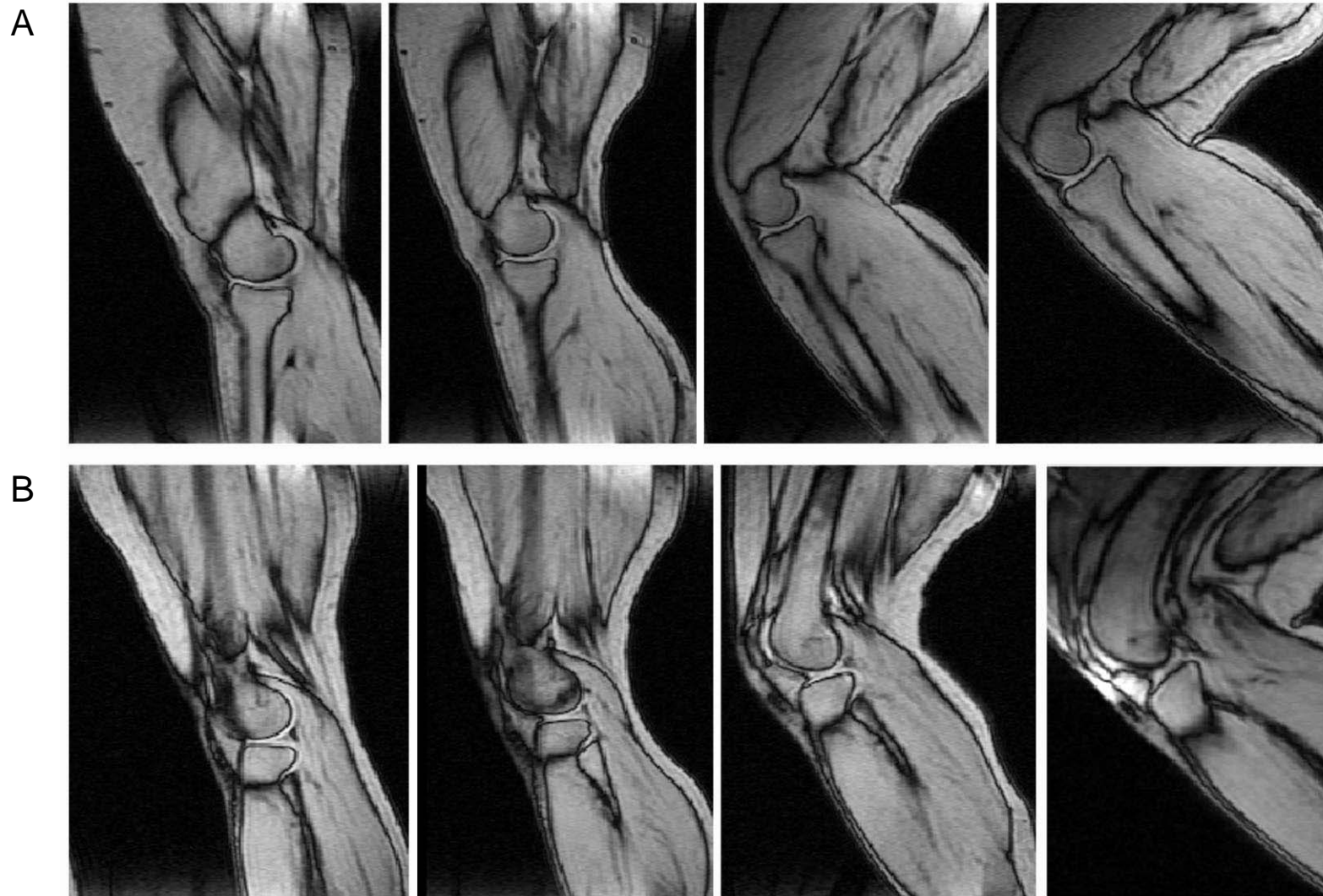
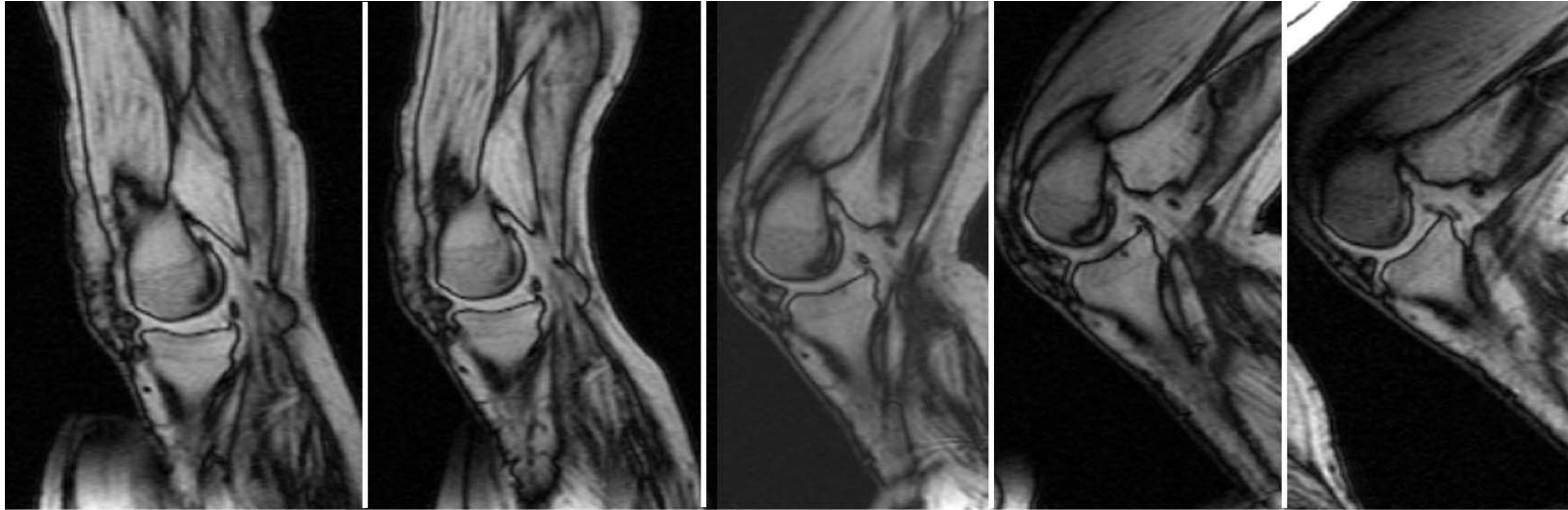


Figure 10.3: Gradient echo images of the A) medial and B) lateral compartments of the knee of a typical healthy subject, from 0° to 90° flexion. The medial femoral condyle was anterior on the tibial plateau in extension, then during flexion remained centred on the tibial plateau. The lateral femoral condyle continued to roll back through flexion.

A



B

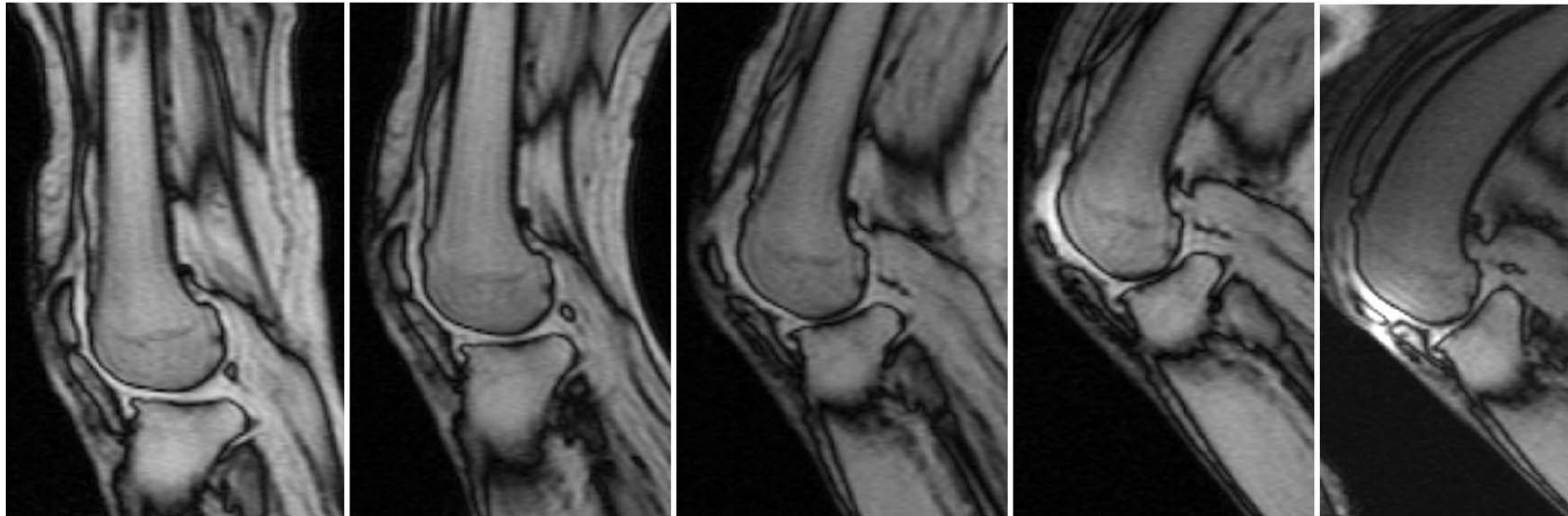


Figure 10.4: Gradient echo images of the A) medial and B) lateral compartments of the osteoarthritic knee of a typical subject, from 0° to 90°. The medial femoral condyle was fixed anteriorly on the tibial plateau. The lateral femoral condyle did not roll back through flexion, but remained centred on the tibial plateau.

