# Chapter 10
# ACL Injury Mechanisms

**Hideyuki Koga and Takeshi Muneta**

**Abstract** Model-based image-matching (MBIM) technique has enabled detailed video analysis of injury situations that had been limited to simple visual inspection. We have analyzed anterior cruciate ligament (ACL) injury situations from ten analogs and one HD video sequence using the MBIM technique. The knee kinematical patterns were remarkably consistent, with immediate valgus and internal rotation (IR) motion occurring within 40 ms after initial contact (IC), and then an external rotation was observed. Peak vertical ground reaction force (GRF) occurred at 40 ms after IC. Based on these results, it is likely that the ACL injury occurred approximately 40 ms after IC. 9 mm of abrupt anterior tibial translation at the time of injury was also detected in the HD video. On the other hand, the hip kinematics was constant at abducted, flexed, and IR position during 40 ms after IC. Based on these results together with previous studies, we proposed a new hypothesis for ACL injury mechanisms that valgus loading and lateral compression generate IR motion and anterior translation of tibia, due to the joint geometry, resulting in ACL rupture. Moreover, it seems that the hip is relatively "locked" at IC and cannot absorb energy from GRF and knee is exposed to a larger force, which leads to ACL injury. These results suggest that prevention programs should focus on acquiring a good cutting and landing technique with knee flexion avoiding knee valgus and foot internal rotation and with hip flexion to absorb energy from GRF. Moreover, the fact that the ACL injury occurs 40 ms after IC suggests that "feed-forward" strategies before landing may be critical, as "feedback" strategies are too slow to prevent ACL injuries.

**Keywords** Anterior cruciate ligament • Video analysis • Injury mechanism

---

H. Koga, M.D., Ph.D. (✉) • T. Muneta
Department of Joint Surgery and Sports Medicine, Graduate School of Medicine, Tokyo Medical and Dental University, 1-5-45 Yushima, Bunkyo-ku, Tokyo 113-8519, Japan
e-mail: koga.orj@tmd.ac.jp



M. Ochi et al. (eds.), *ACL Injury and Its Treatment*,
DOI 10.1007/978-4-431-55858-3_10

## 10.1 Introduction

Anterior cruciate ligament (ACL) injuries occur mostly during sports activities, and the incidence remains high, especially in young athletes. Recent development of ACL reconstruction procedures has enabled such athletes' return to sports, and favorable clinical results have been achieved; yet it takes a relatively long period for most athletes to get back to sports activities. It has also been reported that ACL reconstruction could not prevent progression of osteoarthritis [1]. Therefore, importance of ACL injury prevention has been emphasized, and various ACL injury prevention programs have been developed successfully [2–6]; however, it is not well understood how the different elements in these multicomponent programs play particular roles in preventing the injury. The most common mechanism of ACL injuries for the sports commonly associated with this injury was noncontact, except men's contact sports, and the noncontact mechanism predominantly occurs during cutting or one-leg landing maneuvers [7–9]. Nevertheless, to develop more targeted injury preventive programs, a more detailed description of the mechanism(s) of noncontact ACL injuries is needed.

### *10.1.1 Research Approaches to Injury Mechanisms*

As mentioned above, it is important to understand the detailed injury mechanisms in order to develop specific prevention methods for ACL injuries. A number of different methodological approaches have been used to investigate ACL injury mechanisms. These include athlete interviews, clinical studies, laboratory motion analysis, video analysis, cadaver studies, and mathematical simulations [10].

### *10.1.2 Previously Proposed ACL Injury Mechanisms*

Noncontact ACL injury mechanisms have been investigated using the abovementioned approaches, and several theories have been proposed; however, it is still a matter of controversy, with the main opponents favoring either sagittal or non-sagittal plane knee joint loading. DeMorat et al. proposed that aggressive quadriceps loading was responsible, based on a cadaver study which demonstrated that aggressive quadriceps loading could take the ACL to failure [11,12]. In contrast, Mclean et al., based on a mathematical simulation model, argued that sagittal plane loading alone could not produce such injuries [13,14]. A prospective cohort study among female athletes, showing that increased dynamic valgus and high valgus loads increased injury risk, leads Hewett et al. to suggest valgus loading as an important component [15,16]. Some video analyses also showed that valgus collapse seemed to be the main mechanism among female athletes [8,9]. However, cadaver studies and mathematical simulation have shown that pure valgus motion would not produce ACL injuries without tearing the medial collateral ligament (MCL) first [17,18].

