

# Gait Deviations of Anterior Cruciate Ligament Ruptured Patients : A cross-sectional gait analysis study

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## Research article

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# Abstract

**BACKGROUND** Anterior cruciate ligament (ACL) rupture causes instability and can result gait deviations. The purpose of this study was to find kinetic and kinematic deviations of ACL-ruptured knees by comparing them to their contralateral un-injured knee in the sagittal plane.

**METHODS** Gait analysis was performed on 36 males with acute complete ACL rupture who were functionally classified as non-copers. Kinetic and kinematic data were collected at comfortable gait speed after 3 months of conservative treatment. The average time interval between the injury and gait measurement was 4 months. Data from the ACL-ruptured knee and the contralateral un-injured knee were compared.

**RESULTS** The maximal value (27% smaller,  $p=0.002$ ) and peak-to-peak amplitude ( $p<0.001$ ) of the knee extension moment (KEM) were both smaller in the ACL-ruptured knee. Two distinctive kinematic patterns were observed. First, at the initial stance phase, knee flexion angle was smaller in ACL-ruptured knee. Second, during the next mid-to-late stance phase, knee flexion angle was bigger in ACL-ruptured knee (both  $p<0.001$ ). First pattern was correlated with the maximal KEM and second was correlated with the KEM peak-to-peak amplitude (both  $p<0.001$ ).

**CONCLUSIONS** Two distinct kinematic deviations were found on ACL-ruptured knee and it was associated with the peak and amplitude of KEM. It suggests that kinematic control of knee joint is important gait deviation mechanisms of patients with ACL rupture.

## 1. Background

ACL is primary restraint of anteroposterior and secondary restraint of rotational instability. Rupture of ACL results instability of the knee<sup>1,2</sup> and instability can affect kinematics and kinetics of knee joint<sup>3-12</sup>.

Historically, gait deviation of ACL rupture patient was first reported by Berchuck et al. in 1990<sup>6</sup>. They proposed a 'quadriceps avoidance gait' wherein patients with ACL rupture walk with reduced quadriceps activation to decrease anterior tibial translation. It is the most cited gait adaptation mechanism of ACL rupture patients. However, as research continued, counter arguments were raised<sup>5-11,13</sup>. Firstly, several researchers could not find the quadriceps avoidance gait pattern in their group of patients<sup>7,9,12,13</sup>. Secondly, knee was extended at the initial stance phase, which was not explained by the quadriceps avoidance<sup>8,10,14,15</sup>. If the quadriceps avoidance happens, the knee should be in a flexed position at the loading phase due to the antagonistic hamstring muscle. It is worthwhile noting that the methodology among studies are inconsistent. Patient selection criteria, gait exam timing, diagnostic method was varied among the studies and conclusion was inconsistent. Currently, the presence of quadriceps avoidance gait as well as the gait adaptation mechanisms after ACL rupture is inconsistent in the literatures<sup>3-13</sup>.

Information regarding these subjects may provide further insight to the neuromuscular strategies of patients with ACL rupture, in addition to shedding light on the pathophysiology by which arthritis tends to develop in ACL-deficient knees. Therefore, in the present study we performed a comprehensive analysis of knee biomechanics using principal component analysis (PCA). Unlike simple comparison of maximal or minimal values, PCA can analyze a large matrix of data without loss of information.

In this study, we tested the hypothesis that kinetic and kinematic deviations of ACL rupture exist and that these deviations may act as a stabilization strategy. The purpose of this study was to find kinetic and kinematic deviations of ACL-ruptured knees by comparing them to their contralateral un-injured knee in the axial and sagittal planes.

## 2. Methods

### 2.1 Participants

This cross-sectional gait analysis study was conducted on 114 consecutive patients with complete unilateral ACL rupture (Fig. 1). ACL rupture was diagnosed clinically and confirmed by MRI imaging. To minimize possible confounding effects<sup>3,4,16</sup>, patients meeting the following criteria were excluded: 1) chronic ACL tear (more than 6 months at first visit) (N = 10); 2) female sex (because of sex differences in gait properties<sup>17-20</sup>) (N = 9); 3) combined fracture or knee dislocation (N = 6); 4) aged more than 45 years (N = 5); 5) evidence of radiologic osteoarthritis (Kellgren-Lawrence grade II or more) (N = 5); 6) combined bucket-handle tear of meniscus (N = 4); and 7) any prior surgery in the lower extremity (N = 2). After application of the exclusion criteria, 73 eligible patients remained. Initially, the patients were treated conservatively through physical therapy and strengthening exercise. After at least 3 months of conservative treatment, more than half of the patients (40 patients) complained of instability or giving-way, even though they had significantly reduced their activity level prior to injury. We defined these patients as “non-copers” in this paper and included them in the analysis<sup>21</sup>. The non-copers also underwent ACL reconstruction surgery after this study. Of the non-copers, four declined or failed to participate in the study. Therefore, 36 males with unilateral ACL rupture who were functionally classified as non-copers participated in this study. The mean subject age was 27.1 years ( $\pm 7.0$ , range, 19–45 years), the mean height was 172 ( $\pm 7.2$ ) cm, and the mean weight was 71.5 ( $\pm 11.2$ ) kg. The average range of motion (ROM) prior to gait lab analysis was 138.7° ( $\pm 15.8$ ). The average Lysholm score was 67.7 ( $\pm 12.7$ ). The pivot shift test results were Gr I for 11 patients, Gr II for 17 patients, and Gr III for 6 patients. The anterior draw test results showed Gr I for 14 patients, Gr II for 17 patients, and Gr III for 5 patients.

