Manuscript Number:

Title: Clinical biomechanics of instability related to total knee arthroplasty

Article Type: Review Article

Keywords: total knee arthroplasty (TKA), instability, soft tissue, primary and secondary restraints, knee biomechanics

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Abstract: Background: Tibiofemoral instability is a common reason for total knee arthroplasty failure, and may be attributed to soft tissue deficiency and incorrect ligament balancing. There are many different designs of implant with varying levels of constraint to overcome this instability; however there is little advice for surgeons to assess which is suitable for a specific patient, and soft tissue balance testing during arthroplasty is very subjective.

Method: The current theories on primary and secondary soft tissue restraints to anterior/ posterior, varus/ valgus, and internal/ external rotational motion of the knee are discussed. The paper reviews biomechanics literature to evaluate instability in the intact and implanted knee.

Findings: The paper highlights important intra- and extra-capsular structures in the knee and describes the techniques used by clinicians to assess instability perioperatively. In vitro cadaveric studies were found to be a very useful tool in comparing different implants and contributions of different soft tissues.

Interpretation: In vitro cadaveric studies can be utilised in helping less experienced surgeons with soft tissue releases and determining the correct implant. For this to happen, more biomechanical studies must be done to show the impact of release sequences on implanted cadavers, as well as determining if increasingly constrained implants restore the stability of the knee to pre-deficient conditions.

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24th July 2013

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Dear Professor Kim Burton and colleagues,

Re: Manuscript entitled 'Clinical biomechanics of instability related to total knee arthroplasty'

I am pleased to be submitting this literature review to Clinical Biomechanics. I hope that you find the manuscript an interesting and strong addition to the journal and the field of biomechanics.

There is no duplicate publication elsewhere of any part of the work and no related paper previously published in Clinical Biomechanics. Imperial College London has received money from Stryker Corporation to fund this project. The typescript has been read and agreed by all authors, and the corresponding author is Andrew A Amis (Department of Mechanical Engineering, Imperial College London, Exhibition Road, London, SW7 2AZ, a.amis@imperial.ac.uk).

I look forward to receiving comments from the reviewers.

Yours sincerely,

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Imperial College London has received money from Stryker (Europe) Corporation to support Kiron Athwal to work on a research project related to this article.

“Clinical biomechanics of instability related to total knee
arthroplasty”

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Word count: 210 (Abstract)
4,639 (Main text excluding reference list and abstract)
Number of Tables: 7
Number of Figures: 2

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Interpretation: In vitro cadaveric studies can be utilised in helping less experienced surgeons with soft tissue releases and determining the correct implant. For this to happen, more biomechanical studies must be done to show the impact of release sequences on implanted cadavers, as well as determining if increasingly constrained implants restore the stability of the knee to pre-deficient conditions.

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1. Introduction

The use of total knee arthroplasty (TKA) to combat the effects of osteoarthritis has become standard practice for many years. From a survey of 18 different countries, it has been estimated that annually there are 175 total knee procedures for every 100,000 people in the population (Kurtz et al., 2011).

Yet despite being a common procedure, failures of the TKAs are possible, and revision surgery to a more constrained design inevitably presents additional health and emotional issues for the patients as well as financial implications (Sharkey et al., 2002). A major reason for failure is instability, defined as excessive and unnatural movement of the implant components (Rodriguez-Merchan, 2011) which may occur within weeks, months or even many years after the initial surgery.

Sharkey et al. (2002) performed a retrospective review over a three year period at one institution, and found that instability was a major reason for surgery in 21.2% of early stage revisions (occurring less than two years after primary arthroplasty) and 22.2% in late stage revisions. A similar situation was noted in a multicentre prospective cohort study by Mulhall et al. (2006), who found that 28.9% of patients who required revisions suffered from instability.

Instability may be a result of initial and progressive soft tissue deficiency, inadequate soft tissue and gap balancing during surgery, component misalignment, and inappropriate implant restraint, size and design (Mulhall et al., 2006; Sharkey et al., 2002; Vince et al., 2006; Yercan et al., 2005). To prevent instability in TKAs, improvements in surgical technique and TKA design can be enacted with knowledge of how soft tissue deficiency affects the stability after implantation. This review sets out to discuss how laxity/ instability of a TKA-implanted knee joint can be measured, evaluate different methods of experimentation, and present the current ideas of ligamentous and soft tissue restraint to major planes of knee motion.

2. TKA designs

Condylar total knee designs in their current recognisable form have been developed since the 1970s (Robinson, 2005): a metal femoral prosthesis; a metal tibial tray with a proximal polyethylene articulating surface; and occasionally a polyethylene patellar component.

There are a wide variety of TKA designs available, varying in degrees of constraint, bone loss and soft tissue resection. For uncomplicated primary knee replacements the most commonly used types are the cruciate-retaining (CR) and posterior-stabilised (PS) designs. A CR TKA requires resection of the anterior cruciate ligament (ACL) but retains the posterior cruciate ligament (PCL). This type of TKA usually derives stability by having concave articular surfaces on both medial and lateral tibial condyles, which act to locate the femoral condyles under the influence of axial joint compression. Both the conformity of the articulation, and the tensions in the surrounding ligaments, reduce knee laxity (Ishii et al, 2011). The depth and slope of the concavity of the tibial bearing contribute to the inherent stability of the prosthesis, and that may be characterised by force versus displacement testing of the prosthesis while subjected to axial compressive loading (Haider and Walker 2005; ASTM standard F1223, 2008). Increased soft-tissue tension may reduce tibiofemoral laxity, but excessive tension is undesirable; in the CR TKA, for example, the unbalanced tension in the PCL causes tibial anterior subluxation, so that the femoral component bears onto the posterior edge of the tibial articular surface (Heesterbeek et al 2010).

A posterior-stabilised (PS) design removes both cruciate ligaments and instead utilises a post-box-cam mechanism to prevent non-physiological anterior movement of the femur with respect to the tibia when flexed (Fantozzi et al., 2006; Walker et al., 2009). An argument for the implantation of a PS over a CR design is that collateral ligament balancing is more easily achieved than with a CR design (Freeman and Railton, 1988). The post-box-cam mechanism of a PS-implanted knee drives femoral posterior roll-back in knee flexion, which delays posterior impingement and thus leads to greater knee flexion (Jacobs et al., 2005).

The fit of the tibial post into the 'box' between the femoral condyles also limits tibial internal-external rotation.

Less-common designs retain both cruciate ligaments in an attempt to retain knee kinematics which are as close to physiological behaviour as possible (Cloutier et al., 1999). It is unusual for a TKA to incorporate ACL retention, despite the importance of the ACL for stability of the natural knee. This situation arose because, in the era when the TKA procedure was being developed, it was reserved for those with chronic, severe arthritis, and so the ACL was usually incompetent in the presence of degenerative changes such as impinging osteophytes.

Other variations of TKA include mobile-bearing designs where the polyethylene insert can rotate and slide freely on the tibial tray (Most et al., 2003a). More recently, designs have incorporated asymmetrical femoral condyles. These designs have highly stable medial condylar articulations and lateral articulations which allow for more anterior/ posterior freedom, which is believed to replicate more anatomically-correct knee kinematics (Amin et al., 2008; Walker et al., 2010).

If a TKA fails and requires revision, or the patient has multiple ligament or bone deficiencies before even a primary operation (Yang et al., 2012), more constrained condylar knee designs may be implanted. These usually include longer intramedullary stems and larger, more squared tibial posts than a PS design. Further restraint against global instability may be introduced with rotating-hinged designs (Yang et al., 2012), in which the tibial and femoral components are linked together.