Nevertheless, other simulation studies have suggested that valgus loading would substantially increase ACL force in situations where an anterior tibial shear force is applied [19]. In the MRI findings, Speer et al. reported that bone bruises of the lateral femoral condyle or posterolateral portion of tibial plateau occurred in more than 80 % of acute ACL noncontact injuries. They concluded that valgus in combination with internal rotation and/or anterior tibial translation occurred at the time of ACL injuries [20]. Furthermore, it has been shown that valgus loading induces a coupled motion of valgus and internal tibial rotation [21,22].

Although both cadaver studies and MRI studies have suggested that internal rotation is present in ACL injury situations, video analyses have suggested that valgus in combination with external rotation is the most frequent motion pattern [9,23].

### *10.1.3 Development of Model-Based Image-Matching Technique*

Among several different approaches to investigate ACL injury mechanisms, video analysis of injury tapes is the only method available to extract kinematic data from actual injury situations. However, video analyses have so far been limited to simple visual inspection [7,9,24], and the accuracy of this method has been shown to be poor, even among experienced researchers [25]. In addition, simple visual inspection is not sufficient to extract a time course for joint angles, velocities, and accelerations; therefore, it is difficult to determine the point of ACL rupture. The analyses are also compromised with poor video qualities.

Therefore, model-based image-matching (MBIM) technique has been developed as an alternative to simple visual inspection, in order to extract joint kinematics from video recordings using one or more uncalibrated cameras [26–29]. Detailed procedure of the MBIM technique has been described in the literatures; the idea of this technique is that matching a model to the background video sequences gives an estimate of the actual three-dimensional body kinematics using the commercially available program Poser® and Poser® Pro Pack (Curious Labs Inc., Santa Cruz, California, USA). This technique has been validated in noninjury situations in a laboratory environment. The MBIM technique has shown to be much more accurate than the simple visual inspection approach, and the validation study has shown that root-mean-square (RMS) differences for knee flexion, abduction, and rotation with two or three cameras were less than 10°, 6°, and 11°, respectively [25,26]. Another study also found this technique to be feasible for use in actual ACL injury situations [29]. Therefore, videotapes of noncontact ACL injury situations were analyzed using the MBIM technique to describe detailed kinematics of ACL injuries, in order to identify ACL injury mechanisms.

## 10.2 Biomechanics in Noncontact ACL Injuries

Ten ACL injury situations from women's team handball (n = 7) and basketball (n = 3), recorded with at least two analog cameras during TV broadcasts, were analyzed using MBIM technique (Fig. 10.1); all of them occurred during game situations [27]. All the players were handling the ball in the injury situation; seven were in possession of the ball at the time of injury, two had shot, and one had passed the ball. In six cases, there was player-to-player contact with an opponent at the time of injury, all of them to the torso being pushed or held. There was no direct contact to the knee. The injury situations could be classified into two groups; seven cases occurred when cutting and three during one-leg landings.

The knee kinematical patterns were remarkably constant among the ten cases (Fig. 10.2). The knee was relatively straight, with a flexion angle of 23° (range,