### 2.2 Data collection

Gait data were measured in the Human Motion Analysis Lab at our institution. The average time interval between the injury and gait measurement was 4 months (range: 3 months – 8 months). Before gait measurements, subjects had to meet the following criteria: minimal knee effusion, no knee extension

deficit, minimal pain in the injured limb with walking, and no visually identifiable gait impairments. The average pain score as assessed on a numeric rating scale at the time of gait analysis was 1.2 ( $\pm 0.8$ ). Therefore, we could exclude any acute effects of knee trauma because of the criteria above. Patients were asked to perform 5 min of easy walking to warm up. After warming up, reflective markers from a Helen Hayes marker set were placed on each subject's body<sup>22</sup>. Patients were asked to walk at their usual speed along a 9 m track.

Motion (kinematic) data were acquired at a sample rate of 120 Hz using 12 charge-coupled device cameras equipped with a three-dimensional optical motion capture system (Motion Analysis, Santa Rosa, USA). Ground reaction force (kinetic) data were acquired at a sampling rate of 1200 Hz using three AMTI (Advanced Mechanical Technology Inc., Watertown, MA, USA) force plates. The kinetic data were then normalized by height and weight (% body weight  $\times$  height)<sup>23</sup>.

We used Eva Real-Time software (Motion Analysis, Santa Rosa, USA), Microsoft Excel 2016 (Microsoft, Redmond, USA), and MATLAB R2017a (Mathworks, Natick, MA, USA) for real-time motion capture, post-processing and marker data tracking. The average of three representative strides from five or six separate trials was used for the analysis of each session. Right-sided data were used in the analysis. A total of four gait parameters were measured including knee flexion angle, internal knee extension moment (KEM), knee rotation angle, and knee rotation moment.

## 2.3 Statistical analysis

Peak (maximum) kinetic and kinematic data of ACL-ruptured and un-injured limbs were compared using paired Student's t-tests. Four gait parameters were processed via principal component analysis (PCA). PCA was performed via two steps. First, features were extracted from each gait parameter and second, each feature was scored for each subject. Each gait parameter consisted of an  $n \cdot 101$  data matrix where  $n$  rows represent the number of cases and 101 columns represent standardized 101 time points. PCA processes this gait parameter matrix with an orthogonal transformation and extracts the data into a set of gait features that are linearly uncorrelated (principal components, PCs). These transformations allow the major features (PCs) of each gait parameter to be recognized. PCs were selected using a 90% trace criteria and 7 features were extracted from 2 gait parameters (i.e. KEM and knee flexion angle). The next step was the scoring of the extracted features. This was obtained by standardizing individual contributions to the extracted features<sup>24</sup>. In the case of a high score, the standardized individual contribution shows the same direction as the extracted feature. Conversely, when the score is low or negative, it indicates the opposite direction. The PC score is a standardized score (mean = 0, standard deviation = 1) that can be compared to other subjects or among the features. The standardized mean difference (SMD) of each of the PC scores was compared between the ACL-ruptured and un-injured knees. The feature that showed the highest SMD of the PC score was then investigated.

The PC scores of ACL-ruptured and un-injured limbs were compared using paired Student's t-tests. The normality of each PC score was assessed with the Kolmogorov-Smirnov test. All statistical analyses were

performed using SPSS® 19.0.1 for Windows® (SPSS Inc., Chicago, IL, USA). P-values < 0.05 were considered to indicate statistical significance.

### 3. Results

The biggest kinetic difference between the ACL-ruptured and un-injured knees was the peak-to-peak amplitude (i.e. difference between peak and trough) of KEM (Table 1, Fig. 2-a). It was smaller in ACL-ruptured knee and the SMD was 1.02 ( $p < 0.001$ ). The peak KEM in ACL-ruptured knee was 27% smaller than un-injured knee ( $p < 0.002$ , ACL-ruptured: 2.5 (%Bw\*Ht), un-injured: 3.4 (%Bw\*Ht)).

Two distinctive kinematic patterns were observed in knee flexion angle (Table 1, Fig. 2-c). Patients extended their ACL-ruptured knee more at the IDS (initial double limb stance) phase and then flexed more during the SLS (single limb stance) to the TDS (terminal double limb stance) phase (both  $p < 0.001$ ) compared to the contralateral un-injured knee (black and red arrow in Fig. 2-c).

