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Measurement of in vivo anterior cruciate ligament strain during dynamic jump landing

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Abstract

Despite recent attention in the literature, anterior cruciate ligament (ACL) injury mechanisms are controversial and incidence rates remain high. One explanation is limited data on in vivo ACL strain during high-risk, dynamic movements. The objective of this study was to quantify ACL strain during jump landing. Marker-based motion analysis techniques were integrated with fluoroscopic and magnetic resonance (MR) imaging techniques to measure dynamic ACL strain non-invasively. First, eight subjects' knees were imaged using MR. From these images, the cortical bone and ACL attachment sites of the tibia and femur were outlined to create 3D models. Subjects underwent motion analysis while jump landing using reflective markers placed directly on the skin around the knee. Next, biplanar fluoroscopic images were taken with the markers in place so that the relative positions of each marker to the underlying bone could be quantified. Numerical optimization allowed jumping kinematics to be superimposed on the knee model, thus reproducing the dynamic in vivo joint motion. ACL length, knee flexion, and ground reaction force were measured. During jump landing, average ACL strain peaked 55 ± 14 ms (mean and 95% confidence interval) prior to ground impact, when knee flexion angles were lowest. The peak ACL strain, measured relative to its length during MR imaging, was 12 ± 7%. The observed trends were consistent with previously described neuromuscular patterns. Unrestricted by field of view or low sampling rate, this novel approach provides a means to measure kinematic patterns that elevate ACL strains and that provide new insights into ACL injury mechanisms.

Keywords

ACL, anterior cruciate ligament; In vivo; Strain; Dynamic; Motion; MR; Magnetic resonance; Fluoroscopy; Imaging; Injury mechanisms; Kinematics; Jumping; Landing; Valgus

1. Introduction

Over 200,000 anterior cruciate ligament (ACL) injuries occur in the United States every year, half of which are experienced by young athletes between 15 and 25 years of age

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Conflict of interest
The authors have no conflicts of interest to declare with regards to this study.

(Miyasaka et al., 1991; AAOS, 2008). The consequences of ACL deficiency incurred from ACL injury include pain, instability, damage to the menisci, and early-onset osteoarthritis (OA) (Fairclough et al., 1990; Roos et al., 1995; Fithian et al., 2002; Hill et al., 2005). Furthermore, ACL deficiency greatly compromises the ability to participate in sports that require jumping and cutting (van Lent et al., 1994; Fink et al., 2001; Katayama et al., 2004; Maquirriain and Megey, 2006; Kapreli et al., 2009). For these reasons, between 100,000 and 175,000 patients elect to undergo ACL reconstruction annually (Koh, 2005; Griffin et al., 2006). Although surgical intervention provides good short-term outcomes, long-term results are less consistent (Asano et al., 2002; Wolf and Lemak, 2002; Lohmander et al., 2004; von Porat et al., 2004; Grossman et al., 2005). Some studies have suggested that current reconstructive techniques do not decrease the probability of developing OA when compared to non-operative treatment (Fink et al., 2001; Lohmander et al., 2004; von Porat et al., 2004; Lohmander et al., 2007; Butler et al., 2009).

Because ACL injury affects such a young population (Roos et al., 1995; Beynnon et al., 2005) and surgery has mixed results in preventing early-onset OA (Fithian et al., 2002; Lohmander et al., 2007), there has been great interest in developing ACL injury prevention programs (Hewett et al., 1999; Heidt et al., 2000; Myklebust et al., 2003; Gilchrist et al., 2008). Many studies have focused on non-contact ACL injury mechanisms, which account for 70% of all ACL injuries (McNair et al., 1990; Weinhold et al., 2007; Gianotti et al., 2008) and potentially could be prevented with an appropriate training program. However, the levels of success achieved by current prevention programs have shown varied efficacy (Hewett et al., 1999; Soderman et al., 2000; Myklebust et al., 2003; Mandelbaum et al., 2005; Pfeiffer et al., 2006; Barber-Westin et al., 2009) and despite their implementation, high rates of non-contact ACL injuries persist (Agel et al., 2005). These findings suggest that there is an incomplete understanding of the underlying injury mechanisms. Specifically, there are limited in vivo data on ACL strain, a critical parameter for predicting ACL failure.