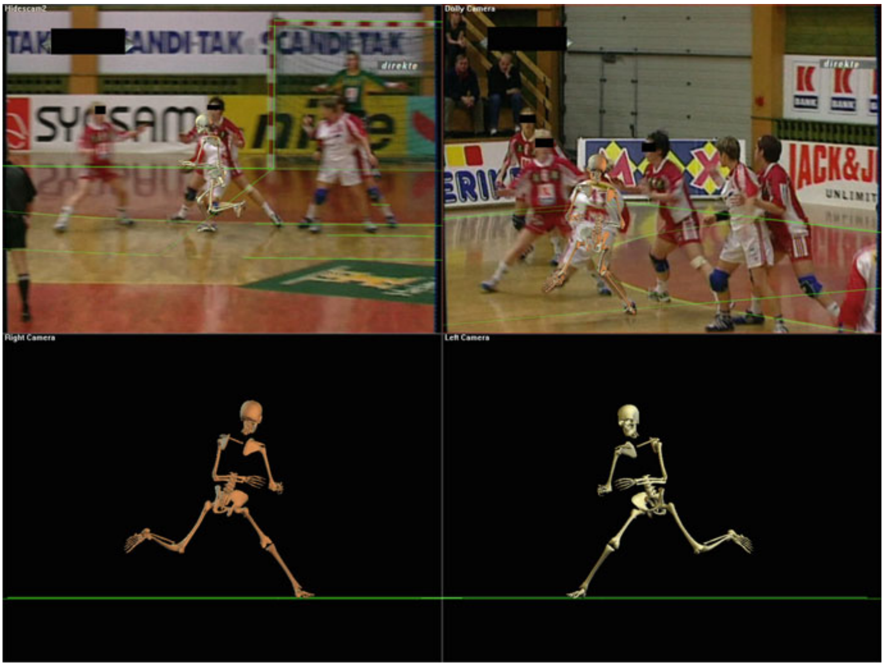



**Fig. 10.1** An example of a video matched in Poser, two-camera handball injury situation 40 ms after initial contact (IC). The two *top panels* show the customized skeleton model and the handball court model superimposed on and matched with the background video image from two cameras with different angles. The two *bottom panels* show the skeleton model from back and side views created in Poser

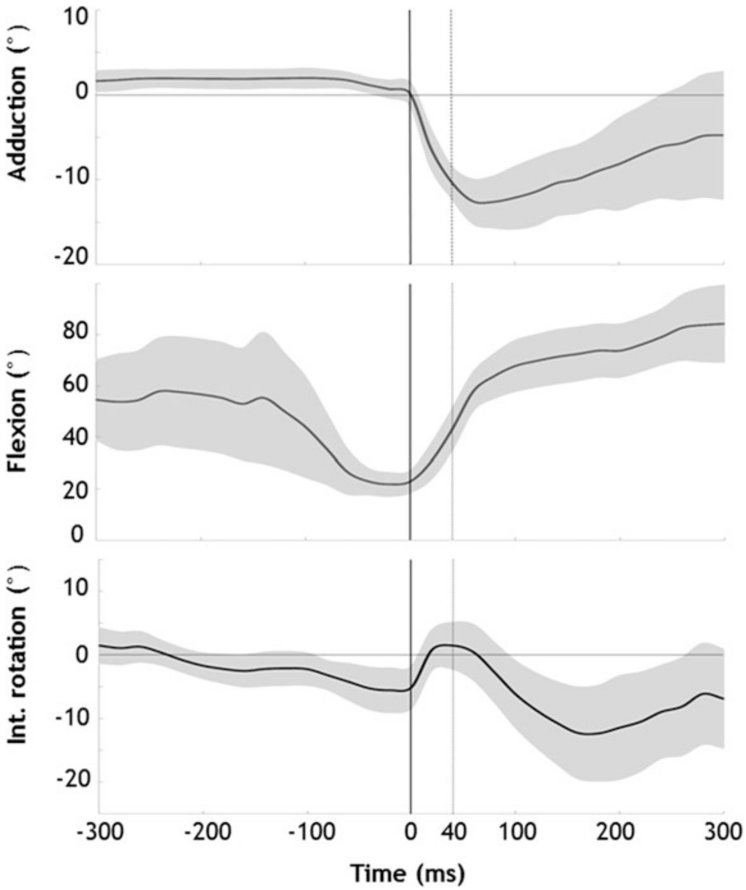



**Fig. 10.2** Time sequences of the mean knee angles (°) (*black line*) of the ten cases with 95 % confidence intervals (CI) (*gray area*). Time 0 indicates IC and the *dotted vertical line* indicates the time point 40 ms after IC

11–30°) at initial contact (IC) and had increased by 24° (95 % CI, 19–29°, $p < 0.001$) 40 ms later. The knee abduction angle was neutral, 0° (range, −2–3°) at IC, but had increased by 12° (95 % CI, 10–13°, $p < 0.001$) 40 ms later. As for knee rotation angle, the knee was externally rotated 5° (range, −5–12°) at IC, but abruptly rotated internally by 8° (95 % CI, 2–14°, $p = 0.037$) during the first 40 ms. From 40 ms to 300 ms after IC, however, we observed an external rotation of 17° (95 % CI, 13–22°, $p < 0.001$). In addition, the estimated peak vertical ground reaction force (GRF) was 3.2 times body weight (95 % CI, 2.7–3.7) and occurred at 40 ms (range, 0–83) after IC. On the other hand, the hip kinematics was constant at 20° abducted, 50° flexed, and 30° IR position during 40 ms after IC (Fig. 10.3).