We subsequently investigated the association between kinetic and kinematic parameters. At the IDS phase (i.e. loading phase), the peak knee flexion angles of the ACL-ruptured knees and un-injured knees were 17.1° and 20.2°, respectively ( $p < 0.001$ ). This angle was positively correlated with the peak KEM (Pearson  $r = 0.694$ ,  $p < 0.001$ ) (Fig. 3). In addition, knee flexion angle at SLS to TDS (i.e. KF PC3) was higher in ACL-ruptured knees ( $p < 0.001$ ) and was negatively correlated with the KEM amplitude (i.e. KEM PC2) (Pearson  $r = -0.710$ ,  $p < 0.001$ ) (Fig. 4).

Cumulatively, these data indicate that patients adopted two distinctive kinematic strategies to reduce KEM peak and amplitude of their ACL-ruptured knee. In the first strategy, patients extended their ACL-ruptured knee at the IDS phase to reduce the peak KEM. In the second strategy, patients flexed their ACL-ruptured knee at the SLS-TDS phase to reduce the KEM amplitude. Linear regression analysis showed that the adjusted  $R^2$  of the first strategy was 0.475 and that of the second strategy was 0.497 (Table 2).

### 4. Discussion

The most important findings of this study are that we found two distinct kinematic deviations on ACL-ruptured knee, and it was associated with the peak and amplitude of KEM. It suggests that kinematic control of knee joint is important gait deviation mechanisms of patients with ACL rupture.

Gait deviations of ACL-rupture patients have been reported in the literature. In 1990, Berchuck and colleagues described the quadriceps avoidance gait<sup>6</sup>. This study became a benchmark for studying the gait characteristics of patients with ACL rupture<sup>6</sup>. However, the presence of quadriceps avoidance gait has been debated. Further studies analyzed the existence of quadriceps avoidance gait by examining muscle strength and EMG<sup>5,16</sup>. However, kinematic control has not been widely studied, even though this type of control is one of the main means of neuromuscular control in patients with ACL rupture.

In this study, we confirmed the decreases in KEM amplitude and peak. In addition, we described the kinematic control of gait and showed that it is associated with kinetics. To our knowledge, this is the first study to describe two distinctive kinematic controls associated with kinetics in patients with ACL rupture. As described previously, Berchuck et al. found that the KEM was decreased and sometimes even reversed in the mid-stance walking phase of patients with ACL rupture, terming this phenomenon “quad avoidance gait”<sup>6</sup>. However, future studies did not yield consistent results. Studies by Georgoulis et al. (and others) found no difference in sagittal-plane kinematics, while studies by Hurd et al. (and others) found significant KEM reduction<sup>3-13</sup>. More recent reports with delicate study designs found that patients with ACL rupture had a reduced KEM, although the KEM was still greater than zero and was not reversed, as in the original article<sup>5,8,13</sup>. We found that studies that did not find reduction of KEM used different patient selection criteria and/or different gait exam timing. For example, Georgoulis and colleagues performed the gait exams on average at  $7.6 \pm 4.3$  weeks after ACL rupture and did not classify patients as copers or non-copers<sup>12</sup>. Furthermore, Berchuck et al. found normal biphasic patterns in 25% of the analyzed patients, suggesting that pattern results may vary according to patient selection criteria<sup>6</sup>. In the present work, we excluded females, copers, and final acute/chronic ACL rupture to minimize these possible confounding effects. As described above, gait features differ according to sex<sup>17-20</sup>. Acute ACL rupture can result in antalgic gait, whereas chronic ACL rupture can result in arthritic gait features<sup>3,12</sup>. Proper control and selection of the patient group is very important. Based on our findings, we conclude that KEM is decreased in patients with ACL rupture.

Another important question is the mechanism by which the KEM decreases. The most common interpretation is direct inhibition of the quadriceps femoris<sup>5,16</sup>. The KEM is generated by eccentric contraction of the quadriceps with a moment opposite to the KFM, which acts as an external flexion force in the loading phase. The KEM has the same size as the KFM, but the opposite mechanical balance. During gait, the KEM can act as an anterior translation force for the proximal tibia, thereby unconsciously suppressing the quadriceps in the ACL rupture<sup>5,6,16</sup>. In support of this hypothesis, studies using EMG have shown that quadriceps muscle activity is suppressed<sup>15,16</sup>. In addition, increased hamstring activity is associated with this suppression. This increase in muscle activity is referred to as muscle co-activation; both of these phenomena are considered major neuromuscular adaptations in patients with ACL rupture<sup>5,14-16,25</sup>. However, quadriceps avoidance cannot fully explain the phenomenon of ‘knee extension in the IDS phase’. The hamstrings and quads are antagonistic to each other; therefore, if quadriceps avoidance occurs throughout the gait cycle, the knee should always be more flexed compared to the opposite side. Although this explanation is consistent with the knee flexion phenomenon from the SLS to the TDS phase<sup>8,15</sup>, it is inconsistent with previous studies that reported knee extensions at the IDS phase and with our results<sup>8,10,14,15</sup>. Based on this inconsistency, we infer that another mechanism exists at the IDS phase. We investigated the relationship between peak KEM and peak knee flexion angle in the IDS phase and found a strong linear relationship (Pearson  $r = 0.694$ ,  $p < 0.001$ ). Therefore, knee extension in the IDS phase reduces the KEM. Extension of the knee in the IDS phase has been observed in previous studies, but was not previously interpreted mathematically as in the present study<sup>3,5,10</sup>. These results suggest