3. Primary and secondary ligamentous and soft tissue restraints

The complex network of ligaments and soft tissue surrounding the knee and within the capsular structure can be classified into primary and secondary stabilisers. A primary restraint can be seen to be the main passive restraint to motion in a specific degree of freedom (DOF) (Noyes et al., 1980), with secondary restraints that resist the motion to a

lesser degree. However, the secondary restraints may become a major stabiliser in the cases when primary restraints are deficient or require resection, for example in many arthroplasty designs. Therefore, understanding how the ligaments and soft tissues interact in the different planes of motion is beneficial for any investigations into TKA instability.

Table 1 lists various papers that investigated ligamentous and soft tissue restraints on intact knees using a variety of in vitro and in vivo methods. There has been less research, however, into the soft tissue restraints post-TKA (Table 2).

3.1 Anterior Translation

It has been well established that the primary restraint to anterior translation of the tibia relative to the femur is the ACL, with Butler et al. (1980) reporting an average 86% of the total resisting force against anterior drawer was provided by it (Fig. 1). The ACL is nearly always resected in TKA implantation, and so designs must incorporate more conforming articulating surfaces to prevent excessive anterior slide of the tibia.

Butler et al. (1980) and Sullivan et al. (1984) described the medial collateral ligament (MCL) as a significant secondary restraint to anterior drawer, a finding supported by Sakane et al. (1999), who reported that the MCL contributed around 60% of the total restraint the ACL carried at 90° flexion. Additionally, other studies highlighted the role of the iliotibial band (ITB) as an 'ACL agonist' (Yamamoto et al., 2006) and, provided the ACL is resected first, the secondary restraint from the medial meniscus (MM) (Allen et al., 2000; Levy et al., 1982). The lateral meniscus (LM) was found not to be a significant restraint (Levy et al., 1989).

3.2 Posterior Translation

The PCL is the primary restraint to posterior translation of the tibia (Fig. 2), offering on average 95% of the total resisting force in the flexed knee (Butler et al., 1980); Race and Amis (1996) showed that this contribution fell as the knee extended, leaving the posterolateral structures (PLS) to resist posterior translation near full extension. Other

authors agreed that the PLS comprising of structures such as the popliteus tendon (Pop T) and the popliteofibular ligament (PFL) act as secondary restraints to tibial posterior translation (Butler et al., 1980; Gollehon et al., 1987).

Whilst the PCL is retained in CR TKAs, a PS TKA resects the PCL and instead utilises a vertical post on the tibial plateau, which engages with a femoral box in flexion, and prevents the tibia from sliding posteriorly relative to the femur (Fantomzi et al., 2006). Some instability may result near knee extension (the weight-bearing posture) if the post-box mechanism only engages in deeper knee flexion which is typically around 50° flexion.

On the medial side of the knee, Robinson et al. (2006) observed the posteromedial capsule (PMC) being well aligned to resist posterior translation at full extension. This was supported by Petersen et al. (2008), who also defined a posterior oblique ligament (POL) between the MCL and PMC as producing significant restraint at all angles of flexion between 0-90° flexion (it is debated whether such a distinct band exists (Amis et al., 2003)). Additionally, Gupte et al. (2003) found the ligaments connecting the LM to the posterior aspect of the femur (the meniscomfemoral ligaments of Humphry and Wrisberg) to be secondary restraints to posterior drawer, contributing 28% of the restraint at 90° flexion; they are resected during TKA.

3.3 Valgus Rotation

The superficial medial collateral ligament (sMCL) is the primary restraint to tibial abduction, which manifests as medial opening of the knee (Fig. 3) (Grood et al., 1981). Robinson et al. (2006) discovered that the deep medial collateral ligament (dMCL), whilst not a significant restraint in an intact knee, contributed appreciably in an sMCL-deficient knee over the range 15-90° of flexion, and thus can be described as a secondary restraint. The same study found that the PMC offered 32% of the total restraint to valgus rotation at full extension, however this reduced noticeably in flexion.

The PCL is considered to be only a secondary restraint (Grood et al., 1981), as it can only generate a small moment arm about the centre of valgus rotation compared with the sMCL (Amis et al., 2003).

3.4 Varus Rotation

The lateral collateral ligament (LCL) is the primary restraint to tibial adduction, which causes lateral opening of the knee (Fig. 4) (Gollehon et al., 1987; Grood et al., 1981). It provides 69% of the total restraint to varus rotation at 25° flexion (Grood et al., 1981), although it has been shown that as flexion increases, the LCL becomes more slack and less well-aligned to restrain varus opening (Sugita and Amis, 2001). The PLS and ACL have also been described as secondary restraints, with the PLS only at low flexion angles and the ACL suffering from only generating a small moment arm about the centre of varus rotation (Grood et al., 1981).

3.5 Internal Rotation

With a 5Nm internal torque applied, Robinson et al. (2006) found increased internal rotation of 6-8° between 30-90° flexion after cutting the sMCL, and thus contended that it provided the primary restraint in flexion (Fig. 5). The same study also concluded that the PMC was a primary restraint only when the knee is in an extended position, as it slackens with flexion. Older studies have described the ACL as a secondary restraint to internal rotation, with results showing only a significant increase in internal rotational laxity experienced in cutting posterior structures if the ACL is sectioned first (Gollehon et al., 1987; Lipke et al., 1981).

3.6 External Rotation

The PLS has been acknowledged as the primary restraint to external rotation (Fig. 6) (Grood et al., 1988). The LCL has also been identified as a major stabiliser albeit only when tense at lower flexion angles (Gollehon et al., 1987), whereas the PFL in particular does not slacken with flexion (Sugita and Amis, 2001). Veltri et al. (1996) and more recently Lim et al. (2012)

found the PFL alone can be considered a primary restraint at all angles of flexion, especially compared with the LCL at 60-90° flexion. This was disputed by Pasque et al. (2003) however, who believe that the PFL must be considered as part of the PLS, and does not offer significant restraint alone.

Grood et al. (1988) signified the PCL as a restraint to tibial external rotation at 90° knee flexion, but only having an effect after the PLS has been sectioned. Robinson et al. (2006) found that in response to an applied 5Nm external torque, transecting the sMCL also increased laxity by 3° and 7° at 0° and 90° of flexion respectively, with the dMCL also demonstrating restraint at higher angles of flexion.

4. Evaluating instability of the knee and the contribution of soft tissues

The definition of instability varies between studies, which can be attributed partly to different study backgrounds. For example a clinically-orientated approach may define instability subjectively as the patient's complaint of the knee 'giving-way', or more objectively from measured increases in tibiofemoral joint laxity when a normal knee is modified by injury or arthroplasty (Stoddard et al., 2013). An engineer, however, may repeat the same path of motion after modification and record the decrease in required displacing force as an objective measure of stability (Rudy et al., 1996).

The methods employed to assess soft tissue instability in clinical knee assessment, surgical judgement during an arthroplasty procedure, and in-vitro cadaver testing, are described below.

4.1 Clinical knee assessment

Clinical assessment of knee laxity is essential both prior to surgery, to indicate the type of implant required, and post-surgery (Ishii et al., 2005), to assess whether satisfactory mechanical stability has been achieved. For example, if a patient has a suspected ACL rupture, a clinician may perform either an anterior drawer test, Lachman's test, or a pivot

shift test (Bach et al., 1988; Kim and Kim, 1995). The anterior drawer and Lachman's tests impose an anterior translation force on the tibia and the resulting laxity is measured (usually by comparison to the contralateral knee), while the pivot-shift is a more 'dynamic' test which moves the knee in an attempt to elucidate instability.

In an attempt to reduce subjective variation between clinicians performing the assessments, arthrometers were devised for a more quantitative assessment of knee ligament laxity. The KT-1000™, and later the KT-2000™ instrument (MEDmetric®, San Diego, CA, USA), allow the clinician to apply a known force to the patient's tibia, and measure the displacement relative to the fixed femur (Benoit et al., 2006). Initially designed to find ACL or PCL laxity in the intact knee, the KT-2000™ has been subsequently utilised in research comparing the anterior-posterior laxity in PCL-retaining and PCL-substituting TKAs (Ishii et al., 2005).

