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Interactions between kinematics and loading during walking for the normal and ACL deficient knee

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Abstract

The relationships between extrinsic forces acting at the knee and knee kinematics were examined with the purpose of identifying specific phases of the walking cycle that could cause abnormal kinematics in the anterior cruciate ligament (ACL) deficient knee. Intersegmental forces and moments in directions that would produce anterior–posterior (AP) translation, internal–external (IE) rotation and flexion–extension (FE) at the knee were compared with the respective translation and rotations of the tibia relative to the femur during four selected phases (heel strike, weight acceptance, terminal extension and swing) of the walking cycle. The kinematic changes associated with loss of the ACL occurred primarily during the terminal portion of swing phase of the walking cycle where, for the ACL deficient knee, the tibia had reduced external rotation and anterior translation as the knee extended prior to heel strike. The kinematic changes during swing phase were associated with a rotational offset relative to the contralateral knee in the average position of the tibia towards internal rotation. The offset was maintained through the entire gait cycle. The abnormal offsets in the rotational position were correlated with the magnitude of the flexion moment (balanced by a net quadriceps moment) during weight acceptance. These results suggest that adaptations to the patterns of muscle firing during walking can compensate for kinematic changes associated with the loss of the ACL. The altered rotational position would cause changes in tibiofemoral contact during walking that could cause the type of degenerative changes reported in the meniscus and the articular cartilage following ACL injury.

Keywords: Anterior cruciate ligament; Knee kinematics; Gait analysis

1. Introduction

The motions of the knee during walking are determined by the interaction between the passive constraints of the joint and the forces acting on the joint. Thus rupture of the anterior cruciate ligament (ACL), one of the four major ligaments of the knee, is likely to cause changes in the kinematics of the knee during walking. In particular, it could be expected that there would be changes in the anterior–posterior (AP) translation and internal–external (IE) rotation of the ACL deficient knee during specific phases of the gait cycle when there is an anterior force or internal–external moment acting at the knee, since the ACL provides primary anterior and rotational stability to the knee (Kanamori et al., 2002; Markolf et al., 1978; Shoemaker and Markolf, 1982).

The AP translation and IE rotation are particularly important for normal knee function even during routine activities such as walking (Andriacchi et al., 1998; La Fortune et al., 1992). Alterations or a shift in AP translational and IE rotational motion (Blankenvoort et al., 1988) of the knee during dynamic activities could have profound effects on secondary restraints such as the medial meniscus. Clinical reports (Buckland-Wright et al., 2000; Daniel et al., 1994; Lohmander and Roos, 1994; Roos et al., 1995; Scott et al., 1979) of the presence of osteoarthritis of the knee in patients with ACL injury suggest that abnormal motion at the knee could be a cause of degenerative changes. Yet, increased passive clinical laxity based on quantitative measurement has not correlated with clinical outcome (Snyder Mackler et al., 1997). The variable clinical results could be related

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to the fact that muscle contraction can generate forces in directions tending to produce AP translation and IE rotations of the knee (Andriacchi, 1990; Beynnon and Fleming, 1998; Ciccotti et al., 1994; Li et al., 1999). Thus, muscle contraction can either produce dynamic stability or produce potentially harmful displacements for the ACL deficient knee. The modification in function identified in some patients with ACL deficient knees (Berchuck et al., 1990; Ciccotti et al., 1994; Vergis and Gillquist, 1998; Wexler et al., 1998) could explain the variable clinical outcomes of patient with ACL injury. However, it has not been shown that alterations in patterns of walking influence the alterations in the AP and IE motion of the ACL deficient knee.

Thus, understanding how the ACL influences the AP and IE motions of the knee during walking is an important requirement for addressing the problem of secondary degenerative changes reported following ACL injury. A fundamental step towards understanding the functional role of the ACL during walking is to identify specific phases of the walking cycle that could cause abnormal kinematics in the ACL deficient knee relative to healthy knees. In particular, a better understanding of the relationship between the AP forces and IE moments and corresponding AP translations and IE rotations at the knee during walking is needed. Further, since force generated by muscle contraction can influence the AP and IE position of the knee, it is important to evaluate the influence of altered patterns of muscle forces on the kinematics of the knee.

The purpose of this study was to test the relationship between extrinsic forces acting at the knee and knee kinematics for the healthy knee with the goal of identifying specific phases of the walking cycle that could cause abnormal kinematics in the ACL deficient knee.