that both kinematic control and muscular control may be associated with the gait of patients with ACL rupture. Patients with ACL rupture extend their knee in order to reduce the KEM at the IDS phase. When the knee is further extended, the transverse vector decreases, reducing the force applied to the anteroposterior direction of the tibia<sup>6</sup>. However, this reduction is expected to increase the GRF distribution in the axial direction (instead of reducing the transverse vector). This increased GRF distribution may increase the impact on the TF joint and may also contribute to the development of TF arthritis or subsequent meniscus injury after ACL rupture<sup>10,11</sup>. We conclude that at the early part of the IDS phase, kinematic control of knee extension is more important than the quadriceps avoidance strategy. This phenomenon likely corresponds to a feed-forward (central control) mechanism of knee joint neuromuscular control<sup>26</sup>. This strategy could be a coordinated way to reduce peak KEM in the early stages through feed-forward signaling at the IDS phase (by kinematic control).

After the IDS phase, the walking strategy from the SLS to the TDS phase was similar to the later quadriceps avoidance pattern and stiffening strategy described by Hurd et al<sup>5,6,8,15</sup>. The quadriceps avoidance mechanism or stiffening strategy (based on increased muscle co-activation) is likely to work here. However, the knee stiffening strategy in the SLS-TDS phases seems to affect the amplitude rather than the KEM peak value. When the two walking strategies were modeled by regression analysis (Table 2), the adj R2 values were 0.475 and 0.497. These correlations could account for significant portions of the KEM peak and amplitude. The rest of the KEM peak and amplitude are likely to be due to direct inhibition or muscle coactivation, which were not included in this study but which have been observed by others<sup>5</sup>.

This study has some limitations. First, participants were restricted to noncoping men, which limits the degree to which the results of this study can be applied to other groups. In this regard, women are known to have greater rotational laxity than men; therefore, the results may be different in women<sup>17-20</sup>. However, since men and women have different gait patterns and skeletal differences, analyzing men and women together without controls could make the results harder to interpret<sup>17-20</sup>. Future research should focus on women. Also, only non-copers were tested. However, analysis of the gait pattern of copers is not as important as that of non-copers at present. Moreover, analyses that fail to discriminate non-copers from copers could lead to incorrect conclusions being drawn. Secondly, the gait and clinical tests were performed between 3 and 8 months after the injury, meaning that pain and stiffness may have affected gait. However, before gait measurements, each participant was verified to have minimal knee effusion, no knee extension deficits, minimal pain in the injured limb with walking, and no visually identifiable gait impairments. These criteria were applied to minimize the effects of pain and stiffness. The average pain numeric rating was  $1.2 \pm 0.8$  and the average range of motion (ROM) prior to gait analysis in the laboratory was  $138.7 \pm 15.8^\circ$ . However, the gait patterns of patients with acute or chronic ACL ligament rupture may be different; therefore, further studies are needed<sup>3,12</sup>.

## 5. Conclusions

Patients showed two distinct kinematic deviations to reduce the KEM peak and amplitude of their ACL-ruptured knee. It suggests that kinematic control of knee joint is important gait deviation mechanisms of patients with ACL rupture.

## 6. Abbreviations

ACL anterior cruciate ligament

EMG electromyography

GRF ground reaction force

IDS initial double limb stance

KEM knee extension moment

MRI magnetic resonance imaging

PC principal component

PCA principal component analysis

ROM range of motion

SLS single limb stance

SMD standardized mean deviation

TDS terminal double limb stance

## 7. Declarations

### ***7.1 Ethics approval and consent to participate***

This study was approved by the Seoul National University College of Medicine/Seoul National University Hospital Institutional Review Board (IRB No.: H-1706-177-864). Each participant gave written, informed consent to participate.

### ***7.2 Consent for publication***

Not applicable

### ***7.3 Availability of data and materials***

Not applicable

#### ***7.4 Competing interests***

Not applicable

#### ***7.5 Funding***

Not applicable

#### ***7.6 Authors' contributions***

DR designed the study and wrote the paper. MC, TK and CY acquired and interpreted the data. JL and HH performed the statistical analysis and JL drafted the manuscript. And ML designed the study and revised the paper.