Other arthrometers include the Telos™ stress device (Metax GmbH, Hungen/ Obbornhafer, Germany), which can be manipulated to test different planes of laxity, including varus/ valgus stress tests (Okazaki et al., 2006; Takeda et al., 2012). However, the Telos device is a means to apply known loads to the knee; the laxity must be measured from pairs of radiographs, taken when the knee was loaded and unloaded.

There are noticeable failings with knee assessment tests. Arthrometers have standardised the direction and magnitude of force applied as well as the measurable displacements, and thus are a less subjective procedure than the original assessment tests. However, like the original tests, there is no obvious way of discovering which ligaments are being scrutinised under the arthrometer loads (Noyes et al., 1980). For example, increased anterior laxity of a knee may not be purely due to an ACL rupture, but could be a result from deficiency of other stabilising structures. There are disagreements between various studies on the repeatability of results of different arthrometers (Cannon, 2002; Forster et al., 1989; Steiner et al., 1990). Furthermore, the forces applied to the knee are much smaller than in normal activity. The circumstances are understandable, as a clinician cannot ethically apply forces that may

cause further damage to the patient's knee, however it means that a knee with a false negative result for ligament deficiency with an assessment test may still experience instability under normal, higher force activities (Noyes et al., 1980).

There are other clinical methods of measuring movement of knee joints of patients. Gait analysis can utilise motion capture to allow clinicians to view how post-TKA surgery has affected the overall kinematics of the joint. However, problems such as skin movement artefacts mean the motion tracking cannot accurately provide detailed laxity information (Benoit et al., 2006). Instead, video fluoroscopy avoids skin motion effects by capturing series of x-ray images that can be used to reconstruct 3-D kinematics of implants during movements such as rising from a chair, and treadmill running (Fantozzi et al., 2006; Zhao et al., 2007). With the invention of moveable video fluoroscopy systems to increase the viewing field (Zihlmann et al., 2006), more detailed motion trials can be recorded. This could be used in detailing mid-range instability (defined differently by different authors, but generally considered to be at flexion positions up to 90° (Parratte and Pagnano, 2008; Stoddard et al., 2013; Vince et al., 2006; Yercan et al., 2005)) experienced by many TKA patients when stepping downstairs (Stoddard et al., 2013), for example.

4.2 Surgical judgement during arthroplasty

The stability of a TKA is heavily dependent on not only the conforming design of the components, but also on the surgical techniques used to implant the TKA in the patient (Yercan et al., 2005). Therefore, particular protocols are followed during surgery to balance the ligaments (in particular the collaterals) and align the TKA components (Walker et al., 2010).

Generally, if the medial side of the knee is tight, some medial structures such as the MCL, PMC, and pes anserinus will be cut at the tibial insertion to allow correction of the varus deformity (Mihalko et al., 2003; Whiteside, 2002). In laterally tight knees, structures such as the LCL, ITB, PLS and Pop T will be cut at the femoral insertion to allow correction of the

valgus deformity (Kanamiya et al., 2002; Whiteside, 2002). Another release technique named pie crusting can also be utilised on the ligaments, involving multiple stabs of a scalpel on the taut region of the structures, which allows the ligament to be stretched to the desired length without detaching it from the bone (Mihalko and Krackow, 2000).

Many articles have noted issues with attempting to address soft tissue tightness during a TKA procedure. Whiteside (2002) admitted that although medial and lateral releases may help varus/ valgus stability, rotational and anteroposterior laxity may be introduced. Ghosh et al. (2012) measured the length-change patterns of the MCL and LCL before and after CR TKA. The study found that, despite a gap balancing technique being used to balance the knee, the collateral ligaments slackened more than in the natural knee after CR TKA when the knee was flexed.

It can be suggested that the testing of soft tissue balance is too dependent on the experience of the surgeon. Mihalko et al. (2003) describe two different techniques to test ligament balancing: distraction testing and trial components. Distraction of the joint with laminar spreaders or tensors allows the surgeon to assess the flexion and extension gaps with spacer blocks in place. Alternatively, trial components may be fitted into the joint space, and varus/ valgus stress tests can assess medial and lateral soft tissue balance.

The use of spacer blocks to determine equal joint spacing in flexion and extension by sight is very subjective. Performing a varus/ valgus stress test at 0° and 90° flexion may give a 'feeling' of laxity at these angles, but does not address the balance at angles between these limits (D'Lima et al., 2011). This is problematic, as an implanted TKA may experience "mid-range instability" if the soft tissue is not tense within 0-90° flexion (Stoddard et al., 2013; Yercan et al., 2005).

Vince et al. (2006) argued that trying to prevent mid-range instability by balancing a knee at mid-flexion would cause the knee to be too tight to allow for full extension. Perhaps, therefore, revising to a more conforming TKA implant offering more varus/ valgus restraint

would be suitable in cases where the patient suffers from mid-range instability. However since a surgeon largely bases varus/ valgus stability on ‘feel’, the authors suggest that a more quantitative approach to determining laxity would be useful in helping less-experienced surgeons decide the correct implant.

4.3 In vitro cadaver testing

In-vivo testing on subjects has advantages such as being able to perform normal activity, but it is heavily constrained by ethical and practical issues (Kessler et al., 2009). For example, it would be highly unfeasible to subject a single patient to multiple surgeries for different implant designs comparisons, or to perform activities that could risk soft tissue damage. Working in-vitro on cadavers resolves such problems. It is possible to compare different implant designs, positioning or surgical techniques on the same specimen, minimising inter-subject variation. Although accurate anatomic replication of active restraints such as muscle forces requires further investigation, there is more control on passive soft tissue restraint than with in-vivo testing.

Noyes, Grood and Butler helped pioneer laxity testing to determine the passive restraints from the primary knee ligaments (Butler et al., 1980; Grood et al., 1981; Noyes et al., 1980). By imposing known displacements/rotations to the tibia at fixed flexion angles and measuring the reduction of displacing force required by cutting ligaments around the knee, the percentage contribution of various ligaments to resisting that movement can be calculated (Race and Amis, 1996).

The limits of laxity across the arc of knee flexion-extension can be described as the envelope of passive knee motion (Blankevoort et al., 1988). During the natural arc of flexion/extension, the tibia is free to rotate or displace in the other five DoF, leading to the passive path of motion. Thus, by applying forces and moments to the tibia along this arc to a limit, beyond which damage to the specimen may occur (Blankevoort et al., 1988), an envelope of laxity along the arc can be determined (Bull et al., 2008).

The introduction of robotic technology to simulate and record more complex loading conditions on knee joints (Fujie et al., 1993) has allowed studies into the “in-situ” force (Fujie et al., 1995) experienced by ligaments during predefined movements.

5. Ligamentous laxity and/or deficiency and its influence on implant constraint

As a general guide, surgeons are recommended to choose implants with as minimal amount of constraint as possible to achieve stability (Naudie and Rorabeck, 2004). Any increase in the constraint in the prosthesis also increases its capability to transmit loads to the fixation; it is preferable for the loads to be transmitted by the soft-tissue stabilisers (Fig. 7). A corollary is that, because constraint allows greater loads to be transmitted, the fixation has to become more massive to prevent loosening, and that is inevitably more destructive during insertion and leaves fewer options if revision is needed.

On that basis, the most common implants used are CR and PS TKAs, with gap balancing and releases if required to restore soft tissue symmetry. However, if there is still persistent laxity, more constraint will be required (McAuley and Engh, 2003). In the case of revisions when a primary TKA needs to be recovered after failure, the soft tissue envelope around the knee may be disrupted or the associated bone loss may mean the attachment sites of the MCL and LCL are lost (Sculco, 2006).