2. Methodology

Patients were selected with a complete rupture of the ACL (documented by examination and MRI) and minimal associated injury to the other major ligaments of the knee joint, articular surface or menisci. A total of 27 knees in 18 subjects were evaluated during walking. There were 9 subjects with an average age of 41 ± 10 years, height 1.7 ± 0.1 m and weight 747.8 ± 196 N and a unilateral ACL deficient knee. The average time past injury was 127 ± 142 months with a range from 3 to 400 months. The 9 contralateral knees of the ACL deficient population were used as a control. Nine knees were randomly selected from a population (33.6 ± 16 years, 1.7 ± 0.1 m, 703 ± 155 N) with bilateral normal knees as a control. All subjects were tested (following informed consent using an IRB approved protocol) during walking.

The instrumentation included a four camera video-based opto-electronic system for 3-D motion analysis, a multi-component force plate for measurement of foot-ground reaction force and a computer system for acquisition, processing and analysis. The hypothesis of this study required testing the relationship between three kinematic parameters (AP translation, IE rotation and flexion–extension) with the corresponding intersegmental forces and moments.

Intersegmental forces and moments were measured using previously described methods (Andriacchi et al., 1997). The leg was idealized as a 3-D linkage with the intersegmental forces and moments at the knee resolved into a coordinate system fixed in the tibia. Forces were normalized to the subject's weight and expressed as a percentage of body weight (%bw) and moments were normalized to the product of weight and height and expressed as a percentage of body weight times height (%bw*ht). The kinematic measurements were derived from 21 light-reflective markers arranged on the two limb segments, creating separate cluster groups of 11 markers on the thigh and 10 on the shank. As previously described (Andriacchi et al., 1998), skin motion artifact was detected by comparing the eigenvalues from each cluster at each time step to the eigenvalues calculated when the subject was in a static reference position. The six-degrees-of-freedom motion of the knee was obtained by first resolving the clusters on the thigh and shank into two orthogonal sets of axes that created the cluster coordinate systems (thigh and shank). These cluster coordinate systems were then related to anatomical coordinate systems based upon palpable bony landmarks (Hoppenfeld and Huton, 1976). The anatomic femoral coordinate system was located at the midpoint of the transepicondylar line of the distal femur and the anatomic tibial coordinate system was set at the midpoint of a line connecting the medial and lateral points of the tibial plateau. The AP translation was determined by calculating the displacement between the origins of the tibial coordinate system relative to a femoral coordinate system projected onto the AP axis of the tibia (Fig. 1). IE rotations were measured by projecting the medial–lateral femoral axis onto a plane created anterior–posterior and medial–lateral axes fixed in the tibia.

The analysis divided the walking cycle into four phases: Heel Strike (HS); from initial foot contact to 10% of the gait cycle; Weight Acceptance (WA); from 10% to 30% of the gait cycle; Terminal Extension (TE); from 30% to toe-off of the gait cycle; and Swing Phase; from toe-off to heel strike. Relative extrema of each of the kinematic and kinetic variables were quantified during each of the four phases as illustrated in the typical curves shown in Fig. 2a and b. The directions of the kinematic and kinetic parameters were compared to test if motion was in the same direction as the

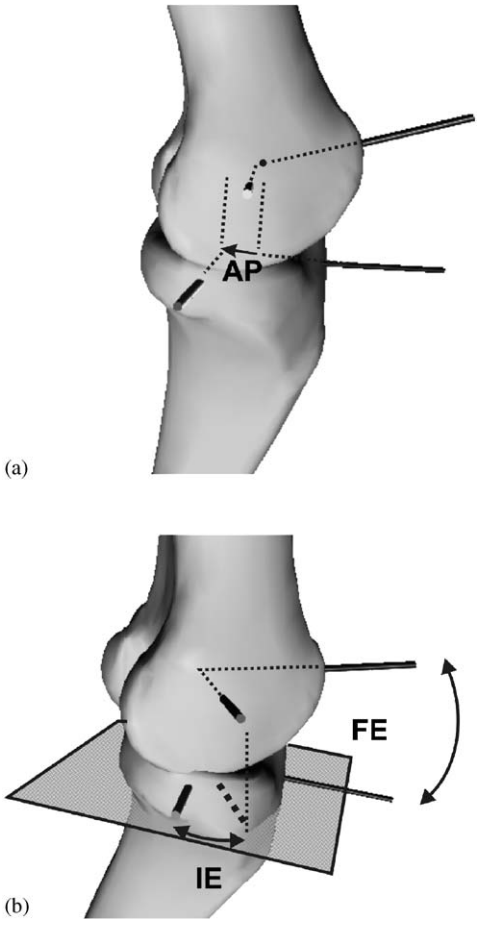

Fig. 1. The AP displacement of the tibia was determined by the displacement of the origin of the tibial coordinate system on the AP-axis of the tibia. Tibial rotation was determined by the projection of the medial–lateral (ML) axis of the tibia onto the ML–AP plane in the femoral reference frame. The anatomic femoral coordinate system was located at the midpoint of the transepicondylar line of the distal femur and the anatomic tibial coordinate system was set at the midpoint of a line connecting the medial and lateral points of the tibial plateau.