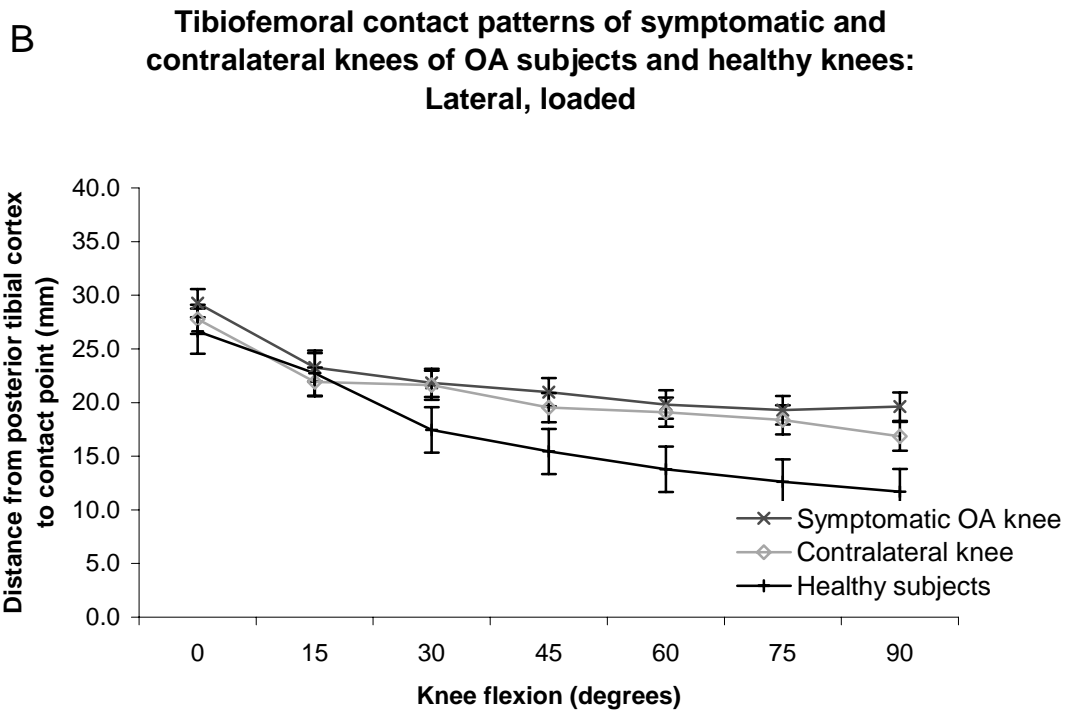
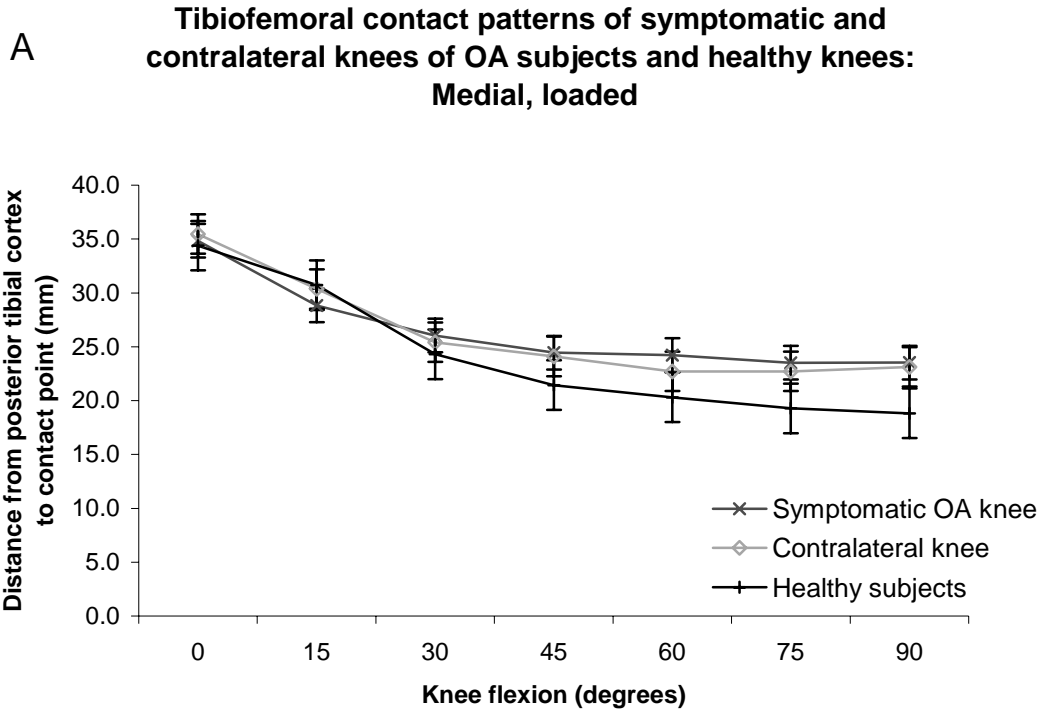


Figure 10.5: The tibiofemoral contact points for symptomatic knees and the contralateral knees of osteoarthritic subjects for the A) medial and B) lateral compartments during a loaded leg press.

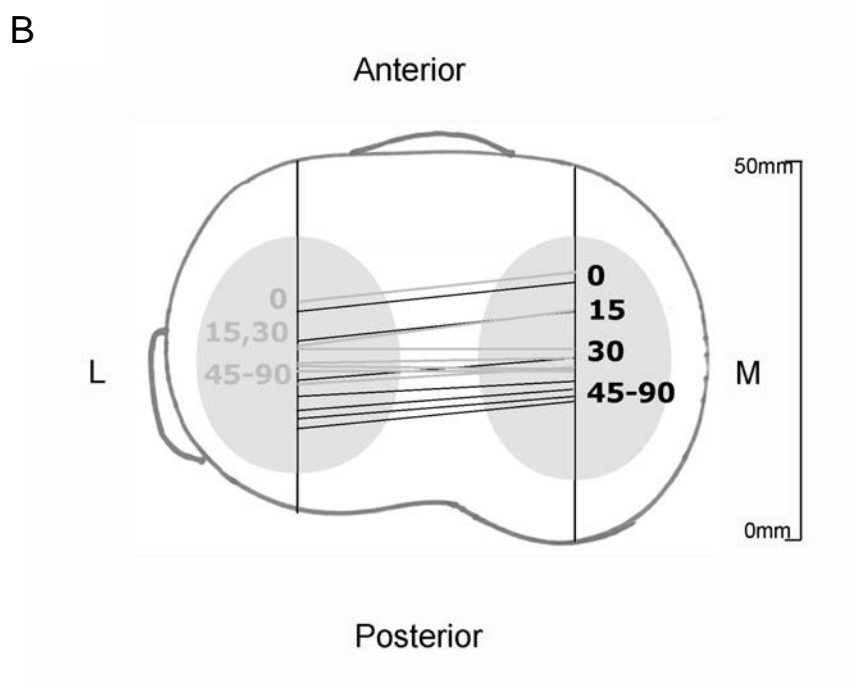
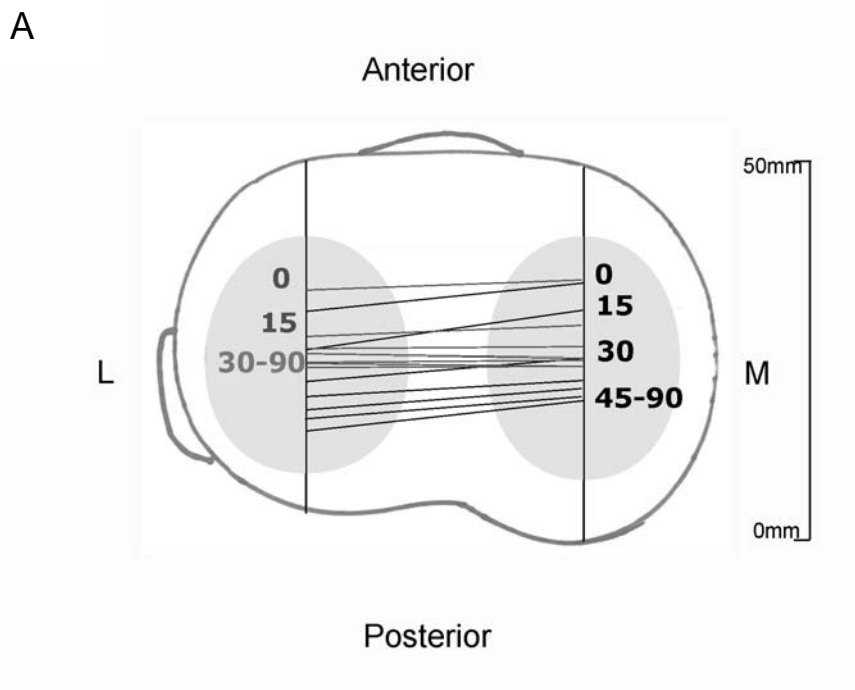


Figure 10.6: Diagram of the tibial plateau demonstrating the mean tibiofemoral contact points between 0° and 90° in the loaded knees, for A) symptomatic osteoarthritic knees (red), B) asymptomatic contralateral knees (yellow) and healthy control subjects (black).

Tibiofemoral contact was different for knees of different severity of osteoarthritis, as recorded by the Kellgren Lawrence grades ($F_{(1,4)} = 8.6$, $p < 0.001$). Post hoc analysis established that contact patterns were not different between knees with Kellgren Lawrence grades 0 and 1 (minimal and doubtful osteoarthritis), but grade 0 was different from grade 3 ($p < 0.001$) and from grade 4 ($p = 0.014$; Figure 10.7). Knees with Kellgren Lawrence grade 2 had a different tibiofemoral contact pattern from those with grade 3 ($p = 0.012$). Knees with Kellgren Lawrence grades 3 and 4 did not have a significantly different contact pattern ($p = 0.689$).

There was an interaction between the tibiofemoral contact pattern and severity of osteoarthritis and the medial or lateral compartments ($F_{(1,2)} = 3.2$, $p = 0.014$), indicating that disease severity affected the contact pattern in lateral compartment more than in the medial compartment (Figure 10.7).