Numerous studies have investigated ACL injury mechanisms using videographic and motion analyses (Chappell et al., 2002; Chappell et al., 2007; Krosshaug et al., 2007a, 2007b; Boden et al., 2009; Hewett et al., 2009; Nagano et al., 2009). These studies provide important kinematic data, but do not directly measure ACL strain. Many previous studies have examined ACL loading in cadavers (Draganich and Vahey, 1990; Markolf et al., 1990; Woo et al., 1991; Li et al., 1999; Kanamori et al., 2000; DeMorat et al., 2004). Although these data give valuable information on ACL function, their application to in vivo environments are restricted by an inability to recreate complex multi-planar loading conditions experienced during dynamic jumping and cutting activities. Using implantable strain gauges, some in vivo measurements of ACL strain have also been reported (Beynnon and Fleming, 1998; Fleming et al., 1999; Cerulli et al., 2003; Fleming and Beynnon, 2004). Beynnon and Fleming et al. (Beynnon et al., 1997; Fleming et al., 1998; Fleming et al., 1999) ran a series of in vivo strain studies to understand how an ACL graft would be loaded during common post-surgical rehabilitation exercises and daily tasks. These data were the basis for many pertinent clinical rehabilitation guidelines. However, there are limited data on ACL strains during sport-specific dynamic movements. Dynamic ACL strain data are needed to accurately predict what motions predispose the ACL to injury. The objective of this study was to measure in vivo ACL strain non-invasively during a dynamic jumping activity using a novel method developed by our laboratory.

2. Methods

Institutional Review Board approval was obtained for this protocol before enrollment of participants. Eight healthy male subjects (mean age: 26 years, range: 19–38 years) were recruited to participate in this study. All volunteers were physically active, participating in

recreational sports or exercise a minimum of three days a week for at least one hour each day. The potential subjects were disqualified if they had a history of knee injury.

All subjects were imaged with a 3T magnet (Trio Tim, Siemens Medical Solutions USA, Malvern, PA). Coronal, sagittal, and axial images were taken with the patient supine and the knee in a relaxed position. MR images were acquired using a double-echo steady state sequence (DESS, flip angle: 25°, TR: 17 ms, TE: 6 ms) and an eight channel receive-only knee coil with a field of view of 15 × 15 cm^2, a matrix of 512 × 512 pixels, and a slice thickness of 1 mm. From these images, the outer margins of the cortical bone and ACL attachment site were outlined using solid-modeling software (Rhinoceros 4.0, Robert McNeel and Associates, Seattle, WA). These tracings were compiled to create subject-specific 3D models of each tested knee. The location of the ACL was confirmed using orthogonal image sets. This methodology accurately measures the location of the ACL footprint center to within 0.3 mm, as described previously (Abebe et al., 2009).

Subjects next underwent a 3D motion analysis using an eight camera motion capture system with a sampling rate of 240 Hz (Motion Analysis Corporation, Santa Rosa, CA). The calibrated capture volume for the system was 3 m long, 2 m wide, and 3 m high. Also, centered within the capture volume were four embedded force plates (AMTI, Boston, MA, USA) with a sampling rate of 2400 Hz. All subjects were provided with standardized form-fitting athletic wear and footwear. Reflective markers were placed unilaterally on various anatomic landmarks used in previous gait studies (Queen et al., 2006; Queen et al., 2008) on the subject's leg. Additionally, non-symmetric clusters of markers were also placed on the thigh and shank until a total of 28 markers were positioned on the leg (Fig. 1). The primary goal of this complex marker set was to over-constrain each anatomical segment (thigh, shank) so that the effects of skin motion could be minimized via numerical optimization, as demonstrated by previous investigators (Andriacchi et al., 1998; Alexander and Andriacchi, 2001; Ngai et al., 2009; Ngai and Wimmer, 2009).