All authors have given approval of the submitted version of the manuscript and agree to be accountable for all aspects of the work.

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## Tables

Table 1. Principal component analysis of kinetics and kinematics

Gait measure	PC	Feature	% of variance	SMD	p-value*
			(cumulative)	(N=36)	
a) knee extension moment (KEM)	PC1	Overall magnitude	68.5	0.15	0.542
	PC2	Peak-to-peak amplitude	89.8	<b>1.02</b>	<b>&lt;0.001</b>
	PC3	Phase shift	95.6	0.11	0.483
<b>Interpretation: less amplitude of moment (of ACL ruptured knee)</b>					
b) knee flexion angle	PC1	Overall flexion angle	56.8	0.3	0.069
	PC2	Flexion at swing phase	75.9	0.26	0.157
	PC3	Flexion at SLS to TDS phase	88.2	<b>0.87</b>	<b>&lt;0.001</b>
	PC4	Flexion at IDS phase	94.8	<b>0.76</b>	<b>&lt;0.001</b>
<b>Interpretation: extension at IDS phase and more flexion at SLS to TDS phase (of ACL ruptured knee)</b>					

\*Comparisons were made with paired t-test. Bold face indicates statistical significance.

PC: principal component, SMD: standardized mean difference, IDS: initial double limb support, SLS: single limb support, TDS: terminal double limb support, IR: internal rotation

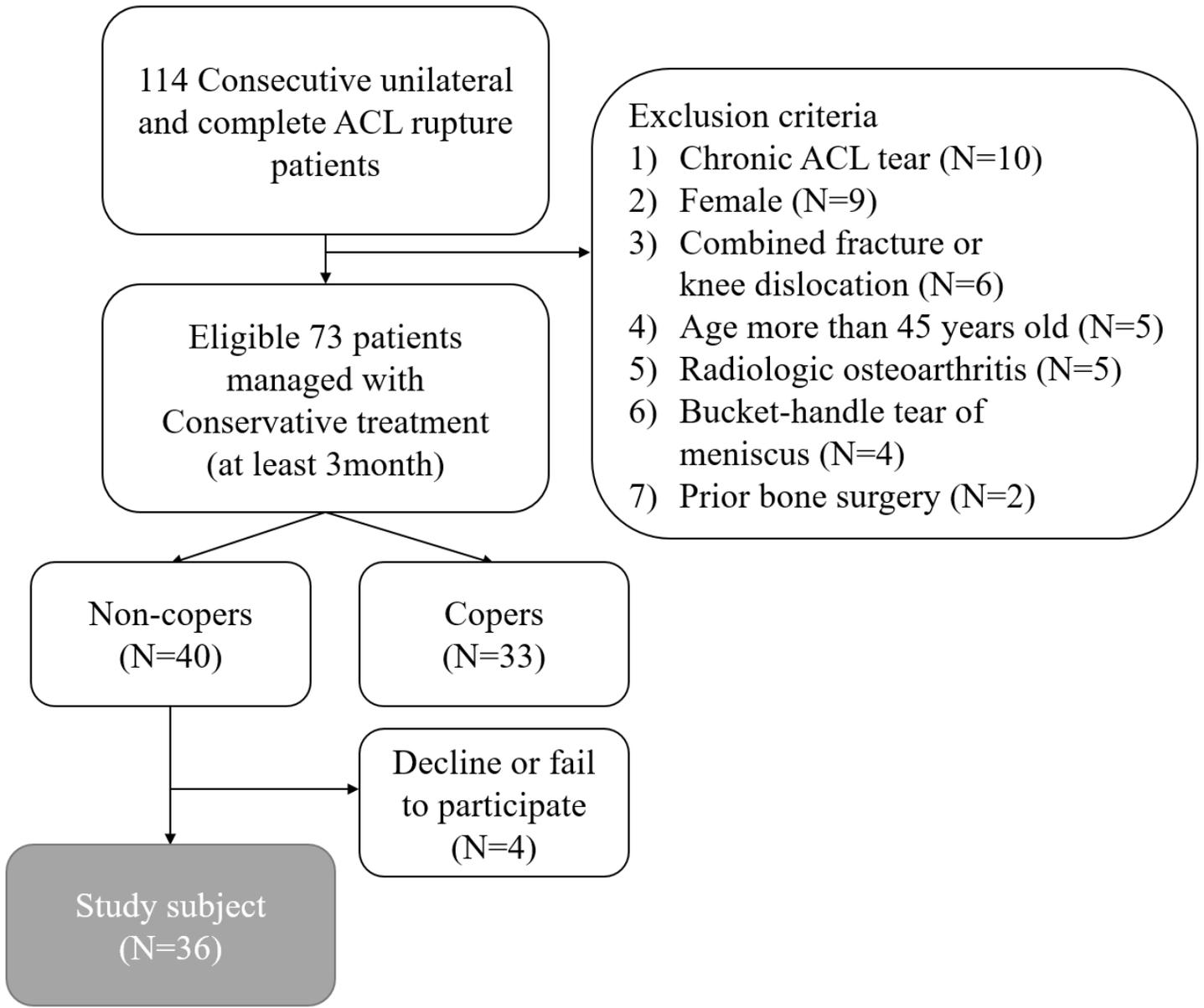
Table 2. Regression model for kinematic strategy