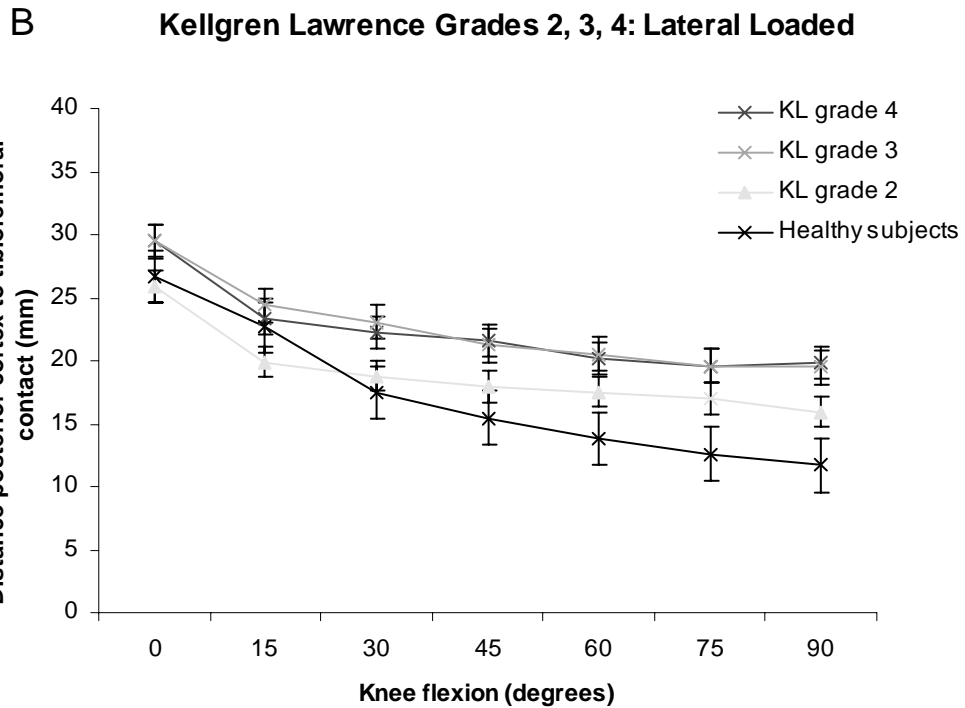
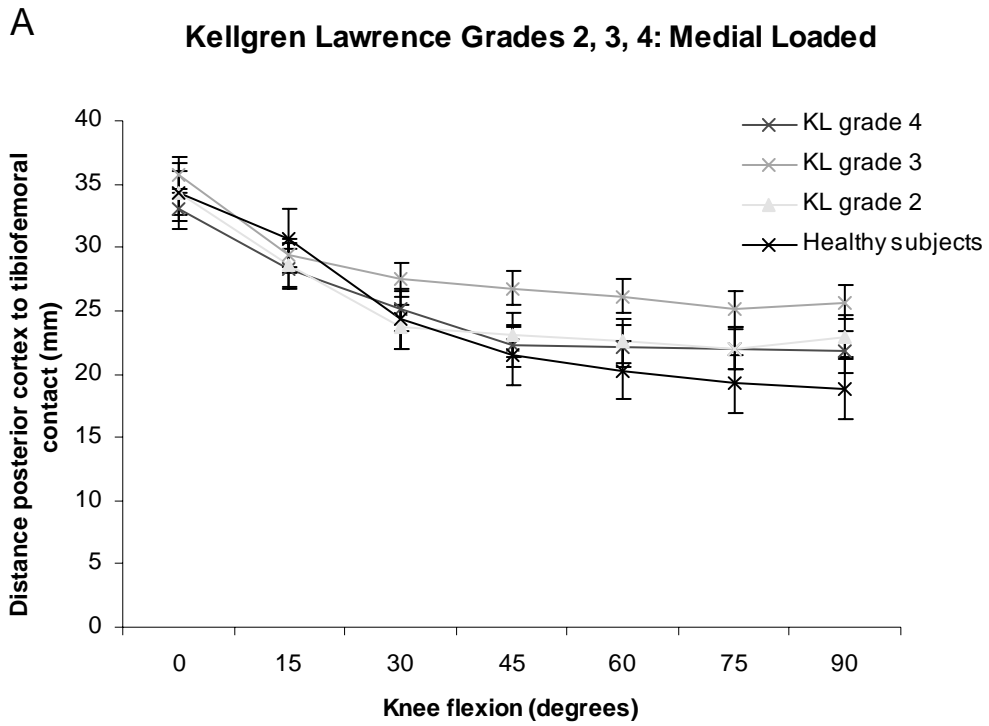


Figure 10.7: Tibiofemoral contact patterns according to the severity of osteoarthritis in the A) medial and B) lateral compartments of the knee. Kellgren Lawrence grades 2, 3 and 4 were different to healthy knee contact patterns.

ACL integrity and kinematics. The 7 subjects with ACL-deficient knees did not have a significantly different contact pattern to the ACL-intact knees ($F_{(1,1)} = 1.062$, $p = 0.304$). When the medial and lateral compartments were considered separately, there was no significant difference ($F_{(1,1)} = 2.89$, $p = 0.092$) in contact pattern between ACL-deficient and ACL-intact knees.

Associations between pain, function and knee kinematics. The WOMAC scores for pain were weakly correlated with severity of osteoarthritis in the symptomatic knee ($r = 0.45$, $p = 0.09$). Total WOMAC scores were moderately correlated with severity of osteoarthritis ($r = 0.55$, $p = 0.05$), indicating that more severe osteoarthritis was associated with more severe pain and poorer function.

WOMAC scores were associated with kinematic changes in the lateral compartment of the knees. To quantify the difference in the contact patterns between the knees of healthy subjects and osteoarthritic knees, the difference between the group mean contact point of healthy subjects and the contact point of the osteoarthritic knee was calculated at each knee flexion angle and then added to give the sum of the difference. In the lateral compartment of the knee in the loaded condition, the sum of the difference between the normal knee and the osteoarthritic knee contact pattern was not correlated with the (total) WOMAC score ($r = 0.41$, $p = 0.14$). In the unloaded condition, the WOMAC score was correlated with the difference in contact pattern ($r = 0.54$, $p = 0.047$). This indicated that as the lateral contact pattern became less like the healthy subjects, the functional level reported by osteoarthritic subjects worsened. The contact pattern of the lateral compartment was more affected by knee osteoarthritis than the medial compartment of the knees and it was in the lateral

compartment of the knees that the association between the WOMAC score and kinematics was evident.

Bone mineral density

There was no significant difference between the osteoarthritic and contralateral knees at the 5 regions of interest examined ($p = 0.891$) (Table 10.4). The mean BMD of each region in the osteoarthritic knees was slightly lower than the contralateral knees, however this was not significant (Figure 10.8). There was similarly no difference in the ratio of bone density in the medial compartment compared to the lateral compartment of the osteoarthritic and contralateral knees. The mean ratio of medial to lateral BMD at the tibia was 1 : 1.5 (± 0.5) at the tibia and 1 : 1.0 (± 0.5) at the femur in the osteoarthritic knees; and 1 : 1.36 (± 0.4) at the tibia and 1 : 0.96 (± 0.4) at the femur in the contralateral knee.

There was no correlation between BMD and severity of osteoarthritis as indicated by the Kellgren Lawrence scores, in either the symptomatic osteoarthritic knee or contralateral knee of osteoarthritic subjects. BMD was, however, associated with pain, stiffness and physical function derived from the WOMAC scores. BMD at regions 1,2,3 and 5 was correlated to pain ($r = 0.46$ to 0.63 , $p < 0.01$) and stiffness ($r = 0.46$ to $r = 0.51$, $p < 0.01$). BMD at all five regions was correlated with physical function ($r = 0.40$ to $r = 0.69$, $p < 0.03$ to $p < 0.001$). These correlations indicate that as bone density increased WOMAC scores for pain, stiffness or physical function worsened.

Table 10.4: Bone mineral density (Mean \pm SD; g/cm²) of the symptomatic osteoarthritic knees and contralateral knees for each region of interest (RIO).

Bone mineral density		ROI 1 Central tibia	ROI 2 Lateral tibia	ROI 3 Medial tibia	ROI 4 Lateral femur	ROI 5 Medial femur
Osteoarthritic knee	mean	0.51	0.73	1.00	0.78	0.74
	SD	0.18	0.29	0.31	0.24	0.33
Contralateral knee	mean	0.52	0.79	1.01	0.83	0.75
	SD	0.22	0.29	0.31	0.24	0.23

BMD in Osteoarthritic (OA) and Contralateral (contra) knee

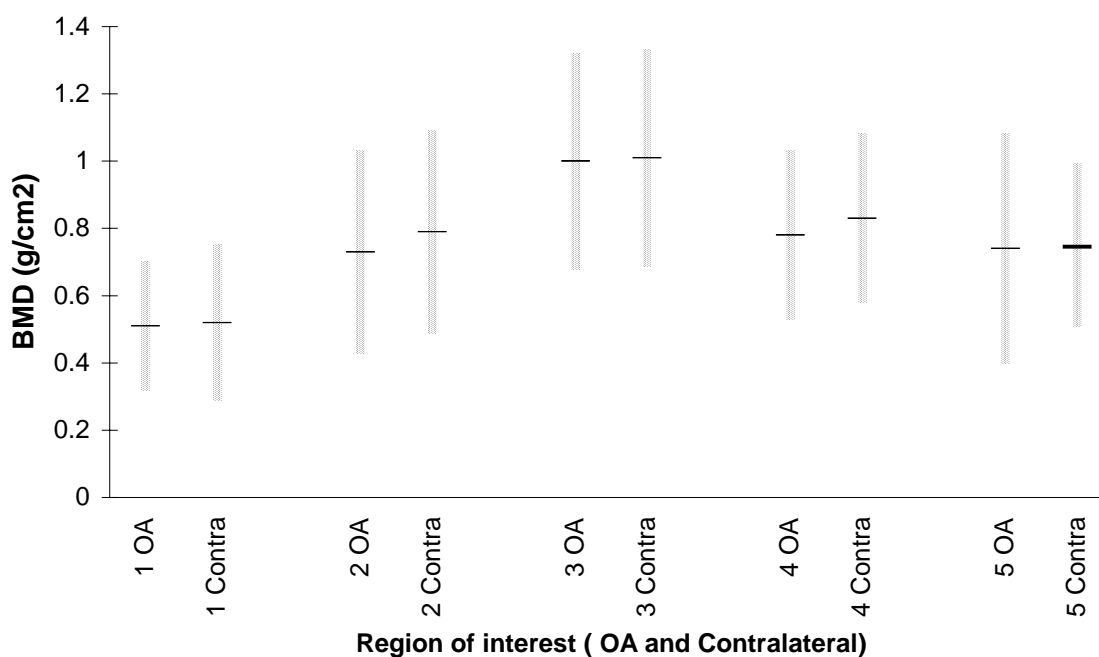


Figure 10.8: Comparison of bone mineral density (BMD; mean \pm SD) in the symptomatic (OA) and contralateral (Contra) knee of osteoarthritic subjects at each region of interest.

Discussion

This study has demonstrated how the kinematic characteristics of osteoarthritic knees differ from healthy knees. Roll back was markedly reduced in the medial and lateral compartments of the osteoarthritic knees. The difference in the tibiofemoral contact pattern that indicates longitudinal rotation in healthy knees was also reduced in the osteoarthritic knees. These kinematic changes were associated with severity of osteoarthritis, as indicated by Kellgren Lawrence grades from x-rays. The magnitude of change in the tibiofemoral contact pattern of the lateral compartment was also associated with symptoms of pain, stiffness and limitations to function.