Marker data were captured initially during a static standing trial with the subject's feet shoulder width apart for one second. Next, subjects performed five successful trials of a jump landing task. Starting from a platform 0.47 m off of the ground and half their standing height away from the force plate's edge, subjects were asked to jump from the platform onto two force plates, then immediately jump straight up with maximal effort and land back on the same force plates again. This task is similar to previous activities studied in the literature that were designed to simulate jumping and landing activities in soccer or basketball (Onate et al., 2005; Chappell et al., 2007).

Upon completion of the 3D motion capture session, subjects were imaged with the markers still in the same positions using biplanar fluoroscopy (DeFrate et al., 2004; Caputo et al., 2009) while performing a static standing trial similar to the one performed prior to motion capture. Due to the difficulties associated with visualizing all of the markers at one time during fluoroscopy, the femur and tibia were centered individually for each imaging trial. From these image sets, the 3D orientation of the markers relative to the underlying bones was defined. First, the fluoroscopic test environment was reproduced in the solid-modeling software. Then, the 3D joint model was imported into the environment and viewed from two orthogonal directions corresponding to the location of the image sources of each fluoroscope. Next, the position and orientation of the model were manipulated manually in six degrees-of-freedom (6DoF) until their projections, as viewed from the two orthogonal directions, matched the outlines on the fluoroscopic images (Fig. 2). Previous validation has shown that this method can measure 3D kinematics to within 0.1 mm and 0.3° (DeFrate et al., 2006; Caputo et al., 2009). Positions of the markers were calculated from the area

centroids of each marker on the orthogonal image set. In this fashion, the relationship of the markers to the underlying bones and ACL attachment sites was quantified.

To create the dynamic in vivo model, the marker data obtained from fluoroscopy was initially registered to the static standing trial data from the motion analysis via numerical optimization one bone at a time. Assuming that the array of points measured from biplanar fluoroscopy was fixed relative to each bone, this marker set was rigidly translated and rotated in 3D space until the distance to each corresponding marker, as measured from the motion analysis, during the standing trial was minimized as such:

$$\min_{o,a,t,\ \boldsymbol{\delta}_x,\boldsymbol{\delta}_y,\boldsymbol{\delta}_z} \sum_{i}^{n} (\underline{\mathbf{p}}_i^* - \underline{\mathbf{q}}_i) \cdot (\underline{\mathbf{p}}_i^* - \underline{\mathbf{q}}_i), \tag{1}$$

where

$$\underline{\mathbf{p}}_i^* = \underline{\mathbf{R}}(\underline{\mathbf{p}}_i + \underline{\boldsymbol{\delta}}) \tag{2}$$

where $\underline{\mathbf{p}}_i$ is the position of the *i*th marker in the fluoroscopic model, $\underline{\mathbf{q}}_i$ is the position of the *i*th marker during the motion analysis standing trial, $\underline{\mathbf{R}}$ is a 3D rotation matrix composed from rotations **o**, **a**, and **t** about the *x*, *y*, and *z* axes, and $\underline{\boldsymbol{\delta}}$ is a translation vector consisting of three translations along the *x*, *y*, and *z* axes ($\boldsymbol{\delta}_x$, $\boldsymbol{\delta}_y$, $\boldsymbol{\delta}_z$). The stated initialization step registers the underlying bone and ACL attachment to the motion analysis based global coordinate system.