More-constrained designs, whilst offering more global stability than CR and PS TKAs, rely less on soft tissue contributions and thus increase the stresses on the implant-bone interface and increase risk of early loosening (Nelson et al., 2003; Fig. 7). If these more-constrained devices fail, their revision is a serious task; therefore it is important that surgeons receive guidance about the limits of use of less-constrained devices so that they do not need to resort unnecessarily to the more-invasive procedures when in doubt about the ability of the lesser device to maintain knee stability in the face of specific soft tissue deficiencies. However it is difficult to judge how much soft tissue deficiency/laxity necessitates this extra constraint (Morgan et al., 2005), and there is little published data which might guide this.

Girard et al. (2009) performed a retrospective analysis on TKAs in valgus knees, and identified, following the use of tensioning devices and spacer blocks, a threshold of 5° residual valgus laxity in extension and 3mm in tibiofemoral gap difference between flexion and extension, which if exceeded were indications for the use of more-constrained condylar implants rather than a normal PS TKA.

Sculco (2006) noted that the process of determining level of constraint is subjective, but recommends that, if after spacer blocks or laminar spreaders have attempted to introduce soft tissue symmetry, laxities of 7-10mm under stress tests suggest a more-constrained implant is required. Sculco (2006) further advises on the choices between the constrained designs, believing that constrained condylar implants are suitable if the MCL and/or LCL are present, but remain lax even after attempted balancing. However, if one of those ligaments is either deficient or missing, a rotating hinged implant would be required to restore stability.

Gustke (2005) reported a more specific plan for implant choice based upon preoperative ligament function, confirmed through physical varus/valgus stress examinations and radiographs. An LCL lax knee may suffice with a constrained condylar design, as well as an MCL lax knee if the ligament can be reconstructed. However if the MCL is absent, or both MCL and LCL are insufficient, then only a rotating hinge implant gives acceptable stability. What is universally agreed, is that a rotating hinge design should only be considered as a last resort if all other less constrained implants cannot provide sufficient stability.

6. Conclusion

This review discussed the different methods of evaluating instability in an intact and implanted knee, as well as present current ideas of primary and secondary passive restraint to anterior/ posterior drawer, internal/ external rotation and varus/ valgus rotation. Clinical tests can be performed quickly on patients to determine the laxity of the knee approximately, but cannot accurately define the health of a specific ligament. In vitro testing can investigate specific ligaments in turn for a more detailed description of the passive restraint of the knee,

although instability due to active restraints such as muscle forces is difficult to replicate. More research is required on how the ligaments are affected by implantation of a TKA, to decide which implant is suitable for the patient.

It is imperative to have greater understanding of whether the standard soft tissue releases during arthroplasty actually improve the kinematics of the TKA to an idealised normal intact state, or whether some actually further introduce instability during various angles of knee flexion/extension. For example, with a medially tight knee, releasing medial structures such as the sMCL may correct the varus deformity (Whiteside, 2002) and, after choosing the correct TKA component thickness to tense the collateral ligaments, improve the varus-valgus restraint that the LCL and MCL can now give, becoming more tensed while the limb is the the corrected alignment. However if the sMCL is considered a secondary restraint to anterior drawer (Butler et al., 1980) and a primary restraint to internal rotation (Robinson et al., 2006), is the stability in these planes of motion then adversely affected by the release?

The authors of this study suggest that more research is required on the biomechanical variations caused by soft tissue deficiency as well as on the controlled releases performed by surgeons, so that a more quantifiable assessment can be made by less experienced surgeons on the choice of TKA implant for the specific patient.

Acknowledgments

We would like to acknowledge Stryker Corporation and the National Health Service (NHS) for funding.

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426 **References**

427 Allen, C.R., Wong, E.K., Livesay, G.A., Sakane, M., Fu, F.H., Woo, S.L.Y., 2000. Importance
428 of the medial meniscus in the anterior cruciate ligament-deficient knee. J. Orthop. Res. 18,
429 109-115.
430 Amin, A., Al-Taiar, A., Sanghrajka, A.P., Kang, N., Scott, G., 2008. The early radiological
431 follow-up of a medial rotational design of total knee arthroplasty. Knee 15, 222-226.
432 Amis, A.A., Bull, A.M.J., Gupte, C.M., Hijazi, I., Race, A., Robinson, J.R., 2003.
433 Biomechanics of the PCL and related structures: posterolateral, posteromedial and
434 meniscofemoral ligaments. Knee Surg. Sports Traumatol. Arthrosc. 11, 271-281.
435 ASTM F1223, 2008, "Standard Test Method for Determination of Total Knee Replacement
436 Constraint", ASTM International, West Conshohocken, PA, 2008, DOI: 10.1520/F1223-08,
437 www.astm.org.
438 Bach, B.R., Warren, R.F., Wickiewicz, T.L., 1988. The pivot shift phenomenon - results and
439 description of a modified clinical-test for anterior cruciate ligament insufficiency. Am. J.
440 Sports Med. 16, 571-576.

- Barink, M., van Kampen, A., Malefijt, M.D., Verdonchot, N., 2005. A three-dimensional dynamic finite element model of the prosthetic knee joint: simulation of joint laxity and kinematics. *Proc. Inst. Mech. Eng. Part H-J. Eng. Med.* 219, 415-424.
- Battaglia, M.J., Lenhoff, M.W., Ehteshami, J.R., Lyman, S., Provencher, M.T., Wickiewicz, T.L., et al., 2009. Medial Collateral Ligament Injuries and Subsequent Load on the Anterior Cruciate Ligament A Biomechanical Evaluation in a Cadaveric Model. *Am. J. Sports Med.* 37, 305-311.
- Bendjaballah, M.Z., ShiraziAdl, A., Zukor, D.J., 1997. Finite element analysis of human knee joint in varus-valgus. *Clin. Biomech.* 12, 139-148.
- Benoit, D.L., Ramsey, D.K., Lamontagne, M., Xu, L.Y., Wretenberg, P., Renstrom, P., 2006. Effect of skin movement artifact on knee kinematics during gait and cutting motions measured in vivo. *Gait Posture* 24, 152-164.
- Blankevoort, L., Huijskes, R., Delange, A., 1988. The envelope of passive knee-joint motion. *J. Biomech.* 21, 705-&.
- Bull, A.M.J., Kessler, O., Alam, M., Amis, A.A., 2008. Changes in knee kinematics reflect the articular geometry after arthroplasty. *Clin. Orthop. Rel. Res.* 466, 2491-2499.
- Butler, D.L., Noyes, F.R., Grood, E.S., 1980. Ligamentous restraints to anterior-posterior drawer in the human knee - biomechanical study. *J. Bone Joint Surg.-Am. Vol.* 62, 259-270.
- Cannon, W.D., 2002. Use of arthrometers to assess knee laxity and outcomes. *Sports Med. Arthrosc. Rev.* 10, 191-200.
- Cloutier, J.M., Sabouret, P., Deghrar, A., 1999. Total knee arthroplasty with retention of both cruciate ligaments - A nine to eleven-year follow-up study. *J. Bone Joint Surg.-Am. Vol.* 81A, 697-702.
- D'Lima, D.D., Patil, S., Steklov, N., Colwell, C.W., 2011. The 2011 ABJS Nicolas Andry Award: 'Lab'-in-a-Knee: In Vivo Knee Forces, Kinematics, and Contact Analysis. *Clin. Orthop. Rel. Res.* 469, 2953-2970.
- Daniel, D.M., Malcom, L.L., Losse, G., Stone, M.L., Sachs, R., Burks, R., 1985. Instrumented measurement of anterior laxity of the knee. *J. Bone Joint Surg.-Am. Vol.* 67A, 720-726.
- Fantozzi, S., Catani, F., Ensini, A., Leardini, A., Giannini, S., 2006. Femoral rollback of cruciate-retaining and posterior-stabilized total knee replacements: In vivo fluoroscopic analysis during activities of daily living. *J. Orthop. Res.* 24, 2222-2229.
- Forster, I.W., Warrensmith, C.D., Tew, M., 1989. Is the KT1000 knee ligament arthrometer reliable. *J. Bone Joint Surg.-Br. Vol.* 71, 843-847.
- Freeman, M.A., Railton, G.T., 1988. Should the posterior cruciate ligament be retained or resected in condylar nonmeniscal knee arthroplasty? The case for resection. *The Journal of arthroplasty* 3 Suppl, S3-12.
- Fujie, H., Livesay, G.A., Woo, S.L.Y., Kashiwaguchi, S., Blomstrom, G., 1995. The use of a universal force-moment sensor to determine in-situ forces in ligaments - a new methodology. *J. Biomech. Eng.-Trans. ASME* 117, 1-7.
- Fujie, H., Mabuchi, K., Woo, S.L.Y., Livesay, G.A., Arai, S., Tsukamoto, Y., 1993. The use of robotics technology to study human joint kinematics - a new methodology. *J. Biomech. Eng.-Trans. ASME* 115, 211-217.
- Fukubayashi, T., Torzilli, P.A., Sherman, M.F., Warren, R.F., 1982. An invitro biomechanical evaluation of anterior-posterior motion of the knee - tibial displacement, rotation, and torque. *J. Bone Joint Surg.-Am. Vol.* 64, 258-264.
- Gabriel, M.T., Wong, E.K., Woo, S.L.Y., Yagi, M., Debski, R.E., 2004. Distribution of in situ forces in the anterior cruciate ligament in response to rotatory loads. *J. Orthop. Res.* 22, 85-89.
- Ghosh, K.M., Merican, A.M., Iranpour, F., Deehan, D.J., Amis, A.A., 2012. Length-change patterns of the collateral ligaments after total knee arthroplasty. *Knee Surg. Sports Traumatol. Arthrosc.* 20, 1349-1356.
- Girard, J., Amzallag, M., Pasquier, G., Mulliez, A., Brosset, T., Gougeon, F., et al., 2009. Total knee arthroplasty in valgus knees: Predictive preoperative parameters influencing a constrained design selection. *Orthop. Traumatol.-Surg. Res.* 95, 260-266.