Regression models	Peak KEM		
	$\beta \pm SE^*$	P-value	R <sup>2</sup> adj †
1. Knee extension at IDS	0.152 ± 0.019	<0.001	0.475
	Amplitude of KEM		
	$\beta \pm SE^*$	P-value	R <sup>2</sup> adj †
2. Knee flexion at SLS to TDS	-0.731 ± 0.087	<0.001	0.497

\* Values are given as the  $\beta$  (standardized regression coefficient) and SE (standard error)

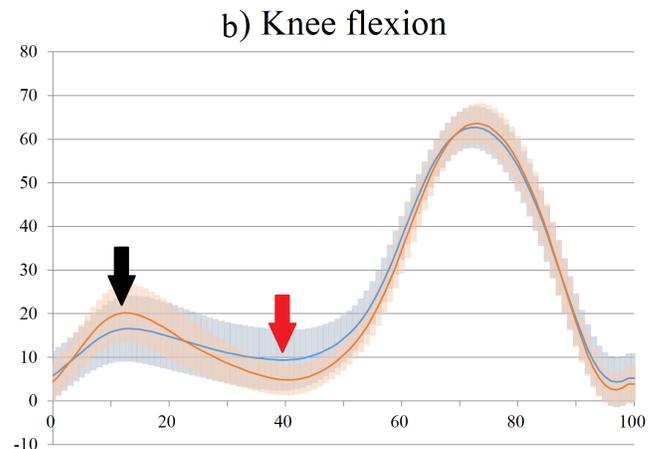
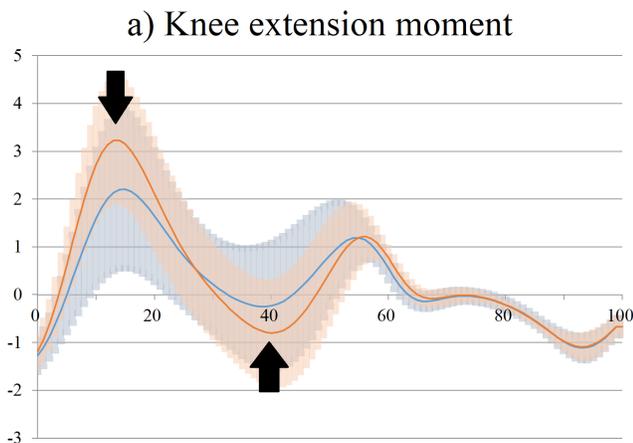
† R<sup>2</sup>adj = % variance explained by each variable