Next, similar minimizations using Eqs. (1) and (2) were performed to rigidly transform the standing trial marker positions to the positions of the markers at each time step during the dynamic trials. To further reduce error, all possible combinations of ten markers on the thigh (out of a total of 17) and seven markers (out of a total of 11) on the shank were minimized. These specific subset counts were selected for their balance of solution stability and computational efficiency. From all these solutions (19,448 combinations for the thigh), the subset that best minimized the objective function was selected for each time step. In this fashion, marker sets which deviated the least from rigid body motion were chosen to optimize the motion of the underlying bony structures. Other investigators have demonstrated that over-constrained marker sets and optimization techniques, such as the point cluster technique (Andriacchi et al., 1998;Alexander and Andriacchi, 2001;Ngai et al., 2009;Ngai and Wimmer, 2009), can be used to minimize error associated with the soft tissue motion.

The 3D models were then transformed according to the optimized kinematics in order to reproduce the motion of the subject's knee during the dynamic jumping activity. From these models, the subject's ACL length and knee flexion were measured as a function of time. Cylinders were fit to the long shafts of the tibia and femur so that flexion could be measured about an axis fixed through the transepicondylar line of the femur. ACL length was defined as the distance between the centroids of the femoral and tibial attachments of the ligament (Li et al., 2005). Because the true resting length of the ACL is difficult to quantify, ACL strain was measured from ACL length changes relative to a reference length as follows:

$$\varepsilon = \frac{\mathbf{L} - \mathbf{L_o}}{\mathbf{L_o}} \times 100\% \quad (3)$$

where ε is the relative ACL strain, **L** is ACL length, and $\mathbf{L_0}$ is a reference length (defined as the length captured during MR imaging, where the fibers appeared taut). Average flexion of the subjects' knees during MR imaging was 16.6 ± 6.2° (mean and 95% confidence interval).

Easily repeatable biomechanical milestones were chosen to define the beginning and end of the movement cycle: toe-off from the platform (0%) and peak vertical ground reaction force (100%). All data were linearly interpolated to the nearest integer percent and averaged for five jump trials from each subject and then across all individuals.

2.1. Validation study

To validate the combined motion analysis and biplanar fluoroscopy methodology, measurements of ACL length using this approach were compared to those obtained from only a biplanar fluoroscopy technique used previously (Jordan et al., 2007; Abebe et al., 2010). Since our biplanar fluoroscopy system is not equipped for high speed imaging and has a limited field of view, we could not analyze dynamic jumping activities. Therefore, a quasi-static lunge was analyzed.

Four subjects first performed a quasi-static lunge from 0° to 45° of flexion in increments of 5° in the gait laboratory using the combined protocols outlined above. These data were used to generate a curve of length versus flexion as measured from the 3D models. Next, each subject performed a similar lunge under biplanar fluoroscopy only, and a similar graph was generated. These curves were compared at 0°, 15°, 30°, and 45° of knee flexion. From these points, a graph of ACL length as measured from the combined approach versus ACL length as measured from imaging techniques alone was produced (Fig. 3).

3. Results

3.1. Validation study

Linear regression demonstrated that the two techniques had excellent correlation, with a coefficient of determination of 0.92. Root mean square error between the measurements was 0.5 mm. These results indicate that the combined methodology accurately measures ACL deformation up to 45° of flexion during a quasi-static lunge.

3.2. Jump landing trials

The results of the jump landing trials are depicted in Fig. 4. Time of initial contact with the ground was defined as the incidence of a minimum 10 N vertical ground reaction force (GRF) (Ford et al., 2006). GRF observed in this experiment exhibited two peaks typical of a heel–toe contact sequence while landing from a jump (Kovacs et al., 1999).

During the jump landing movement cycle, flexion initially increased as the knees were brought to the front of the body (analogous to swing phase in normal walking patterns) to an average of 48.9° ± 14.7° (mean and 95% confidence interval). Next, flexion decreased for 302 ± 51 ms to a minimum of 11.2° ± 3.5° and then increased in anticipation of landing prior to the impact with the ground. Approximately 4° of flexion was recovered in this period. After ground contact, the knee began to flex rapidly as the force of impact was dissipated.