Kinematic characteristics of osteoarthritic knees

Loss of normal roll back in the osteoarthritic knees demonstrated the change to the normal roll/glide characteristics of knee motion. In the healthy knee the femur predominantly rolls across the tibial plateau between 0° and 30°, then from 30° onwards the femur combines roll with glide on the surface of the tibial plateau, otherwise it would reach the posterior the tibial plateau rim before flexion was completed (Muller 1983). In addition, insufficient roll back would cause the shaft of the femur to impinge on the posterior tibial rim, also limiting knee flexion. Control of the roll/glide behaviour was attributed to the cruciate ligaments (Muller 1983). In the osteoarthritic knee the amount of roll back was reduced and tibiofemoral contact occurred anteriorly on the tibial plateau. This loss of normal roll back may be a contributor to reduced flexion range of motion.

Reduced range of knee flexion often accompanies osteoarthritis (Walker et al. 2001). In healthy knees studied in deep flexion, the position of the femur on the posterior rim of the

tibial plateau has been described (Hefzy et al. 1998; Nakagawa et al. 2000; Komistek et al. 2003). The medial femoral condyle rides up onto the posterior horn of the meniscus and the lateral femoral condyle rolls back to the posterior rim of the tibial plateau, displacing the posterior horn of the meniscus posteriorly also (Nakagawa et al. 2003). In total knee arthroplasty, loss of normal femoral roll back has been considered a limiting factor in achieving knee flexion and cruciate retaining prostheses have been designed to facilitate roll back (Bellemans et al. 2002). In the osteoarthritic knees, the posterior translation of the femur on the tibial plateau was markedly restricted. It could be anticipated that this could be a reason for compromised flexion range of motion in osteoarthritic knees.

The loss of the normal roll back in the osteoarthritic knees was more apparent in the lateral compartment than in the medial compartment, which already has little roll back in the healthy knee. In the healthy knees it is the roll back of the lateral femoral condyle while the medial femoral condyle remains centred on the tibial plateau that indicates the longitudinal rotation of the knee during flexion. The loss of roll back in the lateral compartment in particular, demonstrated the loss of longitudinal rotation in the osteoarthritic knees. The lateral compartment of the healthy knee has greater mobility than the medial compartment (Brantigan and Voschell 1941), which permits the lateral compartment rollback during knee flexion. In the osteoarthritic knees the lateral compartment behaved like fixed-axis ball-and-socket joint. The loss of longitudinal rotation in osteoarthritic knees has previously been described in standing subjects in the last 30° knee extension (Nagao et al. 1998). Nagao et al (1998) only examined knee flexion from 0° to 30°, but Figure 10.3 suggests that the difference in kinematics may be greater in deeper knee flexion. The present study shows that this loss of longitudinal rotation in osteoarthritic knees may continue throughout knee flexion.

The kinematic changes are apparent in early stages of osteoarthritis, as evidenced by the altered kinematics of asymptomatic knees with radiographic signs of osteoarthritis. Indeed asymptomatic knees, with no pain and no stiffness, may already have begun to exhibit altered kinematics. Knees with grade 2 osteoarthritis already had a different tibiofemoral contact pattern to healthy knees.

Kinematic characteristics and wear

Medial compartment contact patterns may be related to the pattern of wear in the medial tibial plateau. At total knee replacement, anteromedial wear areas have been described on the tibial plateau (White et al. 1991) and these wear areas increase in size and depth as osteoarthritis progresses (Fukubayashi and Kurosawa 1980; Harman et al. 1998). In the present study, there was increasing kinematic abnormality in the medial compartment as severity progressed between grade 2 and grade 3 osteoarthritis (Figure 10.7). In knees with grade 3 osteoarthritis, the tibiofemoral contact pattern was more anterior on the tibial plateau throughout knee flexion than in other grades of osteoarthritis. It is possible that the area of wear in the medial compartment of knees with grade 3 osteoarthritis tends to hold the femur in the wear cupola, anteriorly on the tibial plateau.

The severity of knee osteoarthritis of our subjects ranged from minimal to severe in the symptomatic knees and from none to moderate in the asymptomatic knee. The high incidence of asymptomatic radiographic osteoarthritis in the contralateral knees was probably because many of these subjects had primary idiopathic osteoarthritis, which is most commonly bilateral (Radin 1987). One subject had an ACL injury in the symptomatic knee, but may have had concurrent primary idiopathic osteoarthritis. Therefore it is not

surprising that 7 out of 14 subjects had asymptomatic osteoarthritis in the contralateral knee.

Role of the ACL in kinematic changes

The loss of longitudinal rotation could be due to attrition of the ACL or changes to the soft tissue structures of the knee limiting the extension range of motion (Nagao et al. 1998). The ACL is considered important to guide the rotation of the knee in terminal extension (Hallen and Lindahl 1966; Fuss 1992) and the incidence of ACL abnormality in osteoarthritic knees has been reported to be 50% to 75% (Allain et al. 2001). In our study, while 7 subjects had ACL deficiency, loss of longitudinal rotation was not associated with ACL deficiency, but rather was associated with severity of disease.

The incidence of ACL deficiency in the present study was similar to that previously reported (Wada et al. 1996; Allain et al. 2001). However, conclusions about the attrition of the ACL in knee osteoarthritis cannot be drawn from this small sample of convenience. Wada et al (1996) found 50% of knees had a completely absent ACL, 25% partly torn and only 25% had an intact ACL (Wada et al. 1996). The PCL, however, was intact in all the knees. Allain et al (2001) found similar incidence of ACL damage in osteoarthritic knees, but in 75% of cases where the ACL was torn the PCL also had histological evidence of degeneration. They suggested that the absence of the ACL indicated that the quality of the PCL as a viable restraint structure was probably also compromised. The menisci also, are frequently found to be macerated or absent in advanced knee osteoarthritis (Buckwalter and Mankin 1997). In our sample of 14 subjects, only one had no meniscal damage. Thus, the evidence of joint damage in this sample of osteoarthritic subjects is consistent with that reported by others.

BMD, function scores and radiographs as indicators of osteoarthritis

In the present study there was no difference in BMD between the symptomatic and contralateral knees of osteoarthritic subjects and no observed relationship between radiological severity of osteoarthritis and BMD. This could be due to the wide standard deviation and wide spread of results. BMD has been used to indicate severity of disease in osteoarthritis and disease progression (Hurwitz et al. 1998; Andriacchi et al. 2000; Hurwitz et al. 2001; Wada et al. 2001; Hulet et al. 2002). In studies of osteoarthritic subjects, subchondral BMD increases measured by dual photon absorptiometry and DEXA has been associated with progression of the radiological stage of osteoarthritis (Madsen et al. 1994). In a group with severe osteoarthritis BMD was correlated with the varus/valgus angulation of the knee, clearly demonstrating the relationship between medial compartment osteoarthritis and increased BMD (Hurwitz et al. 2000). In the present study, increased BMD was associated with worsening pain stiffness and function of the osteoarthritic subjects. The WOMAC questionnaire was designed to be an indicator of the effect of osteoarthritis on quality of life and as such is an indicator of severity of symptoms of osteoarthritis. It is interesting, however, that BMD increase was associated with disease severity in terms of symptoms but not radiographic changes. Measures of severity in terms of impact on function and quality of life were developed because there is poor agreement between radiographic measures of severity of osteoarthritis and symptoms (Peat et al. 2001).

We have shown that altered tibiofemoral contact patterns in osteoarthritic knees were associated with severity of osteoarthritis graded from x-rays. We have also shown that change in the tibiofemoral contact pattern in the lateral compartment of the unloaded knee was associated with worse symptoms and poorer function. Kinematic changes appear to

occur early in the disease process, frequently before the onset of symptoms. A question arises from this work as to whether kinematic changes can be used as an early predictor of osteoarthritis, so that early intervention or prevention of deterioration might be possible. The loss of longitudinal rotation and in particular lateral compartment roll back was associated with poor pain and function scores. It may be that interventions to restore the longitudinal rotation may provide relief of symptoms. This warrants further investigation. There is still much to learn regarding the characteristics of knee kinematics expressed by tibiofemoral contact patterns in the osteoarthritic knee, but this study has provided a description of osteoarthritic knee kinematics that was previously unknown.

Conclusions

The kinematics of osteoarthritic knees were different from healthy knees and the kinematic abnormalities were a function of the severity of osteoarthritis. The difference in the knee kinematics of osteoarthritic knees was greatest in the lateral compartment of the knee, but both medial and lateral compartments exhibited a loss of the normal posterior translation of the femur, which normally occurs during knee flexion. In the healthy knees the lateral compartment contact pattern demonstrated that the femur moved posteriorly during knee flexion from 30° – 90°, more than in the medial compartment and this indicated the occurrence of longitudinal rotation of the knee during flexion. In the osteoarthritic knees this longitudinal rotation was markedly reduced. This loss of longitudinal rotation was related to the severity of osteoarthritis.

CHAPTER 11

CONCLUDING REMARKS

Prior to the research work presented in this thesis, the field of knee kinematics had concentrated on understanding the normal knee, knowledge that was required for development of knee prostheses, and understanding the kinematics of the prosthetic knee in vivo. The relationship between abnormal kinematics and degenerative change had not been examined since the early experiments in the 1970's using rudimentary 2-dimensional imaging (Frankel et al. 1971). Three-dimensional kinematics had been examined in terms of the flexion/extension axis using MRI (Todo et al. 1999; Hill et al. 2000), and in terms of the relative motion of the femur and tibia, in normal, recently injured or reconstructed knees (Niitsu et al. 1991; Beard et al. 2001; Brandsson et al. 2001; Brandsson et al. 2002; Hollman et al. 2002; Georgoulis et al. 2003). Of events occurring at the tibiofemoral interface very little was known, despite this interface being the site of degeneration, and in particular, little was known about the kinematics of chronically injured or osteoarthritic knees. Consequently, there was poor understanding of the relationship between the aberrant knee kinematics of injured knees and the consequent degenerative changes.