## Figures



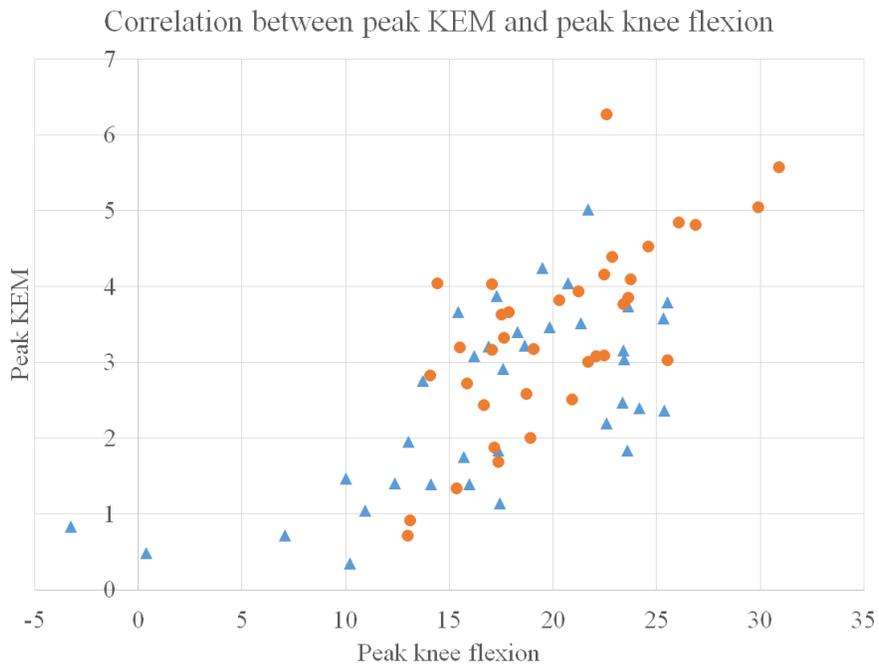
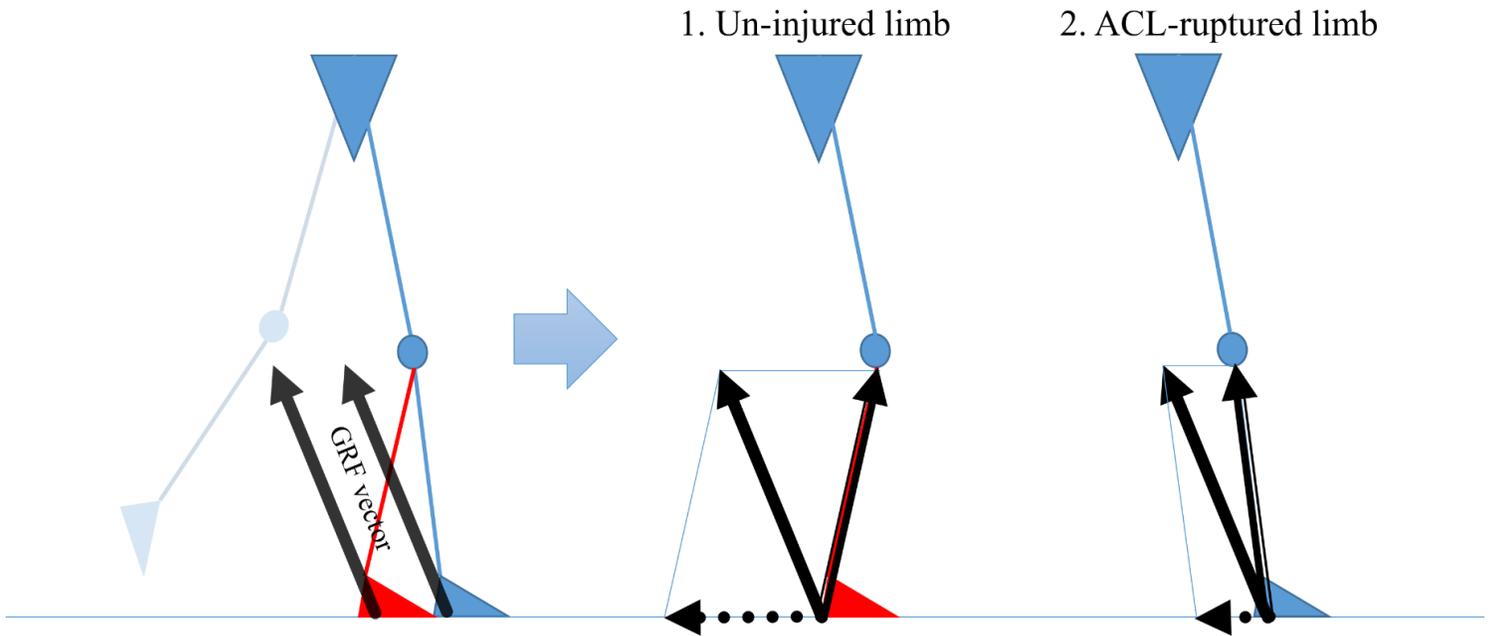
**Figure 1**

Study design and eligibility criteria. Thirty-six patients were included in this cross-sectional study.