In general, the length of the ACL during the jump landing decreased with increasing flexion angle. For example, as the legs were extended in anticipation of landing, the length of the ACL increased. Peak ACL length was observed 55 ± 14 ms before initial force plate contact, when flexion angles were at their lowest values. This point corresponded to an average relative ACL strain of 12 ± 7%. After ground contact, the ACL length initially spiked to a local maximum, but quickly decreased as the knee bent. The post impact local maximum in the ACL length demonstrated 5% less relative strain than the absolute maximum prior to impact.

4. Discussion

Intrinsic loading during non-contact events are the most prevalent cause of ACL injuries. Recently, much emphasis has been placed on identifying the underlying mechanisms of these injuries with the intent of developing prevention programs to curtail their rates of incidence. Prevention programs have shown mixed results (Hewett et al., 1999; Grandstrand et al., 2006; Pfeiffer et al., 2006), and a decrease in the injury rates has yet to be realized (Agel et al., 2005). This is most likely due to a gap in the literature on the relationship of in vivo joint kinematics and ACL strain. Biplanar fluoroscopic studies have provided invaluable data on joint kinematics during various in vivo activities (Bey et al., 2008; Caputo et al., 2009; Anderst and Tashman, 2010; Brainerd et al., 2010; Torry et al., 2010; Wu et al., 2010). However, high speed biplanar fluoroscopy may not be readily available and may be potentially limited by a small field of view (Benoit et al., 2007). To address limitations in sampling rate and field of view of our biplanar fluoroscopy system, we developed a non-invasive method using a combination of MR imaging, biplanar fluoroscopy, and traditional marker-based motion analysis capable of approximating in vivo ACL strain during dynamic physical tasks.

Previous studies on ACL injury mechanisms have attempted to explain the etiology by comparing high risk populations. By examining populations who are known to suffer greater incidence rates of ACL injury, such as females, many hypothesized that because they exhibit certain unique kinematic patterns when compared to males, these discrepancies may be the principal cause of their elevated injury risk (Boden et al., 2000; Huston et al., 2001; Malinzak et al., 2001; Chappell et al., 2002; Ford et al., 2003; Hewett et al., 2005a, 2005b; Yu et al., 2005; Brown et al., 2009; Nagano et al., 2009). For some, sagittal plane knee mechanics are perceived to be the primary cause as anterior shear of the tibia loads the ACL most directly (Markolf et al., 1995), and there are significant differences in knee flexion angles between genders as observed during jump landing (Huston et al., 2001; Malinzak et al., 2001; Chappell et al., 2007). Others have theorized knee coronal plane based injury mechanisms, citing videographic evidence of an observable valgus collapse pattern within a high number of ACL injuries captured during actual sporting events (Olsen et al., 2004; Krosshaug et al., 2007a, 2007b; Hewett et al., 2009; Quatman and Hewett, 2009). More recent reports of kinematic differences at the hip, ankle, and trunk between high and low risk populations (Griffin et al., 2000; McLean et al., 2005; Boden et al., 2009; Hewett et al., 2009) suggest that an injury mechanism may not be fully explained by differences at the knee. Given the number of intrinsic variables that can affect the in vivo mechanical environment, it is difficult to predict how complex, multi-planar loading conditions experienced during jumping and cutting motions load the ligament. The system developed in this study has the potential to address these questions.

In this study, eight male subjects performed a jumping task to simulate a basketball or soccer maneuver. Average peak relative ACL strain was 12%, which falls below the 15–19% ultimate strain threshold range reported by Butler et al. (Butler et al., 1986; Butler et al., 1992). It is interesting that peak ACL strain occurred 55 ± 14 ms prior to ground impact,