Synthesis of findings

Healthy knee kinematics

We established and validated a technique using MRI to reliably plot the position of contact of the femoral condyle with the tibial plateau. Using this technique, we were able to complete the first successful mapping of the tibiofemoral contact pattern of normal knees during flexion. The kinematic characteristics derived from the contact pattern included the nature of

roll and glide occurring at the tibiofemoral interface: the femoral condyles rolled on the tibial plateau between 0° and 30°, but between 30° and 90° the medial condyle remained centred on the tibial plateau and predominantly glided in place, whereas the lateral femoral condyle continued to roll back across the tibial plateau during flexion. This difference between the medial and lateral tibiofemoral contact patterns described the longitudinal rotation of the knee during flexion. While the medial condyle remained in place during flexion, the lateral condyle continued to roll back, indicating that the axis of rotation is probably located at the center of the medial compartment.

The lack of difference we found in the contact pattern between the loaded and unloaded conditions in healthy knees was not predicted by previous work in this area. While tibiofemoral contact patterns have not been described, other kinematic characteristics have been reported as different in loaded knees. These differences include suppression of longitudinal rotation (Hill et al 1999), unloading of the ACL (Tountoungi et al 2000) and an increase in the tibiofemoral contact area (Fukubayashi and Kurosawa 1980). However, in our study of healthy control subjects there was no difference between tibiofemoral contact patterns in loaded and unloaded knees. We did not find a change in the tibiofemoral contact pattern to indicate suppression of rotation, but our experiment was more strictly controlled than that of Hill et al (1999). Hill et al (1999) compared knee flexion/extension in sitting (which is an open-chain exercise) with a standing squat position (which is a closed-chain exercise). Thus, there are two changed variables; the body position changed from sitting to standing and the exercise changed from open to closed-chain, in addition to loaded versus unloaded conditions. It may be the closed-chain conditions that suppressed rotation in the knee when compared to the open-chain (sitting) condition. In our study, the body position was

controlled and remained supine, with the feet on the footplate (closed-chain), and the load of 150N was much less than body weight. There are many variations in kinematics within the available envelope of passive motion of the knee (Blankevoort et al 1988). Our experiment in healthy knees demonstrated that with other variables controlled, the tibiofemoral contact pattern was unchanged when the subjects pressed down through the feet against a 150N load. The lack of difference we found in healthy controls may change the way we describe the influence of loading and muscular activity on knee kinematics.

The new findings presented in this thesis provide a clear description of the normal tibiofemoral contact pattern, and form a basis from which to explore the effect of injury on kinematics. Exploration of changes in tibiofemoral interface events is relevant to understanding the process of articular cartilage degeneration in injured knees and is also relevant to issues of wear in knee prostheses. Tibiofemoral contact patterns can be used to quantify and analyse the changes in kinematics due to knee injury and pathology now that the normal contact pattern has been mapped.

Pathological knee kinematics

The tibiofemoral contact patterns were mapped for three stages of knee pathology; recent ACL-injured knees, chronic ACL-deficient knees and osteoarthritic knees, finding distinctly different characteristics at each stage. The tibiofemoral contact pattern in ACL-injured knees demonstrated that although the characteristic longitudinal rotation of the knee was maintained, the contact occurred more posteriorly on the tibial plateau. The difference between the contact pattern in chronic ACL-deficient knees and contralateral healthy knees

was associated with the degree of degeneration and damage to the articular surface of the knees. In addition, the difference in tibiofemoral contact patterns in osteoarthritic knees was associated with the severity of osteoarthritis in the knees.

In the ACL-injured knees, the contact pattern occurred more posteriorly on the tibial plateau, but with the posterior tibiofemoral contact pattern more pronounced in the lateral compartment. This indicated that the roll and glide characteristics were maintained, but with a more medial axis of rotation compared to the normal knee. This shift in axis could suggest an increased role for the MCL in maintaining stability of the ACL-injured knee. In the loaded condition the contact pattern was still significantly different to the contact pattern in healthy contralateral knees. The abnormal contact pattern in the loaded condition suggests that using a closed-chain condition for exercises, rather than an open-chain exercise, is not sufficient to normalise the contact pattern in ACL-injured knees. Thus, it could be dangerous to assume closed-chain exercises are benign for unstable and reconstructed knees.

The chronic ACL-deficient knees showed a pattern of tibiofemoral contact that was more posterior than the healthy contralateral knee, and particularly in the medial compartment, not the lateral compartment as in the recently ACL-injured knees. This posterior pattern in the medial compartment results from the secondary restraints to anterior tibial translation in the ACL-deficient knee, the MCL and medial meniscus, becoming attenuated or stretched or from degenerative changes influencing their effectiveness as restraints. The altered kinematics of the medial compartment were associated with severity of joint damage in the chronic ACL-deficient knee. In contrast, time since injury was not associated with degenerative change,

indicating that it is not time, but kinematics that is associated with the degenerative change process.

Osteoarthritis produced pronounced changes to the tibiofemoral contact pattern. The lateral femoral condyle no longer rolled back on the tibial plateau and the knee behaved like a hinge, rather than a multiaxial joint. The loss of femoral roll back in osteoarthritic knees is likely to be responsible for the loss of flexion range of motion in the knees. The relationship between loss of roll back and loss of flexion should be further investigated, particularly because these impairments are potentially remediable by physiotherapy.

At the late stages of the degenerative process ACL deficiency was not associated with altered kinematics. That is, although ligament damage in early osteoarthritis may have affected the kinematics early in the process of degeneration, it did not appear to influence the kinematics of severely osteoarthritic knees. The relationship between ligament integrity and kinematics in the osteoarthritic knee is integral to the issue of ligament retention or sacrifice in knee arthroplasty. However, kinematics in late-stage osteoarthritis were not affected by ACL integrity. This is likely to be because other changes associated with knee osteoarthritis, for example, changes to bony structure, and fibrous thickening of the synovium and capsule are having a greater role in controlling knee kinematics. In the light of these findings the arguments for ACL retention in knee arthroplasty need to be re-evaluated.

Hamstrings autograft reconstruction of ACL-injured knees appears to restore the normal contact pattern to that of the healthy contralateral knee, though neither the reconstructed nor contralateral knees of injured subjects had a normal healthy contact pattern at 2 years.

Longitudinal studies following subjects after knee reconstruction are required to examine the effect of time on changes in the tibiofemoral contact pattern. There appear to be changes with time in both the reconstructed knee and the healthy contralateral knee, which may be clarified by a prospective, controlled study. While the tibiofemoral contact pattern of the reconstructed knee was the same as the healthy contralateral knee, it was not restored to the contact pattern of healthy control subjects. The long-term outcome of this variation in the tibiofemoral contact pattern is unclear. While altered tibiofemoral contact patterns have been associated with the degenerative process, the degenerative consequences of these altered tibiofemoral contact patterns shown at 2 years following knee reconstruction are not known. Studies of knee reconstruction are limited to 7-year follow-up, so the incidence of osteoarthritis in the reconstructed knee over the long-term is not yet clear (Jomha et al. 1999; Pinczewski et al. 2002; Ruiz et al. 2002). Until subjects with hamstrings autografts can be followed-up for the long term, knowledge about the ability of knee reconstruction to correct the tibiofemoral contact pattern is an incremental advance in predicting whether knee reconstruction will be able to prevent the sequellae of degenerative change that are evident in the unoperated ACL-deficient knee.

Tibiofemoral contact mapping using MRI, may have potential for development as a screening tool to indicate which patients are best advised to have surgery. There may be patients with good neuromuscular coordination, who have minimal side-to-side difference in contact pattern, and may avoid degenerative sequellae from ACL-deficiency. There may be others with a greater side-to-side difference, for whom surgery may be indicated. Knowledge about kinematic behaviour of a patient's knee may in the future inform decisions about whether surgery is indicated, as well as optimal time-frames for intervention.

There were characteristic changes in the tibiofemoral contact pattern for recently injured knees, that were different from chronic ACL-deficient knees, and associated with the extent of degenerative changes present in the knee. It is not possible to determine from this cross sectional study whether the altered tibiofemoral contact pattern causes the degenerative changes evident in chronic ACL-deficient, or even osteoarthritic knees, or whether the tibiofemoral contact pattern is reflecting the structural and mechanical changes that are occurring as a result of the degenerative process. Either way, tibiofemoral contact pattern mapping provides a robust method to quantify and compare kinematic changes and is sensitive to altered kinematics in a variety of pathological states.

Questions arising from the work

Major questions that have arisen from this body of work include:

- Should closed chain exercises continue to be recommended for rehabilitation, given that the loaded ACL-injured knee exhibited an abnormal tibiofemoral contact pattern?
- Does the lack of difference in the contact pattern between the unloaded and loaded knees in all stages of pathology studied infer that subjects were effectively stabilising the knee by contracting the hamstrings and quadriceps muscle groups?
- Can the degenerative consequences of chronic ACL-deficiency be avoided by training dynamic stability?

The prescription of closed-chain exercises for strengthening unstable knees has a strong basis from both in vitro and in vivo studies which show that the anterior translation resulting from quadriceps activity is reduced during joint compression. Our study did not examine the

tibiofemoral contact pattern produced during open-chain quadriceps exercises, nor compare open with closed-chain exercises, instead we compared the relaxed (unloaded) condition with a closed-chain condition (the leg-press). We found that the tibiofemoral contact pattern for both unloaded and loaded (closed-chain) conditions were abnormal in the ACL-injured knee. Therefore, performing closed-chain exercises did not normalise the contact pattern, so it can not be assumed that exercising using only closed-chain exercises will protect the knee from degenerative sequelae.

The tibiofemoral contact pattern did not differ between the unloaded and loaded conditions in either healthy or ACL-injured knees, suggesting that muscular activity could have been controlling the knee kinematics. EMG could not be used to measure muscle activity during this kinematic analysis, as the MRI field would induce a current in the leads. There are already many EMG studies of muscle recruitment and timing of muscle activation during a variety of functional tasks that suggest ACL-deficient subjects can develop very effective dynamic stability skills (Steele and Brown 1999). In order to perform a loaded leg-press, subjects balance the muscle activity of quadriceps and gastrocnemius muscles, both of which produce a vector component of anterior tibial translation, with hamstrings muscles, which have a component of posterior tibial translation due to the insertion posterior and distal to the knee. In order for no net translation effect to be measured at the tibiofemoral contact, it is likely subjects have developed neuromuscular coordination skills required to stabilise the knee. However, the tibiofemoral contact pattern in the ACL-injured knees was significantly different from the intact knees, both when relaxed and when loaded. The dynamic stability produced by neuromuscular coordination skills is effective, but perhaps the muscles cannot quite compensate for the loss of the ACL as a passive restraint in the joint.