495 Gollehon, D.L., Torzilli, P.A., Warren, R.F., 1987. The role of the posterolateral and cruciate
 496 ligaments in the stability of the human knee - a biomechanical study. *J. Bone Joint Surg.-*
 497 *Am.* Vol. 69A, 233-242.
 498 Grood, E.S., Noyes, F.R., Butler, D.L., Suntay, W.J., 1981. Ligamentous and capsular
 499 restraints preventing straight medial and lateral laxity in intact human cadaver knees. *J.*
 500 *Bone Joint Surg.-Am.* Vol. 63, 1257-1269.
 501 Grood, E.S., Stowers, S.F., Noyes, F.R., 1988. Limits of movement in the human knee -
 502 effect of sectioning the posterior cruciate ligament and posterolateral structures. *J. Bone*
 503 *Joint Surg.-Am.* Vol. 70A, 88-97.
 504 Gupte, C.M., Bull, A.M.J., Thomas, R.D., Amis, A.A., 2003. The meniscomfemoral ligaments:
 505 secondary restraints to the posterior drawer - Analysis of anteroposterior and rotary laxity in
 506 the intact and posterior-cruciate-deficient knee. *J. Bone Joint Surg.-Br.* Vol. 85B, 765-773.
 507 Gustke, K.A., 2005. Preoperative planning for revision total knee arthroplasty. *J. Arthroplast.*
 508 20, 37-40.
 509 Haider, H., Walker, P.S., 2005. Measurements of constraint of total knee replacement. *J*
 510 *Biomech* 38, 341-348.
 511 Heesterbeek, P.J.C., Keijsers, N.L.W., Wymenga, A.B., 2010. Ligament releases do not lead
 512 to increased postoperative varus-valgus laxity in flexion and extension: a prospective clinical
 513 study in 49 TKR patients. *Knee Surg. Sports Traumatol. Arthrosc.* 18, 187-193.
 514 Heesterbeek, P.J.C., Keijsers, N.L.W., Jacobs, W., Verdonschot, N., Wymenga, A.B., 2010.
 515 Posterior cruciate ligament recruitment affects antero-posterior translation during flexion gap
 516 distraction in total knee replacement. *Acta Orthop* 81, 471-477.
 517 Hoher, J., Harner, C.D., Vogrin, T.M., Baek, G.H., Carlin, G.J., Woo, S.L.Y., 1998. In situ
 518 forces in the posterolateral structures of the knee under posterior tibial loading in the intact
 519 and posterior cruciate ligament-deficient knee. *J. Orthop. Res.* 16, 675-681.
 520 Ishii, Y., Matsuda, Y., Ishii, R., Sakata, S., Omori, G., 2005. Sagittal laxity in vivo after total
 521 knee arthroplasty. *Arch. Orthop. Trauma Surg.* 125, 249-253.
 522 Ishii, Y., Noguchi, H., Takeda, M., Kiga, H., Toyabe, S.I., 2011. Effect of voluntary soft tissue
 523 tension and articular conformity after total knee arthroplasty on in vivo anteroposterior
 524 displacement. *Knee* 18, 11-14.
 525 Jacobs, W.C.H., Clement, D.J., Wymenga, A.B., 2005. Retention versus removal of the
 526 posterior cruciate ligament in total knee replacement - A systematic literature review within
 527 the Cochrane framework. *Acta Orthop.* 76, 757-768.
 528 Jilani, A., ShiraziAdl, A., Bendjaballah, M.Z., 1997. Biomechanics of human tibio-femoral
 529 joint in axial rotation. *Knee* 4, 203-213.
 530 Kanamiya, T., Whiteside, L.A., Nakamura, T., Mihalko, W.M., Steiger, J., Naito, M., 2002.
 531 Ranawat Award paper. Effect of selective lateral ligament release on stability in knee
 532 arthroplasty. *Clin Orthop Relat Res.* 24-31.
 533 Kanamori, A., Sakane, M., Zeminski, J., Rudy, T.W., Woo, S.L., 2000. In-situ force in the
 534 medial and lateral structures of intact and ACL-deficient knees. *Journal of orthopaedic*
 535 *science : official journal of the Japanese Orthopaedic Association* 5, 567-571.
 536 Kesman, T.J., Kaufman, K.R., Trousdale, R.T., 2011. Popliteus Tendon Resection During
 537 Total Knee Arthroplasty An Observational Report. *Clin. Orthop. Rel. Res.* 469, 76-81.
 538 Kessler, O., Bull, A.M.J., Amis, A.A., 2009. A method to quantify alteration of knee
 539 kinematics caused by changes of TKR positioning. *J. Biomech.* 42, 665-670.
 540 Kim, S.J., Kim, H.K., 1995. Reliability of the anterior drawer test, the pivot shift, and the
 541 lachman test. *Clin. Orthop. Rel. Res.*, 237-242.
 542 Koh, H.S., In, Y., 2013. Semimembranosus Release as the Second Step of Soft Tissue
 543 Balancing in Varus Total Knee Arthroplasty. *J. Arthroplast.* 28, 273-278.
 544 Krackow, K.A., Mihalko, W.M., 1999. Flexion-extension joint gap changes after lateral
 545 structure release for valgus deformity correction in total knee arthroplasty - A cadaveric
 546 study. *J. Arthroplast.* 14, 994-1004.
 547 Kurtz, S.M., Ong, K.L., Lau, E., Widmer, M., Maravic, M., Gomez-Barrena, E., et al., 2011.
 548 International survey of primary and revision total knee replacement. *Int. Orthop.* 35, 1783-
 549 1789.