intersegmental force. The average position of the tibia relative to the femur during the walking cycle was calculated by calculating the area under the IE and AP curves divided by the time of the walking cycle. An offset between the average IE and AP position was calculated as the difference between the ACL deficient and contralateral knees. Finally, the influence of the muscle generated moments was evaluated by testing the correlation using a linear regression model, between the moment tending to produce flexion–extension at the knee and the average position of the IE and AP curves. Differences between contralateral and ACL deficient knees were tested using a paired Student's *t*-test with an $\alpha = 0.05$ to determine significance between the ACL deficient knee and the contralateral knee. The contralateral knee was also tested relative to the normal population.

3. Results

There were common temporal characteristics of the IE, AP and flexion–extension kinetic and kinematic parameters during walking for ACL deficient and healthy knees. In particular, the extrema for each of the parameters were located in the same phase of the gait cycle for all subjects.

There was a correspondence between the direction of the intersegmental force and motion through all of stance phase. The extrema of the AP and IE displacements during the stance phase of walking were in the same direction as the corresponding components of the intersegmental forces or moments. The relationship between the kinematic and kinetic parameters for the normal knee was characterized during four phases of the gait cycle (Fig. 2a and b) as follows:

Heel strike phase: During the heel strike phase, the AP component of the intersegmental force (F^{ant}) at the tibia had a maximum of $13.2 \pm 1.9\%$ bw in the anterior direction and the tibia reached a maximum anterior position (AP^{ant}) of 16.3 ± 8.3 mm at heel strike.

AP^{ant} also reaches a maximum at the end of swing phase (Fig. 2b). The rotational torque had a maximum (Tq^{ext}) of $0.3\% \pm 0.1$ bw∗ht in a direction tending to produce external rotation while the tibia was near a maximum external rotation. The knee was at maximum extension (Fl^{min}) of $2.9 \pm 0.5^\circ$ while the flexion–extension moment reached a maximum (M^{ext}) of $3.5 \pm 1.2\%$ bw∗ht in a direction tending to extend the knee.

Weight acceptance phase: During the weight acceptance phase, the AP component of the intersegmental force was in a posterior direction and the tibia moved in a posterior direction. The flexion–extension moment had a maximum (M^{flex}) of $3.6 \pm 1.0\%$ bw∗ht in a direction tending to flex the knee while knee flexed to a relative maximum (Fl^{wa}) of $22.1 \pm 4.6^\circ$.

Terminal extension phase: During the terminal extension phase, the AP component of the intersegmental force reached a maximum (F^{post}) of $36.3 \pm 4.5\%$ bw in a posterior direction while the tibia continued to translate posteriorly AP^{te}. The rotational torque reached a maximum (Tq^{int}) of $1.1 \pm 0.2\%$ bw∗ht, while the tibia continued to internally rotate to a maximum internal rotation of $3.2 \pm 5.7^\circ$. The flexion–extension moment reached a relative maximum $2.2 \pm 0.8\%$ bw∗ht in a direction tending to extend the knee while the M^{ext2} of tibia reached a relative minimum flexion (Fl^{te}) of $5.7 \pm 4.2^\circ$.

Swing phase: During swing phase, the tibia reached a maximum AP position (AP^{post}) of 20.3 ± 8.9 mm in a posterior direction and maximum external rotation (IE^{ext}) of $13.0 \pm 4.3^\circ$. In addition, knee flexion reached a maximum of $62.8 \pm 4.8^\circ$ during swing phase. At the end of swing phase, the knee extended as the tibia