The question concerning whether neuromuscular training, as used to improve dynamic stability, can prevent the degenerative sequelae of chronic ACL-deficiency would need to be examined over a prolonged period. An intervention study should be conducted to examine whether training in dynamic stability and neuromuscular re-education normalises the TF contact pattern in the short and the long term. Although it would be interesting to measure the neuromuscular characteristics of our group of subjects with no degenerative changes, this was outside the scope of our study. The three subjects who coped well with chronic ACL-deficiency and with no degenerative changes participated in non-pivoting sports to maintain strength and fitness. However, this sample is too small to enable determination of risk factors for degeneration. Whether kinematic stability can prevent degenerative change is still to be tested, but it is known that aberrant kinematics are associated with degenerative change.

Understanding of normal and pathological tibiofemoral contact patterns may be useful for knee prosthetic design, as it is commonly understood that interface wear determines lifespan of the prosthesis. Yet biomechanists involved in design of knee prostheses have been aiming to replicate the kinematics of a normal healthy knee, rather than an osteoarthritic knee (Freeman 1998; Stiehl et al. 2000; Banks et al. 2003). However, the contact pattern of osteoarthritic knees is not a normal contact pattern, its dysfunction may be responsible for the loss of flexion range of motion, and the loss of rotational mobility important to activities of daily living. It may be that implanting a prosthetic knee designed to replicate normal kinematics is not optimal. The osteoarthritic knee has altered bony and soft tissue features, including a thickened synovium, weak muscles, and frequently attenuated cruciate and collateral ligaments. Perhaps this is the reason that kinematics of the replaced knee are far from normal, and exhibit paradoxical roll-forward and condylar lift-off in vivo (Dennis et al.

2001; Bellemans et al. 2002). A better understanding of kinematics of osteoarthritic knees provides a better framework from which to anticipate how kinematics might behave with a replaced knee.

This is the first time the tibiofemoral contact pattern of osteoarthritic knees has been mapped. It is evident that loss of longitudinal rotation and loss of roll back in osteoarthritic knees is not due to attrition of the ACL, but other structural and mechanical changes to the knee. The role of the ACL in guiding the kinematics of the knee is finally ended in the late stages of osteoarthritis.

Conclusion

These findings describe the effect of ACL injury on knee kinematics, and the relationship between kinematics and degenerative change. In a model of ACL injury, the changes to the kinematics of the knee and changes to the structure of the knee during the degenerative process are reflected in the altered tibiofemoral contact pattern. Only in the late stages of osteoarthritis and in the environment of gross structural changes to the knee is the affect of ACL-deficiency no longer apparent. This body of work has provided a fresh insight into kinematics and degenerative change in ligament-injured knees.

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Comparison of kinematics in the healthy and ACL injured knee using MRI

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Abstract

Magnetic Resonance Imaging (MRI) was used to examine the characteristics of abnormal motion in the injured knee by mapping tibiofemoral contact. Eleven healthy subjects and 20 subjects with a unilateral ACL injury performed a leg-press against resistance. MRI scans of both knees at 15° intervals from 0° to 90° of flexion were used to record the tibiofemoral contact pattern.

The tibiofemoral contact pattern of the injured knees was more posterior on the tibial plateau than the healthy knees, particularly in the lateral compartment. The tibiofemoral contact pattern of the loaded knees did not differ from the unloaded knees. The difference in the tibiofemoral contact pattern in the ACL injured knee was associated with more severe knee symptoms, irrespective of the passive anterior laxity of the knee.

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1. Introduction

Abnormal kinematics in the ACL injured knee has been considered responsible, at least in part, to the degenerative changes observed (Friden et al., 1993; Osternig et al., 2000; Vergis et al., 1997). Anterior cruciate ligament (ACL) injuries are common, the incidence being 30 per 100,000 of the population per year in the USA (Miyasaka et al., 1991). Unfortunately, the risk of developing osteoarthritis after ACL injury rises to 60–90%, with the highest incidence being in those who return to sport (Clatworthy and Amendola, 1999; Gillquist and Messner, 1999; Otto et al., 1998). Degenerative change in the ACL injured knee is due in part to instability leaving the knee vulnerable to repeat trauma, and in part to altered kinematics including shearing at the articular cartilage. An understanding of

how the kinematics of the knee is altered by ACL injury is important in order to relate the aberrant kinematics to the process of degeneration.

Kinematics of the ACL injured knee have been studied using plain films to measure anterior instability in patients performing a standing lunge (Friden et al., 1993) and fluoroscopy (Dennis et al., 1996). However, two-dimensional data have limited application for analysis of the complex three-dimensional motion of the knee. Three-dimensional analysis using electrogoniometry (Vergis and Gillquist, 1998; Vergis et al., 1997), and radiostereometry (Brandsson et al., 2001, 2002) has demonstrated anterior translation and external rotation of the tibia in the ACL injured knee, but these are complex, expensive and invasive techniques and are generally impractical in the clinical situation.

Magnetic Resonance Imaging (MRI), like radiostereometry, is a direct visualisation method, which records the position and hence the motion of the knee with precision. Advantages of MRI are that it is non-invasive, and is readily accessible in the clinical situation. Open field (Hill et al., 2000a; Vedi et al.,

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1999) and standard closed MRI (Scarvell et al., 2001, 2002; Smith et al., 1999; Todo et al., 1999) have been used to analyse knee kinematics in three dimensions. The kinematics have been described in terms of a tibiofemoral contact map. This allows the roll/glide characteristics and the longitudinal rotation of the knee to be visualised and quantified. In normal healthy subjects studied using this technique, a high degree of variation has been demonstrated, but with good right to left consistency within individuals, indicating that the uninjured knee may be reliably used as a control (Scarvell et al., 2001; Smith et al., 1999). The purpose of this study was to record the motion of the ACL deficient knee using MRI, in order to assess the characteristics of abnormal motion in the ACL injured knee that may contribute to progressive degenerative change. Finally this technique may be useful in predicting those patients at risk of osteoarthritis due to the kinematic behaviour.

2. Method

Twenty subjects aged between 21 and 52 years, with a unilateral ACL injury were recruited. Subjects were excluded if there were any contraindications to MRI, may have been pregnant, or they were over 180 cm tall (to permit knee flexion in the MRI tunnel). Subjects were also excluded if history of injury or symptoms were present in the contralateral knee, so that the uninjured knee could act as the control. Eight subjects were male, 12 female. All ACL injuries were sustained within 3 years of testing. All subjects provided informed consent. Ethics approval for the study was obtained from the Australian Capital Territory Department of Health and Community Care Human Ethics Committee, and the University of Sydney Human Ethics Committee

2.1. MRI imaging procedure

To enable standardisation of knee flexion positions between 0° and 90° a wooden frame with a sliding footplate was fitted to the MRI couch. For the loaded and unloaded conditions a rope and pulley weighted with 150 N was attached to the frame (Fig. 1). Elastic straps maintained neutral tibial rotation and thigh adduction by stabilising the feet and ankles. Imaging of both knees simultaneously was performed using a 1.5 T Siemens Magnetron Vision (Erlangen, Germany). A body coil was used to generate parasagittal images, perpendicular to each tibial plateau. Images were taken of both knees, in the loaded and unloaded conditions, at seven angles of 15° increments from full knee extension (0°) to 90° flexion.

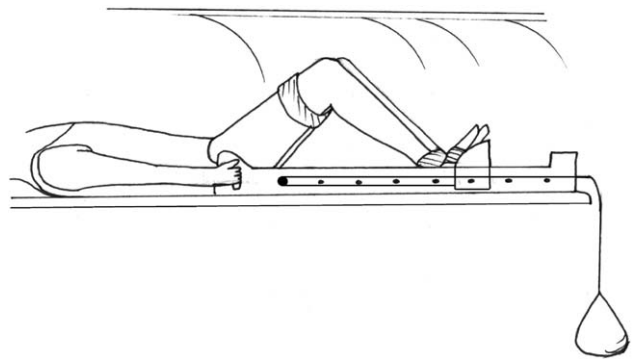


Fig. 1. Diagram of supine subject in MRI tunnel. Knee flexion angle was controlled by the position of a sliding wooden footplate. A 150 N weight and pulley provide resistance to knee extension.

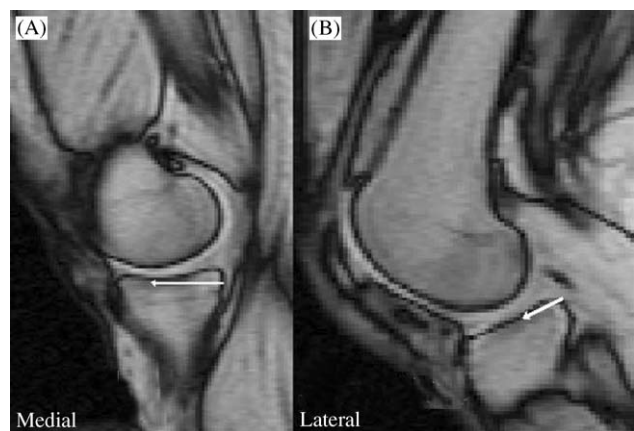


Fig. 2. Sagittal plane MRI scans demonstrating the measurement of the tibiofemoral contact point, referenced from the posterior tibial cortex, for the medial (A) and lateral (B) compartments of the knee.

2.2. Tibiofemoral contact point measurement

The position of the femoral condyle on the tibial plateau was recorded as the distance from the posterior tibial cortex to the point of tibiofemoral (TF) contact of the medial and lateral femoral condyle (Fig. 2). Where contact occurred over a wide area, the area centroid was used. To account for variation in the size of subjects, cortex to contact point distance measurements were normalised in proportion to mean tibial dimensions. The mean AP diameter of the medial tibial plateau was 50 ± 4.4 mm, and the lateral tibial plateau was 42 ± 2.17 mm. The TF contact points were mapped onto a tibial plateau of standard dimensions.

2.3. Knee injury variables recorded

In addition to the knee kinematics, the passive anterior laxity, function, and knee damage visible at arthroscopy were recorded for each subject. The KT 1000 arthrometer was used to measure anterior laxity,

by quantifying the anterior displacement in the Lachman's manoeuvre (Daniel et al., 1985). We measured anterior tibial displacement in both knees five times: at 15, 20, 30 lb of anterior drawer, during an unloaded straight leg raise, and with a maximum manual anterior drawer using the KT 1000. A side-to-side difference of 3 mm or more on a manual maximum anterior drawer is considered indicative of an ACL injury, with a sensitivity of 85% (Daniel et al., 1985). The Cincinnati knee score (Barber-Westin et al., 1999) was used to measure the symptoms, functional limitations, activity levels of subjects and details of the physical examination. The result is a score out of 100, where 100 is normal, with full function. The Cincinnati knee rating score has high reliability and is sensitive to changes in the ACL injured population (Barber-Westin et al., 1999).