550 Levy, I.M., Torzilli, P.A., Gould, J.D., Warren, R.F., 1989. The effect of lateral meniscectomy
551 on motion of the knee. *J. Bone Joint Surg.-Am.* Vol. 71A, 401-406.

552 Levy, I.M., Torzilli, P.A., Warren, R.F., 1982. The effect of medial meniscectomy on anterior-
553 posterior motion of the knee. *J. Bone Joint Surg.-Am.* Vol. 64, 883-888.

554 Li, G., Most, E., DeFrate, L.E., Suggs, J.F., Gill, T.J., Rubash, H.E., 2004. Effect of the
555 posterior cruciate ligament on posterior stability of the knee in high flexion. *J. Biomech.* 37,
556 779-783.

557 Li, G., Zayontz, S., Most, E., Otterberg, E., Sabbag, K., Rubash, H.E., 2001. Cruciate-
558 retaining and cruciate-substituting total knee arthroplasty - An in vitro comparison of the
559 kinematics under muscle loads. *J. Arthroplast.* 16, 150-156.

560 Lim, H.C., Bae, J.H., Bae, T.S., Moon, B.C., Shyam, A.K., Wang, J.H., 2012. Relative role
561 changing of lateral collateral ligament on the posterolateral rotatory instability according to
562 the knee flexion angles: a biomechanical comparative study of role of lateral collateral
563 ligament and popliteofibular ligament. *Arch. Orthop. Trauma Surg.* 132, 1631-1636.

564 Lipke, J.M., Janecki, C.J., Nelson, C.L., McLeod, P., Thompson, C., Thompson, J., et al.,
565 1981. The role of incompetence of the anterior cruciate and lateral ligaments in anterolateral
566 and anteromedial instability - a biomechanical study of cadaver knees. *J. Bone Joint Surg.-*
567 *Am.* Vol. 63, 954-960.

568 Matsueda, M., Gengerke, T.R., Murphy, M., Lew, W.D., Gustilo, R.B., 1999. Soft tissue
569 release in total knee arthroplasty - Cadaver study using knees without deformities. *Clin.*
570 *Orthop. Rel. Res.*, 264-273.

571 McAuley, J.P., Engh, G.A., 2003. Constraint in total knee arthroplasty - When and what? *J.*
572 *Arthroplast.* 18, 51-54.

573 Mihalko, W.M., Krackow, K.A., 2000. Anatomic and biomechanical aspects of pie crusting
574 posterolateral structures for valgus deformity correction in total knee arthroplasty - A
575 cadaveric study. *J. Arthroplast.* 15, 347-353.

576 Mihalko, W.M., Whiteside, L.A., Krackow, K.A., 2003. Comparison of ligament-balancing
577 techniques during total knee arthroplasty. *J. Bone Joint Surg.-Am.* Vol. 85A, 132-135.

578 Morgan, H., Battista, V., Leopold, S.S., 2005. Constraint in primary total knee arthroplasty. *J.*
579 *Am. Acad. Orthop. Surg.* 13, 515-524.

580 Most, E., Li, G., Schule, S., Sultan, P., Park, S.E., Zayontz, S., et al., 2003a. The kinematics
581 of fixed- and mobile-bearing total knee arthroplasty. *Clin. Orthop. Rel. Res.*, 197-207.

582 Most, E., Li, G.A., Sultan, P.G., Park, S.E., Rubash, H.E., 2005. Kinematic analysis of
583 conventional and high-flexion cruciate-retaining total knee arthroplasties - An in vitro
584 investigation. *J. Arthroplast.* 20, 529-535.

585 Most, E., Zayontz, S., Li, G.A., Otterberg, E., Sabbag, K., Rubash, H.E., 2003b. Femoral
586 rollback after cruciate-retaining and stabilizing total knee arthroplasty. *Clin. Orthop. Rel.*
587 *Res.*, 101-113.

588 Mulhall, K.J., Ghomrawi, H.M., Scully, S., Callaghan, J.J., Saleh, K.J., 2006. Current
589 etiologies and modes of failure in total knee arthroplasty revision. *Clin. Orthop. Rel. Res.*, 45-
590 50.

591 Nagamine, R., White, S.E., McCarthy, D.S., Whiteside, L.A., 1995. Effect of rotational
592 malposition of the femoral component on knee stability kinematics after total knee
593 arthroplasty. *J. Arthroplast.* 10, 265-270.

594 Naudie, D.D., Rorabeck, C.H., 2004. Managing instability in total knee arthroplasty with
595 constrained and linked implants. *Instructional course lectures* 53, 207-215.

596 Nelson, C.L., Gioe, T.J., Cheng, E.Y., Thompson, R.C., 2003. Implant selection in revision
597 total knee arthroplasty. *J. Bone Joint Surg.-Am.* Vol. 85A, 43-51.

598 Noyes, F.R., Grood, E.S., Butler, D.L., Ratterman, L., 1980. Knee ligament tests- what do
599 they really mean. *Phys. Ther.* 60, 1578-1581.

600 Okazaki, K., Miura, H., Matsuda, S., Takeuchi, N., Mawatari, T., Hashizume, M., et al., 2006.
601 Asymmetry of mediolateral laxity of the normal knee. *J. Orthop. Sci.* 11, 264-266.

602 Parratte, S., Pagnano, M.W., 2008. Instability after total knee arthroplasty. *J. Bone Joint*
603 *Surg.-Am.* Vol. 90A, 184-194.