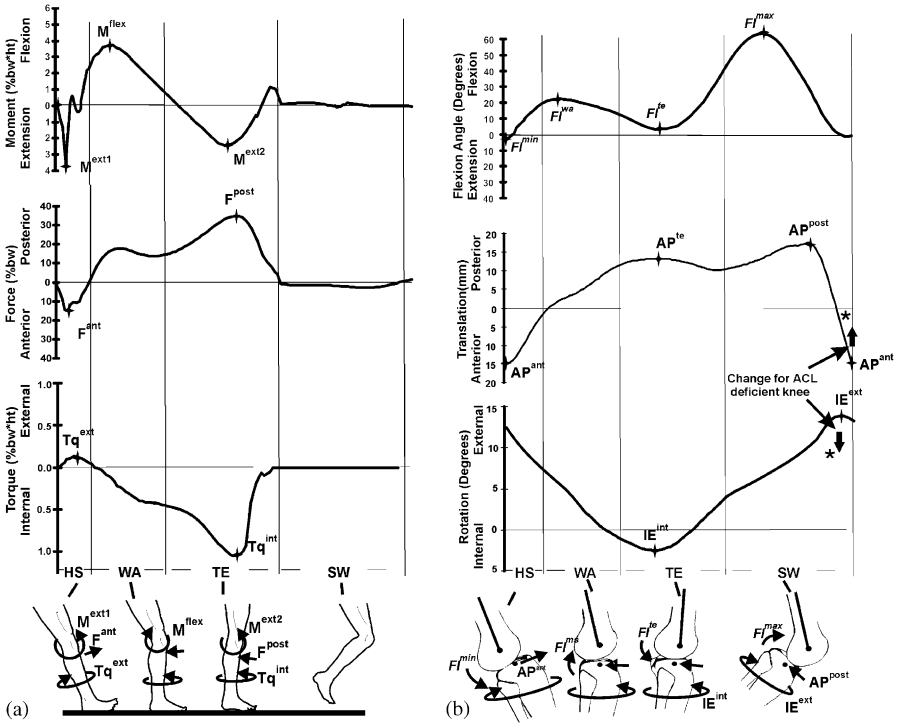

Fig. 2. (a and b) The analysis divided the gait cycle into four phases (HA, WA, TE and SW). The extrema of intersegmental anterior–posterior force (F^{ant}, F^{post}) were in the same direction as the corresponding displacements (AP^{ant} and AP^{te}) during stance phase. The extrema of intersegmental internal–external moment (Tq^{ext} and Tq^{int}) were in the same direction as the corresponding rotations (IE^{ext} and IE^{int}) during stance phase. The extrema of the displacements were in the same phase of the gait cycle for all subjects. The arrow* indicates the direction of significant changes for the ACL deficient knee.

externally rotated and translated to its maximum anterior position AP^{ant} just prior to heel strike.

ACL deficient vs. *healthy knee*: There were kinematic differences between ACL deficient knee and the contralateral healthy side for specific phases of the walking cycle (Fig. 2b). During the terminal portion of swing phase the average maximum anterior displacement (AP^{ant}) in the ACL deficient knee (10.7 ± 4.6 mm) was significantly less ($p < 0.05$) then the average AP^{ant} of the contralateral healthy knee (16.3 ± 8.3 mm). During swing phase the average maximum external rotation (IE^{ext}) in the ACL deficient ($8.3 \pm 3.4^{\circ}$) was significantly less ($p < 0.05$) then the healthy contralateral side (13.0 ± 4.3). There was no significant difference between the kinematics of the contralateral side of the ACL deficient knee and knees from subjects with bilateral normal knees.

The average position of the ACL deficient knee during the gait cycle was significantly ($p < 0.05$) offset towards internal rotation ($2.9 \pm 4.0^{\circ}$) relative to the contralateral healthy knee. The magnitude of the rotational offset was significantly correlated ($p < 0.05$) with magnitude of the flexion moment during weight acceptance (M^{flex}), demonstrating an increase in the internal rotation offsets for the ACL deficient knee increased as the magnitude of the flexion moment increased during the weight acceptance phase of the gait cycle (Fig. 3). While there was a only a trend towards a significant posterior offset in the ACL deficient knee, there was a significant correlation between the offset of the AP position of the tibia towards a more posterior position with increased flexion moment during weight acceptance (Fig. 3).

It is interesting to note that patients that had a lower than normal magnitude of the flexion moment M^{flex} ($3.5 \pm 1.2\%$ bw*ht) had the offset in the AP and IE positions approach normal values. M^{flex} varied substantially more in the ACL deficient population (coefficient of variation = 53%) than the normal subjects (coefficient of variation = 29%). The average of the AP or IE curve offsets for the normal or contralateral side of the ACL deficient knees were not significantly correlated with the flexion moment M^{flex} for either the IE or AP motions.