Injuries to the ACL are frequently associated with injuries to other structures of the knee, either at the time of injury, or subsequent to the injury. These associated injuries may impose confounding effects on the kinematics of the injured knee. All subjects in this study underwent arthroscopic knee reconstruction within two months of testing. At arthroscopy, visible joint damage, meniscal damage and cartilage wear were recorded (Noyes and Stabler, 1989). Thus, the effects of meniscal damage and articular cartilage damage on knee kinematics could be tested.

3. Reliability of protocol

In order to test the reliability of the protocol, 12 healthy subjects (7 male, 5 female, aged 20–50 years) with no history of injury or symptoms in either knee or lower limb were recruited. The technique was evaluated for reliability by testing the effects of MRI image quality, distortion, and slice thickness on the accuracy of the measurement technique for TF contact point measurement (Scarvell et al., 2001).

We compared two MRI sequences for quality of image, reliability of TF contact point measurement and practicality of use. The two sequences were the T1 weighted fast spin echo sequence (SE), and the spoiled gradient echo (GE) sequence. Five healthy subjects (1 male, 4 female) were scanned using an SE sequence, and seven (6 male, 1 female) were scanned using a GE sequence. The SE sequence had a longer scanning time, (2.31 min per sequence, totalling 1½ h session time) but higher image resolution and narrower slices. Using SE 14 slices were obtained in each knee (slice width 7 mm). GE scans were faster, being designed for imaging in a breath hold (Behrens and King, 2000). Each GE sequence takes 40 s, with a session time of 35 min. Using GE eight slices were generated through each knee, approximately 10 mm apart (TR = 160.0, TE = 2.3/1,

TA = 00:46), with a 256 × 256 matrix. It was essential that the subjects did not move during the procedure, or the images were contaminated. Since fatigue during the loaded leg press sequence also produced movement or tremor, the shorter scanning time using GE was preferred.

The reproducibility of the TF contact point measurements from SE and GE sequences were compared for both knees at 15° intervals from 0° to 90° flexion, while unloaded and loaded. The medial and lateral TF contact points were mapped reliably using both SE sequences and GE sequences with Intra Class Correlation $(_{2,1})$ of 0.96 (CI 99% 0.94–0.97) and 0.94 (CI 99% 0.91–0.97), respectively. The good reliability of both SE and GE sequences used for the technique of TF contact point mapping meant that the faster gradient echo sequence could be used without compromising the reliability of the measurements.

3.1. Loaded knee images

A comparison between a 150 N and 250 N load was made in one healthy subject. The TF contact points of the knee were not altered by the heavier load ($p = 0.95$). Since this protocol for imaging knee kinematics is designed for the clinical situation, where injured subjects are likely to have muscle weakness the procedure was continued using a 150 N load.

3.2. Content validity

The recorded TF contact maps were tested for content validity against three statements drawn from the current literature on knee kinematics: that the right and left knees of healthy subjects would be symmetrical (Smith et al., 1999); that the medial and lateral compartments of the knee would be different, reflecting the longitudinal rotation of the knee (Elias et al., 1990; Hollister et al., 1993; Kärrholm et al., 2000; Todo et al., 1999); and that loading the healthy knee would not produce a different contact map (Hill et al., 2000a, b). The contact map data were analysed using repeated measures ANOVA with factors of right and left knee, loaded and unloaded knee, and medial and lateral side of the knee. Results were consistent with the three statements: the left and right knee were consistent for each individual ($F_{1,80} = 0.005$; $p = 0.943$); the medial and lateral TF contact maps were different ($F_{1,80} = 253.9$; $p < 0.0001$), and the loaded and unloaded knees were not different in the healthy knee ($F_{1,80} = 0.007$; $p = 0.935$). The content validity of the contact maps was supported for each of the three statements. MRI analysis of knee kinematics as described by this technique of TF contact point mapping provides a reliable method of recording the TF kinematics of the knee.

3.3. Statistical analysis

The TF contact points recorded for the healthy and ACL injured knees were compared using repeated measures ANOVA. Factors included in the analysis included the angle of knee flexion, the injured or contralateral knee, the loaded and unloaded condition, and the medial and lateral compartments of the knee.

Difference between the passive anterior laxity of the injured and the contralateral knee was analysed using a paired student's *t*-test.

The deviation from the healthy knee contact pathway exhibited by the injured knee is quantified as the side-to-side difference in the TF contact points. Articular cartilage damage recorded at arthroscopy was compared to the mean of the side-to-side difference recorded at each knee flexion angle, using one-way ANOVA. Passive anterior laxity and Cincinnati knee score were analysed for their correlation with the mean of the side-to-side difference in the contact points, using a Pearson's *r*.

4. Results

The mechanism of injury for the twenty ACL injured subjects included 18 playing sports, 2 by falling from a height of ≈ 1 m. There were 9 left knees injured and 11 right knees. At arthroscopy, internal knee joint damage was present in 9 subjects. Damage included femoral cartilage fibrillation of the medial femoral condyle in 2 subjects, (2a and 2b changes, and 1b changes) (Noyes and Stabler, 1989), and patellofemoral cartilage damage in 2 subjects (1a and 1b). There was no tibial cartilage damage in any of the subjects. In 3 subjects there was medial meniscal injury including one small stable tear of the posterior horn, one tear of the posterior horn requiring partial meniscectomy and one bucket handle tear. In 3 subjects there was damage to the lateral meniscus including one mild crush, one small tear which was trimmed, and one old partial meniscectomy seen, all of the posterior horn. There were 11 subjects with ACL tears with no other associated joint damage.

Anterior laxity at 30 lb, the straight leg raise, and maximum manual anterior drawer, of the injured knee were each significantly greater than the healthy knee ($p < 0.001$). Passive anterior laxity using the KT 1000 arthrometer supported the clinical diagnosis of ACL injury in 19 of the 20 subjects by a side-to-side difference of ≥ 3 mm at manual maximum anterior drawer. The side-to-side difference in anterior displacement of the tibia with a 30 lb anterior drawer was 2.7 ± 2.7 mm. The side-to-side difference with the straight leg raise test was 3.9 ± 2.6 mm, and for the manual maximum anterior drawer was 5.5 ± 3.3 mm. The ACL tear was confirmed at arthroscopy for all 20 subjects.

4.1. MRI knee kinematics

The TF contact pattern was located posteriorly on the tibial plateau in the injured knee ($F_{(1,152)} = 6.5$, $p = 0.012$) compared to the healthy knee (Fig. 3). In the medial compartment of the knee this difference was most pronounced at 0° and 15° . At 0° the medial femoral condyle was located 32.2 ± 5.1 mm from the posterior tibial cortex landmark in the ACL injured knee, and 33.2 ± 3.9 mm in the healthy knee. Further into knee flexion the medial compartment of the injured knee followed a similar pattern to the healthy knee. In the lateral compartment of the knee, the femur rested more posteriorly on the tibial plateau throughout the range of knee flexion. The lateral femoral condyle was located at 24.7 ± 4.5 mm from the posterior tibial cortex landmark in the injured knee and 25.9 ± 3.1 mm in the healthy knee (Fig. 4). The posterior femoral contact position appeared more pronounced in the lateral than the medial compartment of the knee, but this was not significant (Repeated measures ANOVA for interaction between angle of knee flexion, lateral and medial compartment and healthy or injured knee $F_{(1,152)} = 1.3$, $p = 0.25$.) To achieve 80% power a sample size of 144 subjects would have been required, due to the wide variation between individuals. The femoral contact point was on average 1.3 mm more posterior than the healthy knee in the lateral compartment of the loaded knee, and 0.5 mm more posterior in the medial compartment of the loaded knee (Fig. 4). However, individuals exhibited as much as 9.7 mm of posterior femoral contact displacement in the medial compartment and 9.5 mm in the lateral compartment.

In the healthy knee the medial and lateral tibiofemoral contact patterns are distinctly different, reflecting the longitudinal rotation of the knee that occurs through

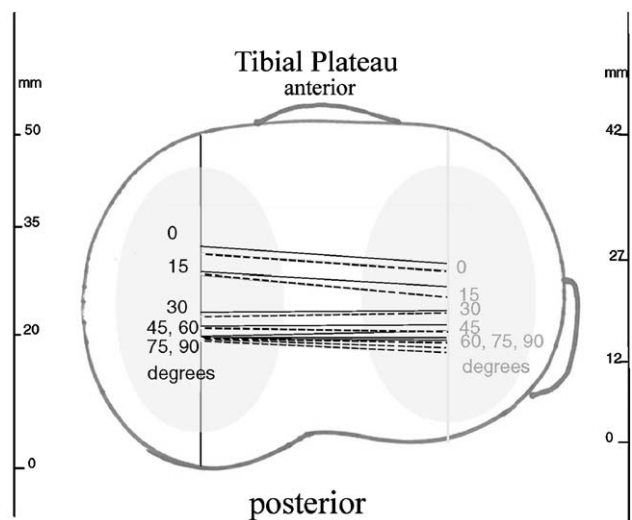


Fig. 3. Diagram represents the tibiofemoral contact pattern in the normal knees (solid line) and ACL injured knees (dotted line).

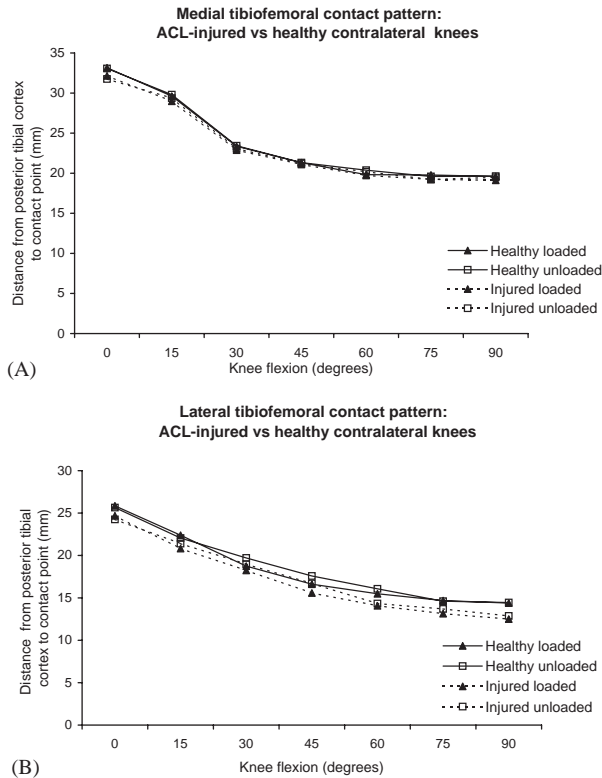


Fig. 4. Tibiofemoral contact patterns of the medial (A) and lateral (B) compartments of the knee, in the healthy and injured knees, demonstrating the slightly posterior position of the tibiofemoral contact point on the tibia plateau in the ACL injured knee, but preservation of the profile of the curve.

flexion. The healthy mediolateral asymmetry of the contact point pattern was preserved in the injured knees (Repeated measures ANOVA used to test medial and lateral patterns (between subjects): $F_{(1,152)} = 310$, $p < 0.001$). The ACL injured knee rotated about the longitudinal axis throughout flexion from 0 to 90°.