when the knee was maximally extended. Using implantable strain gauges, one previous study found that ACL elongation increases prior to ground contact during a jumping task, with peak elongation occuring at the maximum knee extension (Lamontagne et al., 2008). This pattern might suggest a strong quadriceps contraction in anticipation of ground impact, as suggested in a study by Chappell et al. (2007), who described a surge in quadriceps EMG activity around 50 ms before landing during a stop-jump task. Others have shown similar preparatory increases in quadriceps activity during various cutting and jump landing activities (Zazulak et al., 2005; Landry et al., 2009; Ebben et al., 2010). Following peak extension/strain, the knee began to flex before impact by approximately 4°, signifying an increase in hamstring co-contraction due to neuromuscular programming. The observed delay in hamstring activity is also supported by the literature as others found comparable increases in hamstring EMG data towards the terminus of the pre-contact flight cycles (Chappell et al., 2007; Landry et al., 2009). Landry et al. (2009) suggested that knee stability is maximized at ground impact during a cutting maneuver by the preprogrammed neuromuscular firing of hamstring muscles 50–100 ms prior to impact. Again, this corresponds to the peak ACL strain and extension angle observed in this study 55 ms prior to impact.

Because the maximum strain happens before impact, we can hypothesize that the neuromuscular timing of this milestone is crucial to when injury occurs. A hypothetical injury scenario could be attributed to disruptions in the timing of this critical sequence of events. For example, if an athlete were perturbed while in midair, the predetermined neuromuscular programming to land safely would be subject to change. In the current study, we detected peak ACL strains 55 ms prior to impact, when flexion angles were their lowest. This is consistent with the finding that non-contact ACL injuries most commonly occur with the knee in less than 30° of flexion (Griffin et al., 2000; Cochrane et al., 2007). Moreover, videographic analyses of real time sports injuries have determined that a significant number of non-contact injuries are associated with a perturbation prior to contact with the ground (Olsen et al., 2004; Krosshaug et al., 2007a, 2007b; Boden et al., 2009), a time when we observed higher ACL lengths. Regardless, further investigation is needed to confirm this theoretical mechanism.

One limitation of the combined method is that it was unable to be validated dynamically. This was due to limitations of our fluoroscopic system, including a small field of view and inability to measure high speed motions. Thus, a quasi-static lunge was examined in lieu of a dynamic activity. A second limitation is that the presented technique was only validated to measure ACL deformations accurately up to 45° of flexion. However, because most injuries occur at flexion angles less than 30° (Chaudhari and Andriacchi, 2006; Griffin et al., 2006; Cochrane et al., 2007; Yu and Garrett, 2007; Renstrom et al., 2008), this method would still provide useful data on ACL strains and 6DoF knee motions in the range where the ACL is thought to be susceptible to injury. Finally, strain was approximated by normalizing ACL length to the reference length measured in a relaxed position during MR imaging, where the fibers of the ACL appeared taut. It is difficult to know precisely the reference length of the ACL in vivo since it cannot support axial compression. Although there is some uncertainty with knowing the unloaded length of the ACL in our study, this measurement of relative strain is linearly related to the true strain.

Although only kinematic data on flexion angles of the knee were included with this report, it is possible for future data sets to incorporate all 6DoF motions. A complete data series that couples ACL strain with 6DoF measurements opens up the possibility for reverse engineered explanations of injury mechanisms. Instead of assuming that a set of motions loads the ACL in an unfavorable manner, an account of specific 6DoF movements that most

directly deform the ACL give a much better idea of what motions should be avoided. Such data would be paramount to better focusing and improving current prevention programs.

In conclusion, we have developed a new methodology to measure in vivo ACL strain during dynamic tasks. In this pilot study, eight male subjects performed a jumping activity similar to a basketball or soccer maneuver. The study showed that the peak ACL strain occurred 55 ± 14 ms prior to impact when ACL length was 12 ± 7% longer than an MRI based reference length. In the future, this system will be used to examine kinematic parameters that elevate ACL strain. These data will provide valuable information for developing prevention programs aimed at reducing the incidence of ACL injury.