4. Discussion

The results of this study provide new insights into the functional role of the ACL as well as the relationship between knee kinematics and the forces that act at the knee during walking. In particular, during swing phase the ACL deficient knee had reduced external rotation as the tibia extended prior to heel strike, suggesting a loss of the normal screw home movement (Hallen and

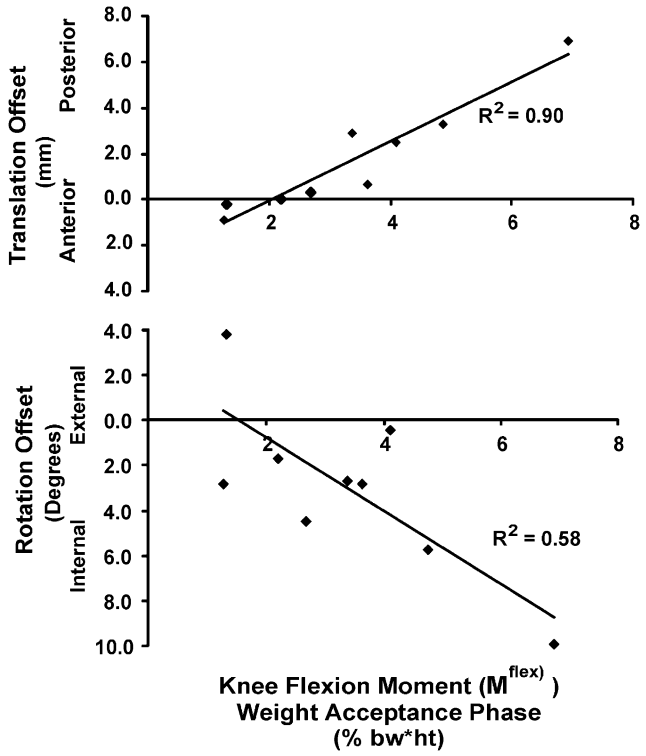

Fig. 3. The offsets in the average AP and IE position of the tibia in the ACL deficient knee relative to the contralateral knee were correlated to the maximum flexion moment (M^{flex}) during weight acceptance. Patients that adapted a gait pattern with lower flexion moments had less offset from the normal AP and IE position.

Lindahl, 1966). The loss of screw home movement at the end of swing phase produced an offset towards internal rotation in the average position of the tibia relative to the healthy knee that was maintained through stance phase. The reduced anterior displacement of the ACL deficient knee at heel strike also appears to be related to modified kinematics during terminal swing phase. There is normally substantial anterior tibial translation during terminal swing phase. The reduced anterior translation in the ACL deficient knee that occurs at heel strike appears to be related to the reduced external rotation at the end of swing phase. Thus, the transition between swing phase and stance is an important consideration in evaluating the ACL deficient knee (Beynnon et al., 2002).

The offsets of the average rotational position for the ACL deficient knee support the observation that loss of the ACL can change the normal position of the tibiofemoral contact (Ma et al., 2000). A shift in the normal tibiofemoral position could explain the counter intuitive finding of reduced anterior position of the tibia during heel strike in subjects with ACL deficient knees since the displacement would take place from a position that is more internally rotated than normal.

The correlation between offsets from the AP and IE positions with flexion moment (balanced by a net quadriceps moment), indicated that adaptations to the patterns of muscle firing could compensate for the loss of the ACL (Andriacchi, 1990; Ciccotti et al., 1994; Draganich et al., 1987; Li et al., 1999). The correlation (Fig. 3) demonstrated that the offset from normal of the tibiofemoral position increased with an increase in the magnitude of the flexion moment. Thus by adapting a gait pattern with a lower flexion moment during weight acceptance (M^{flex}) the ACL deficient patient can achieve a more normal tibiofemoral position during walking. The fact that there was substantially more variation in (M^{flex}) for the ACL deficient knees compared to normal knees suggests that all patients do not develop the same adaptive strategies following ACL injury. It has been reported that time past the index injury can influence the development of gait alterations following ACL injury (Wexler et al., 1998).

The potential for changes in muscle firing patterns to alter the offset in the normal tibiofemoral contact position has important implications for the secondary changes to meniscus (Bellabarba et al., 1997; Cipolla et al., 1995) and cartilage (Lohmander and Roos, 1994) in the ACL deficient knee. Clinical and laboratory studies (Kvist and Gillquist, 2001; Wu et al., 2000; Yao and Seedhom, 1993) have suggested that changes in contact position could be a factor in the degenerative changes reported in patients with ACL deficient knees. In particular, rotational malalignment has been related to an increased incidence of knee osteoarthritis (Nagao et al., 1998; Yagi and Sasaki, 1986). The change in the rotational position at the knee could cause specific regions of the cartilage to be loaded that were not loaded prior to the ACL injury (Brandsonn et al., 2001). Thus, it could be possible to reduce the rate of secondary changes following ACL loss by adapting patterns of locomotion that reduce the positional offsets of tibiofemoral rotation.

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