Loading the injured and healthy knees did not alter the TF contact pattern ($F_{(1,152)} = 0.557$, $p = 0.46$). There was no difference in the contact pattern between subjects when pressing down through the weighted footplate, and when unloaded.

The difference in TF contact pattern due to injury was moderately correlated with the Cincinnati knee score. The scores for the ACL injured subjects ranged from 35 to 76/100, with a mean of 57 ± 10.8 , which is defined as fair/good. The difference in TF contact pattern due to injury for each subject was measured as the mean of the side-to-side difference in the contact points, at each knee flexion angle. The Cincinnati knee scores were correlated with the mean of the side-to-side difference in the contact points in the unloaded knee (Pearson's $r = 0.40$, $p = 0.07$), but not in the loaded knees ($r = 0.25$, $p = 0.28$). The reported knee symptoms taken in isolation (scored out of 20) also correlated with the mean of the side-to-side difference ($r = 0.37$, $p = 0.10$).

Thus, there is a weak association between poorer knee scores and greater difference in contact pattern in the injured knee.

Damage to the articular cartilage and menisci were not correlated with a greater difference in the TF contact pattern of the knee. The subjects with meniscal damage did not have a significantly different mean side-to-side difference in contact points ($F_{(1,19)} = 2.48$, $p = 0.133$), nor did the subjects with chondral damage ($F_{(1,19)} = 3.69$, $p = 0.71$).

Increased passive anterior laxity of the knee did not correlate with a greater difference in contact pattern. Side-to-side difference at manual maximum anterior drawer, quantified by the KT 1000 measure did not correlate with side-to-side difference in the contact pattern ($r = 0.12$, $p = 0.60$). Neither passive instability nor damage to the knee joint was related to the active instability demonstrated on MRI.

5. Discussion

The TF contact pattern of the ACL injured knees was significantly different to the healthy contralateral knees. The pilot study by Smith et al. (1999), and the study of 12 healthy control subjects reported here, have shown that while there is wide variation between subjects, within subjects there is high right to left consistency. Therefore, the contralateral knee can reliably be used as a control for TF contact pattern analysis. The difference in contact pattern between the ACL injured knee and the healthy contralateral knee can be confidently attributed to the pathology.

The posterior tibiofemoral contact pattern due to ACL injury has been reported by other authors. Dennis et al. (1996) used videotaped fluoroscopic images to examine the TF contact patterns in the ACL deficient knee in standing deep knee flexion. They describe the ACL injured subjects moving from posterior tibiofemoral contact positions in knee extension, to variations in normal and posterior contact positions through flexion. The average difference in contact position between the injured and intact knee was only 1.6 mm, but the difference ranged from 0.5 to 13.7 mm in some knees. This study used two-dimensional imaging at low resolution, so was unable to draw out information regarding rotation.

The shift in the axis of longitudinal rotation of the knee due to ACL deficiency could not be confirmed in the current study, due to the wide variations in the kinematics of individuals. It did appear, however, that the effect of the ACL injury was to move the tibiofemoral contact pattern more posteriorly in the lateral compartment than the medial compartment of the knee. This would indicate that the axis of longitudinal rotation of the knee had shifted medially. It was

observed by Brandsson (Brandsson et al., 2001) using radiostereometry that while there was no difference in anteroposterior displacement of the medial compartment, there was a difference in the lateral compartment of the injured knees. The reference points for the tantalum beads used in Brandsson's analysis were established for the injured knee in neutral supine extension, rather than using the direct bony landmarks used in our MRI study, so parallel analysis is difficult. However, the medial/lateral characteristics of the injured knee motion are similar. The role of the ACL in the control of longitudinal rotation of the knee is recognised (Anderson and Dyhre-Poulsen, 1997; Benvenuti et al., 1999; Buckland-Wright et al., 1994; Grood et al., 1988; Woo et al., 2002), so it is reasonable to suspect that having lost the primary restraint of the ACL, the secondary restraint of the MCL may have an increased role in control knee rotation, from the medial side of the knee.

The mean side-to-side difference in tibiofemoral contact was 1.3 ± 0.3 mm, which is similar to that reported by Brandsson, using radiostereometry. The anterior tibial position recorded using plain radiography in patients performing a standing lunge was mean 12 mm (Friden et al., 1993). Vergis using electrogoniometry to analyse anterior tibial shift in a step up activity describes a 5 mm anterior tibial shift (Vergis and Gillquist, 1998; Vergis et al., 1997). Fleming compared KT 1000, plain radiography and radiostereometry to measure passive anterior displacement of the knee (Fleming et al., 2002). Plain radiography and KT 1000 arthrometry being two dimensional are not able to account for longitudinal rotation of the knee, and describe anterior tibial shift as a two dimensional feature of the ACL deficient knee. Radiostereometry and MRI techniques are able to isolate rotation from anterior translation and hence record side-to-side difference in displacement of similar magnitude.

In our study, loading the knees did not change the longitudinal rotation. Hill et al. (2000a), using open field MRI, saw suppression of rotation in subjects performing a standing squat. However, there are several biomechanical differences between a supine leg press and a standing weight-bearing activity (Dennis et al., 1996; Friden et al., 1993; Hill et al., 2000a). Subjects in supine experience different proprioceptive feedback to standing subjects. Joint compression in standing is greater, and joint contact occurs over a wider area. Our subjects were pressing down against a 150 N weight, substantially less than the body weight supported in a standing lunge, although we found no difference when the weight was increased to 250 N. This protocol uses the available closed tunnel MRI unit, and weights sustainable by the injured population. Thus, comparison of findings between a supine leg press and standing lunge may be inappropriate.

In the healthy knees the pattern of tibiofemoral contact is different from the femoral condylar motion reported by Todo et al. (1999) and Hill et al. (2000b). They reported that while the lateral femoral condyle continued to roll posteriorly throughout flexion, the medial femoral condyle remained central above the medial tibial plateau from 15° to 90° . However, they found that the femoral condylar motion could be altered by imposing either internal or external rotation. In our study of healthy knees the medial femoral condyle continued to roll back on the tibial plateau in flexion, though to a lesser extent than the lateral condyle. The knee is capable of a wide variety of kinematic behaviours depending on demands, loads, and restraints of a particular movement. Blankevoort et al. (1988) described this as the envelope of passive motion of the knee, within which a variety of active movement patterns were available. The supine leg press used in this study is different in its loads and constraints to the activities performed by the subjects in the studies of Todo et al. (1999) and Hill et al. (2000b). The resultant TF contact pattern is therefore also different.

Closed chain resisted exercises have been advocated for rehabilitation after ACL injury and reconstruction (McGinty, 2000; Toutoungi, 2000), in order to minimise stress on the ACL by decreasing tibiofemoral shear forces and increasing muscular co-contraction (Beynon et al., 1995; MacWilliams et al., 1999). The supine leg press exercise is therefore one that has been commonly prescribed, as the foot is fixed, and the axial force in line with the tibia. This study showed that the tibiofemoral contact pattern of the ACL injured knee was different from the healthy contralateral knee performing a supine leg press. In effect, that to press down through the feet did not normalise the TF contact pattern. Testing of the ACL strain in vivo of subjects performing a leg press is necessary to clarify the relative "safety" of this exercise.

It has been suggested that increasing knee symptoms correlate with instability during activity, regardless of passive anterior laxity (Friden et al., 1993). Friden suggested subjects with less displacement had learnt to actively control the instability of the knee, and therefore experienced fewer symptoms. Our study has also shown a relationship between symptoms and active instability, reflected by the TF contact pattern, but no relationship between passive anterior laxity and difference in the TF contact pattern.

The TF contact appears to occur over a more posterior region of the tibial plateau in the ACL injured knee, which may be implicated in the degeneration of the articular cartilage of the knee. While it is difficult to isolate damage to the knee joint caused by repeat episodes of trauma over time, from damage caused by aberrant kinematic behaviour, there is an increasing body of evidence that aberrant knee kinematics do contribute to articular cartilage damage. In a study of

541 ACL injured subjects, Myers et al. (2001) found that articular cartilage damage seen at arthroscopy was correlated with time since injury. Harman also correlated absence of a functional ACL with articular cartilage wear over a more posterior area of the tibial plateau (Harman et al., 1998). Damage to the menisci is also known to contribute to the development of osteoarthritis (Fairbank, 1948). The menisci act as a secondary restraint to anterior tibial translation in the absence of the ACL and suffer from chronic loading and repeat trauma in the ACL deficient knee (Allen et al., 2001; Bellabarba et al., 1997; Finsterbush et al., 1990; Irvine and Glasgow, 1992; Keene et al., 1993). The damaged menisci are less able to protect the articular cartilage of the knee by improving load distribution. In this manner, the aberrant kinematic behaviour of the knee is responsible for the degeneration of the menisci, and the consequential osteoarthritis. Our study shows that there is a change in the TF contact pattern resulting from ACL injury, which is likely to be a contributor to accelerated wear over the long term (Bellabarba et al., 1997; Clatworthy and Amendola, 1999; Gillquist and Messner, 1999).

The characteristics of abnormal motion in the ACL injured knee are firstly, that the tibiofemoral contact in the ACL injured knee occurs over a posterior area on the tibial plateau. Secondly, that the longitudinal rotation evident in the healthy knee does occur in the ACL injured knee, however, the axis of rotation may have altered. The difference in the tibiofemoral contact pattern in the ACL injured knee during a closed-chain leg press was associated with more severe knee symptoms, irrespective of the passive anterior laxity of the knee.

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