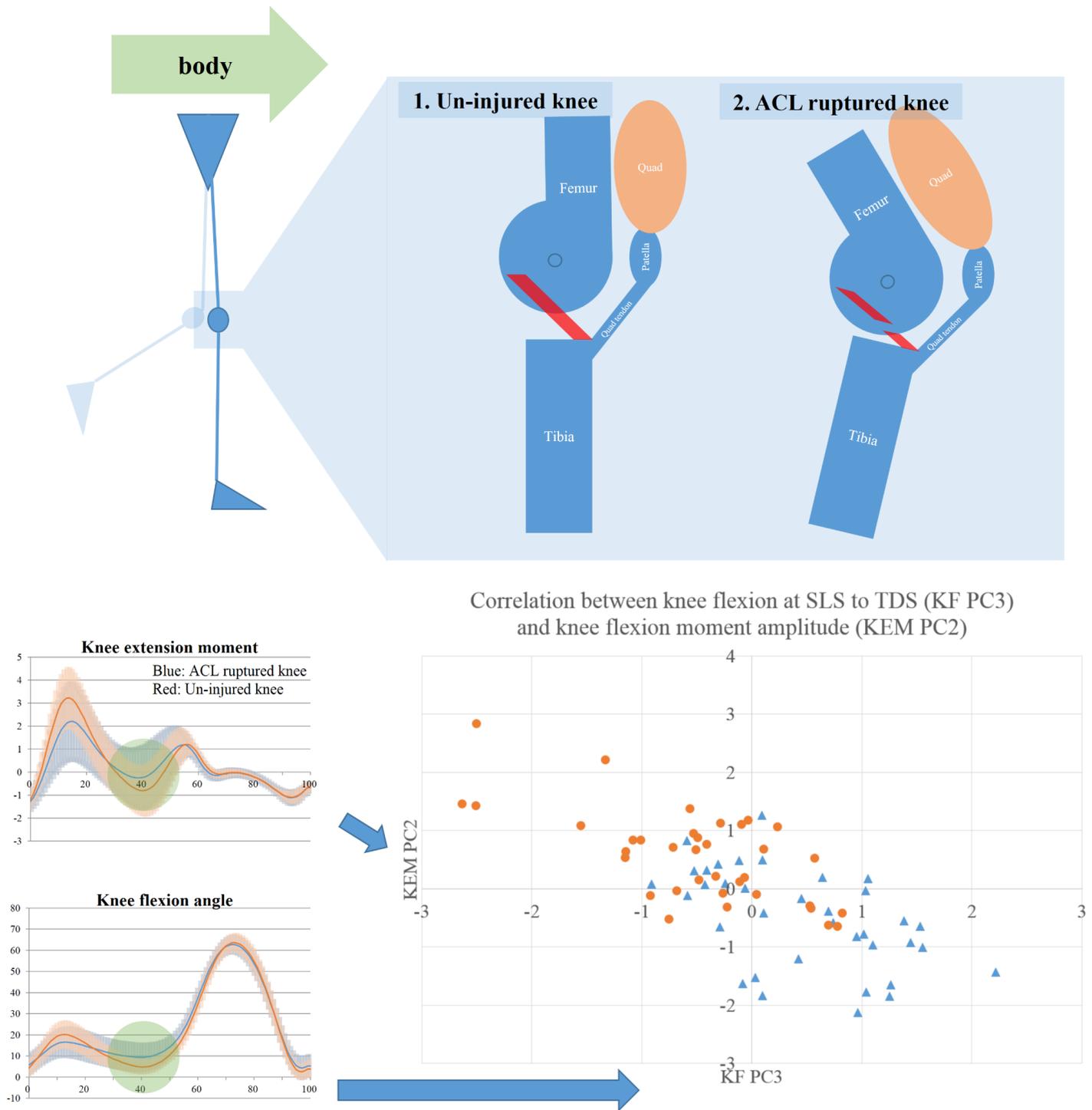
**Figure 2**

Kinetics and kinematics of the knee joint in the sagittal and axial planes. The blue curve indicates the ACL-ruptured limb and the red curve indicates the contralateral un-injured limb. The shaded region represents  $\pm$  one standard deviation. Table 1 present the statistical analysis of the graph. a) Knee extension moment. The knee extension moment peak value and amplitude were both smaller in the ACL-ruptured limb (black arrow). b) Knee flexion angle. The ACL-ruptured knee showed extension at the IDS phase (black arrow) and more flexion from the SLS to the TDS phase (red arrow).



### Figure 3

Schematic representations of knee extension moment (KEM) and knee flexion angle at the initial double limb stance (IDS) phase. During this phase, ground reaction force (GRF) is generated for the repulsive body weight force (black arrow). The GRF can be divided into an axial vector (double arrow) and a transverse vector (dotted arrow). The axial vector runs parallel to the tibia and acts as a compressive force to the tibiofemoral joint. The transverse vector runs parallel to the ground and acts as a knee flexion force (counter to the knee extension moment by the quadriceps). The ACL-ruptured knee can be unstable during this phase, so patients try to reduce the transverse vector by extending their knee (note the difference in knee flexion angle). Instead, the tibiofemoral joint axial force can be increased. The graph shows the correlations between peak KEM and peak knee flexion at the IDS phase. The blue triangle represents the ACL-ruptured limb and the orange circle represents the un-injured limb. Note the strong correlation between two variables (Pearson  $r=0.694$ ,  $p<0.001$ ). Linear regression analysis showed that the adjusted R<sup>2</sup> value of the first strategy was 0.475.



**Figure 4**

Schematic representations of knee extension moment (KEM) and knee flexion angle during progression from the single limb stance (SLS) to the terminal double limb stance (TDS) phase. During this phase, the knee joint is more flexed in ACL-ruptured knees. Extended knees can be unstable during this phase because the KEM rapidly becomes negative (see the green KEM circle). Patients try to decrease the speed of the KEM changes by flexing their ACL-ruptured knee. This strategy has previously been described as the 'quadriceps avoidance or stiffening strategy'. The lower right graph shows the correlation between KF

PC3 (knee flexion principal component 2) and KEM PC2. KF PC3 represents the knee flexion angle during progression from the SLS to the TDS phase. KEM PC2 represents the KEM amplitude. The blue triangle represents the ACL-ruptured limb and the orange circle represents the un-injured limb. Linear regression analysis showed that the adjusted R2 value of the second strategy was 0.497.