- 604 Pasque, C., Noyes, F.R., Gibbons, M., Levy, M., Grood, E., 2003. The role of the
605 popliteofibular ligament and the tendon of popliteus in providing stability in the human knee.
606 J. Bone Joint Surg.-Br. Vol. 85B, 292-298.
- 607 Petersen, W., Loerch, S., Schanz, S., Raschke, M., Zantop, T., 2008. The role of the
608 posterior oblique ligament in controlling posterior tibial translation in the posterior cruciate
609 ligament-deficient knee. Am. J. Sports Med. 36, 495-501.
- 610 Race, A., Amis, A.A., 1996. Loading of the two bundles of the posterior cruciate ligament: An
611 analysis of bundle function in A-P drawer. J. Biomech. 29, 873-879.
- 612 Robinson, J.R., Bull, A.M.J., Thomas, R.R.D., Amis, A.A., 2006. The role of the medial
613 collateral ligament and posteromedial capsule in controlling knee laxity. Am. J. Sports Med.
614 34, 1815-1823.
- 615 Robinson, R.P., 2005. The early innovators of today's resurfacing condylar knees. J.
616 Arthroplast. 20, 2-26.
- 617 Rodriguez-Merchan, E.C., 2011. Instability following total knee arthroplasty. HSS journal :
618 the musculoskeletal journal of Hospital for Special Surgery 7, 273-278.
- 619 Rudy, T.W., Livesay, G.A., Woo, S.L.Y., Fu, F.H., 1996. A combined robotic/universal force
620 sensor approach to determine in situ forces of knee ligaments. J. Biomech. 29, 1357-1360.
- 621 Saeki, K., Mihalko, W.M., Patel, V., Conway, J., Naito, A., Thrum, H., et al., 2001. Stability
622 after medial collateral ligament release in total knee arthroplasty. Clin. Orthop. Rel. Res.,
623 184-189.
- 624 Sakane, M., Livesay, G.A., Fox, R.J., Rudy, T.W., Runco, T.J., Woo, S.L.Y., 1999. Relative
625 contribution of the ACL, MCL, and bony contact to the anterior stability of the knee. Knee
626 Surg. Sports Traumatol. Arthrosc. 7, 93-97.
- 627 Sculco, T.P., 2006. The role of constraint in total knee arthroplasty. J. Arthroplast. 21, 54-56.
- 628 Shapiro, M.S., Markolf, K.L., Finerman, G.A.M., Mitchell, P.W., 1991. The effect of section of
629 the medialcollateral ligament on force generated in the anterior cruciate ligament. J. Bone
630 Joint Surg.-Am. Vol. 73A, 248-256.
- 631 Sharkey, P.F., Hozack, W.J., Rothman, R.H., Shastri, S., Jacoby, S.M., 2002. Why are total
632 knee arthroplasties failing today? Clin. Orthop. Rel. Res., 7-13.
- 633 Shirazi-Adl, A., Moglo, K.E., 2005. Effect of changes in cruciate ligaments pretensions on
634 knee joint laxity and ligament forces. Computer Methods in Biomechanics and Biomedical
635 Engineering 8, 17-24.
- 636 Steiner, M.E., Brown, C., Zarins, B., Brownstein, B., Koval, P.S., Stone, P., 1990.
637 Measurement of anterior-posterior displacement of the knee - a comparison of the results
638 with instrumented devices and with clinical examination. J. Bone Joint Surg.-Am. Vol. 72A,
639 1307-1315.
- 640 Stoddard, J.E., Deehan, D.J., Bull, A.M.J., McCaskie, A.W., Amis, A.A., 2013. The
641 kinematics and stability of single-radius versus multi-radius femoral components related to
642 Mid-range instability after TKA. Journal of orthopaedic research : official publication of the
643 Orthopaedic Research Society 31, 53-58.
- 644 Sugita, T., Amis, A.A., 2001. Anatomic and biomechanical study of the lateral collateral and
645 popliteofibular ligaments. Am. J. Sports Med. 29, 466-472.
- 646 Sullivan, D., Levy, I.M., Sheskier, S., Torzilli, P.A., Warren, R.F., 1984. Medial restraints to
647 anterior-posterior motion of the knee. J. Bone Joint Surg.-Am. Vol. 66A, 930-936.
- 648 Takahashi, T., Wada, Y., Yamamoto, H., 1997. Soft-tissue balancing with pressure
649 distribution during total knee arthroplasty. J. Bone Joint Surg.-Br. Vol. 79B, 235-239.
- 650 Takeda, M., Ishii, Y., Noguchi, H., Matsuda, Y., Sato, J., 2012. Changes in varus-valgus
651 laxity after total knee arthroplasty over time. Knee Surg. Sports Traumatol. Arthrosc. 20,
652 1984-1989.
- 653 Veltri, D.M., Deng, X.H., Torzilli, P.A., Maynard, M.J., Warren, R.F., 1996. The role of the
654 popliteofibular ligament in stability of the human knee - A biomechanical study. Am. J. Sports
655 Med. 24, 19-27.
- 656 Vince, K.G., Abdeen, A., Sugimori, T., 2006. The unstable total knee arthroplasty - Causes
657 and cures. J. Arthroplast. 21, 44-49.

- Walker, P.S., Sussman-Fort, J.M., Yildirim, G., Boyer, J., 2009. Design Features of Total Knees for Achieving Normal Knee Motion Characteristics. *J. Arthroplast.* 24, 475-483.
- Walker, P.S., Yildirim, G., Arno, S., Heller, Y., 2010. Future directions in knee replacement. *Proc. Inst. Mech. Eng. Part H-J. Eng. Med.* 224, 393-414.
- Whiteside, L.A., 2002. Soft tissue balancing - The knee. *J. Arthroplast.* 17, 23-27.
- Yamamoto, Y., Hsu, W.H., Fisk, J.A., Van Scyoc, A.H., Miura, K., Woo, S.L.Y., 2006. Effect of the iliotibial band on knee biomechanics during a simulated pivot shift test. *J. Orthop. Res.* 24, 967-973.
- Yang, J.H., Yoon, J.R., Oh, C.H., Kim, T.S., 2012. Primary total knee arthroplasty using rotating-hinge prosthesis in severely affected knees. *Knee Surg. Sports Traumatol. Arthrosc.* 20, 517-523.
- Yercan, H.S., Selmi, T.A.S., Sugun, T.S., Neyret, P., 2005. Tibiofemoral instability in primary total knee replacement: A review, Part 1: Basic principles and classification. *Knee* 12, 257-266.
- Zantop, T., Schumacher, T., Diermann, N., Schanz, S., Raschke, M.J., Petersen, W., 2007. Anterolateral rotational knee instability: role of posterolateral structures. *Arch. Orthop. Trauma Surg.* 127, 743-752.
- Zhao, D., Banks, S.A., D'Lima, D.D., Colwell, C.W., Fregly, B.J., 2007. In vivo medial and lateral tibial loads during dynamic and high flexion activities. *J. Orthop. Res.* 25, 593-602.
- Zihlmann, M.S., Gerber, H., Stacoff, A., Burckhardt, K., Szekely, G., Stussi, E., 2006. Three-dimensional kinematics and kinetics of total knee arthroplasty during level walking using single plane video-fluoroscopy and force plates: A pilot study. *Gait Posture* 24, 475-481.

Fig. 1. Ligamentous restraints to anterior displacement, in A) lateral, B) anterior, and C) medial views. Soft tissues highlighted in red signify primary restraint, and in yellow signify secondary restraints. ACL = anterior cruciate ligament, dMCL = deep medial collateral ligament, sMCL = superficial medial collateral ligament, MM = medial meniscus.

697 Fig. 2. Ligamentous restraints to posterior displacement, in A) lateral, B) anterior, and C)
 698 medial views. Soft tissues highlighted in red signify primary restraint, and in yellow signify
 699 secondary restraints. Pop T = popliteus tendon, PFL = popliteofibular ligament, PCL =
 700 posterior cruciate ligament, PMC = posteromedial capsule.

701 Fig. 3. Ligamentous restraints to valgus rotation, in A) lateral, B) anterior, and C) medial
 702 views. Soft tissues highlighted in red signify primary restraint, and in yellow signify
 703 secondary restraints. PCL = posterior cruciate ligament, dMCL = deep medial collateral
 704 ligament, sMCL = superficial medial collateral ligament, PMC = posteromedial capsule.

705 Fig. 4. Ligamentous restraints to varus rotation, in A) lateral, B) anterior, and C) medial
 706 views. Soft tissues highlighted in red signify primary restraint, and in yellow signify
 707 secondary restraints. Pop T = popliteus tendon, PFL = popliteofibular ligament, LCL = lateral
 708 collateral ligament, ACL = anterior cruciate ligament.

709 Fig. 5. Ligamentous restraints to internal rotation, in A) lateral, B) anterior, and C) medial
 710 views. Soft tissue highlighted in red signify primary restraint, and in yellow signify secondary
 711 restraints. ACL = anterior cruciate ligament, sMCL = superficial medial collateral ligament,
 712 PMC = posteromedial capsule.

713 Fig. 6. Ligamentous restraints to external rotation, in A) lateral, B) anterior, and C) medial
 714 views. Soft tissues highlighted in red signify primary restraint, and in yellow signify
 715 secondary restraints. Pop T = popliteus tendon, PFL = popliteofibular ligament, LCL = lateral
 716 collateral ligament, PCL = posterior cruciate ligament.

717 Fig. 7. Diagrams of a right knee demonstrating increasing surgical invasiveness and bone
 718 loss, going from A) intact, B) cruciate-retaining implant, C) posterior-stabilised implant, to D)
 719 constrained condylar implant. With increasing prosthetic constraint, the load transmission
 720 moves from the soft tissues to the implant fixation; this is illustrated for a knee abduction
 721 (valgus) moment. In A) the arrows signify the compressive lateral tibiofemoral contact point
 722 and tensile medial collateral ligament (MCL) force restraining the applied valgus moment,

723 which is similarly replicated in B) and C). However in the case of D) with a deficient or
724 absent MCL, the moment is resisted by implant-bone interface stresses distributed along the
725 wall of the box mechanism and the tibial/ femoral intramedullary stems.