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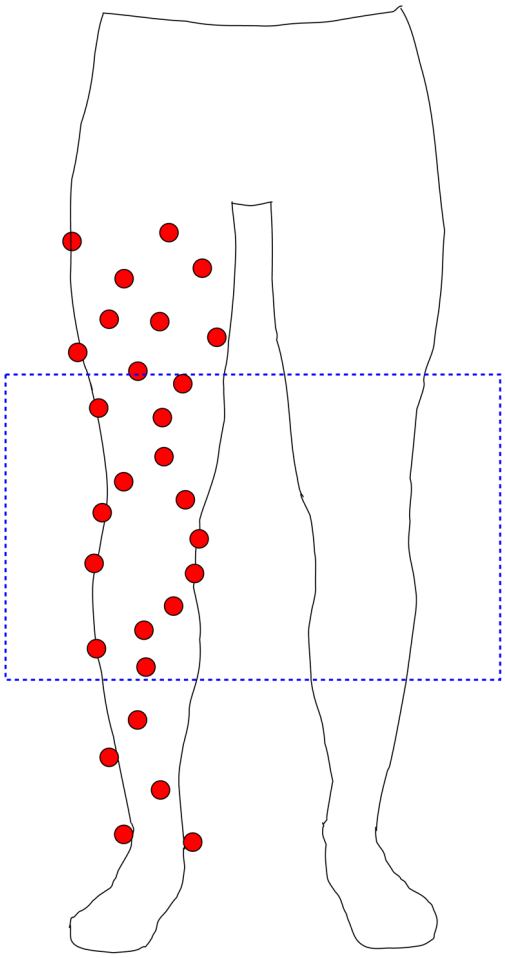

Fig. 1.

Twenty-eight markers were placed on the subject's thigh and shank. Eight markers on the distal femur and six on the proximal tibia were imaged using biplanar fluroscopy, as indicated by the box. These markers were used to position the 3D model of the femur and tibia such that the knee motion of the subject during motion analysis was reproduced.

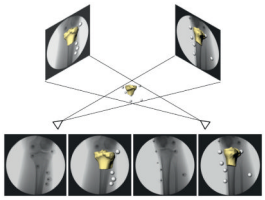

Fig. 2.
Biplanar fluoroscopy was used to register the position of the markers relative to the underlying bone. In this case, the positions of the tibial markers were related to the position of the tibia. A similar registration was performed for the femur.

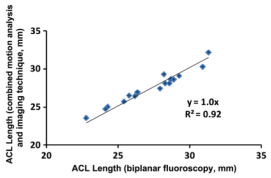

Fig. 3.

ACL length was measured in four subjects using the combined motion analysis and imaging method and compared to the length measured directly from biplanar fluoroscopy during a single-leg lunge. Linear regression indicated a high correlation coefficient and a slope of 1.0, indicating that the two methods resulted in similar measurements of ACL length. Root mean square error for all trials was 0.5 mm.

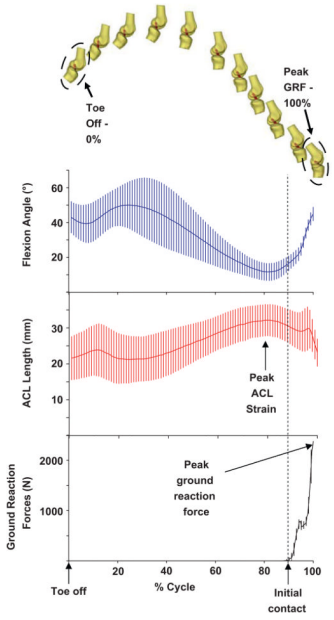

Fig. 4.

The marker data during jumping were used to position each subject's knee model, as shown for one subject (top). From each knee model, the length of the ACL (mean and 95% confidence interval) and flexion angle were measured. Data were described as a function of percentage of movement cycle (bottom) from toe-off (0%) to peak ground reaction force (100%). During the jump landing, flexion decreased initially as the subjects pushed off the platform. Flexion increased as the knee was bent to position the feet in front of the subject, and then extended to prepare for landing. Just prior to impact, flexion increased in anticipation of landing. ACL length peaked approximately 55 ± 14 ms prior to initial contact in all subjects.