Table 1 A summary of studies investigating ligamentous and soft tissue restraints in intact knees.

Lead Author	Sample Type	Method	Ligament/ Soft tissue investigated?							Number of samples	Kinematic test (referenced to the tibia)
			A	P	L	P	M	P	Others		
			C	C	C	L	C	M			
			L	L	L	S	L	C			
Butler et al. (1980)	In vitro	MTS 1 DoF	•	•	•	•	•	•	ITB	11	Ant/Post ± 5mm
Grood et al. (1981)	In vitro	MTS 1 DoF	•	•	•	•	•	•	ITB	10	Var/Val (± 6mm medial/lateral joint opening)
Fukubayashi et al. (1982)	In vitro	MTS 4 DoF	•	•						9	
Sullivan et al. (1984)	In vitro	MTS 5 DoF	•				•	•		10	Ant/Post 100N
Daniel et al. (1985)	In vivo/ in vitro	Arthrometer	•						-	460 ^a	Ant/Post 89N
Gollehon et al. (1987)	In vitro	MTS 5 DoF	•	•	•	•			-	17	Ant/Post 100N, Var/Val 10Nm, Int/Ext 4.5Nm
Grood et al. (1988)	In vitro	Rig with tibia free hanging/vertical		•	•	•			-	15	Ant/Post 100N, Var/Val 20Nm, Int/Ext 5Nm
Levy et al. (1989)	In vitro	MTS 5 DoF	•						MM,LM	11	Ant/Post 100N, Var/Val 20Nm, Int/Ext 6Nm
Shapiro et al. (1991)	In vitro	Cruciate-attached force transducer	•				•			10	Ant 78N, Var/Val 15Nm, Int/Ext 10Nm
Race and Amis (1996)	In vitro	MTS 4 DoF		•					-	9	Ant/Post ± 6mm
Bendjaballah et al. (1997)	In silico	Finite element model	•	•	•		•		MM, LM	-	Var/Val 15Nm
Jilani et al. (1997)	In silico	Finite element model	•	•	•		•		MM, LM	-	Int/Ext 10Nm
Hoher et al. (1998)	In vitro	Robotic		•	•	•			-	8	Post 110N with popliteus muscle 44N
Krackow and Mihalko (1999)	In vitro	Rig with tibia free hanging/vertical			•	•			ITB,MM, LM	12	Var/Val stress test

Sakane et al. (1999)	In vitro	Robotic	•	•	•	•	-	10	Ant/Post 110N
Kanamori et al. (2000)	In vitro	Robotic	•		•	•	-	8	Ant 134N
Gupte et al. (2003)	In vitro	MTS 4 DoF		•			MFL	8	Ant/Post 100N, Int/Ext 5Nm
Pasque et al. (2003)	In vitro	Rig with tibia upside down/vertical 1DoF		•	•	•	-	12	Post 100N, Var 10Nm, Ext 5Nm
Gabriel et al. (2004)	In vitro	Robotic	•				-	10	Ant 134N, Rotatory (Val 10Nm, Int 5Nm)
Li et al. (2004)	In vitro	Robotic		•			-	12	Post 130N with quads 400N and hams 200N
Shirazi-Adl and Moglo (2005)	In silico	Finite element model	•	•	•	•	MM, LM	-	Ant/Post 100N
Robinson et al. (2006)	In vitro	MTS 4 DoF				•	•	-	14
Zantop et al. (2007)	In vitro	Robotic	•		•	•	-	10	Ant 134N, Pivot shift (Ant 134N, Val 4Nm, Int 4Nm)
Petersen et al. (2008)	In vitro	Robotic		•		•	•	-	10
Battaglia et al. (2009)	In vitro	Robotic	•			•	-	10	Ant 125N, Int/Ext 4Nm, Val 10Nm

Key to content: MTS = Materials Testing System, DoF = degrees of freedom, ACL = anterior cruciate ligament, PCL = posterior cruciate ligament, LCL = lateral collateral ligament, PLS = posterolateral structures, MCL = medial collateral ligament, PMC = posteromedial capsule, ITB = iliotibial band, MM = medial meniscus, LM = lateral meniscus, MFL = meniscofemoral ligaments, Ant = anterior translation, Post = posterior translation, Int = Internal rotation, Ext = external rotation, Val = valgus rotation, Var = varus rotation, Quads = quadriceps muscles, Hams = hamstring muscles.

^a Of the 460 samples, 33 were cadaver knees, 338 were normal patients, and 89 were patients with unilateral ACL rupture.

Table 2 A summary of studies investigating ligamentous and soft tissue restraints in total knee arthroplasty implanted-knees.

Lead Author	Sample Type	Method	Ligament/ Soft tissue investigated?							Arthroplasty	Number of samples	Kinematic test (referenced to the tibia)
			A	P	L	P	M	P	Others			
			C	C	C	L	C	M				
			L	L	L	S	L	C				
Nagamine et al. (1995)	In vitro	E. E. test rig ^a				•				TKA	8	Var/ Val 45N applied at ankle, Int/Ext 3Nm
Takahashi et al. (1997)	In vivo	Pressure film (intraop)/ Arthrometer (postop)					•	•		CR TKA	63	Passive flexion, Var/ Val 150N
Matsueda et al. (1999)	In vitro	Cable/spring set-up		•	•	•	•	•	Semi M, ITB	CR TKA	12	Var/ Val 10Nm
Li et al. (2001)	In vitro	Robotic		•					-	CR and PS TKA	9	Passive flexion with quads 400N and hams 200N
Saeki et al. (2001)	In vitro	E. E. test rig ^a		•			•			CR and PS TKA	6	Ant/ Post 35N, Var/ Val 10Nm, Int/Ext 1.5Nm
Kanamiya et al. (2002)	In vitro	E. E. test rig ^a			•	•			ITB	CR TKA	17	Var/ Val 10Nm, Int/ Ext 10Nm
Most et al. (2003b)	In vitro	Robotic		•					-	CR and PS TKA	9	Passive flexion without muscle loads
Barink et al. (2005)	In silico	Finite element model		•	•		•			CR TKA	-	Ant/ Post 100N, Var/ Val 10Nm, Int/ Ext 3Nm
Ishii et al. (2005)	In vivo	Arthrometer	•	•					-	CR and PS TKA	77	Ant 133N, Post 89N
Most et al. (2005)	In vitro	Robotic		•					-	CR and high-flexion CR TKA	10	Passive flexion with quads 400N and hams 200N
Heesterbeek et al. (2010)	In vivo	Calibrated spring (intraop)/				•	•	•	ITB	CR TKA	49	Var/ Val 15Nm

Kesman et al. (2011)	In vivo	Arthrometer (postop) Surgeon var/val stress test	•		PS TKA	18	Var/ Val stress test
Takeda et al. (2012)	In vivo	Arthrometer	•	-	CR and PS TKA, mobile-bearing PS TKA	60	Var/Val 150N
Koh and In (2013)	In vivo	Arthrometer	•	•	Semi M	104	Var/ Val stress test

Key to content: ACL = anterior cruciate ligament, PCL = posterior cruciate ligament, LCL = lateral collateral ligament, PLS = posterolateral structures, MCL = medial collateral ligament, PMC = posteromedial capsule, Semi M = semimembranosus tendon, ITB = iliotibial band, CR TKA = cruciate-retaining total knee arthroplasty, PS TKA = posterior-stabilised total knee arthroplasty, Ant = anterior translation, Post = posterior translation, Int = Internal rotation, Ext = external rotation, Val = valgus rotation, Var = varus rotation, Quads = quadriceps muscles, Hams = hamstring muscles.

^a Knee kinematic testing rig (Experimental Engineering, Little Rock, AR, USA)

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