However, limitations of the abovementioned analysis were how accurate the joint kinematics and timing of peak GRF could be estimated from the relatively low

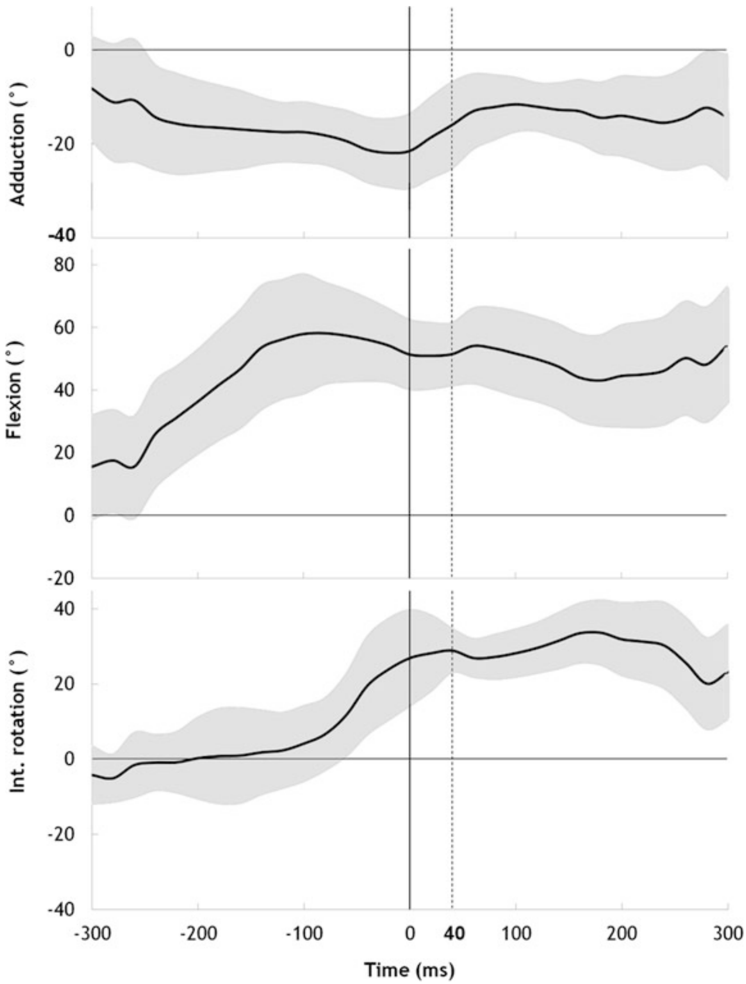



**Fig. 10.3** Time sequences of the mean hip angles (°) (*black line*) of the ten cases with 95 % CI (*gray area*). Time 0 indicates IC and the *dotted vertical line* indicates the time point 40 ms after IC

frame rate (50 or 60 Hz) and low-quality images (768 × 576 pixels) in analog video sequences, and therefore we were unable to assess the anterior translation of the tibia. However, a noncontact ACL injury situation in a male footballer was available which had been recorded using four high-definition (HD) cameras including two high-speed recordings (100 and 300 Hz) [28]. In this case, the 26-year-old male elite football player suffered a noncontact ACL injury to his right knee during a national team match, when he tried to stop after having passed the ball with his right leg. This case was analyzed using the MBIM technique to describe the more detailed joint kinematics, including tibial translations (Fig. 10.4). Knee kinematics in this case was strikingly consistent with the previous analyses of the ten cases


**Fig. 10.4** A soccer injury situation recorded using HD cameras. Each panel shows the customized skeleton model and the football pitch model superimposed on and matched with the background video image from each camera. *Overview camera* and *rear camera* had an effective frame rate after being deinterlaced of 50 Hz, *frontal camera* 100 Hz, and *side camera* 300 Hz

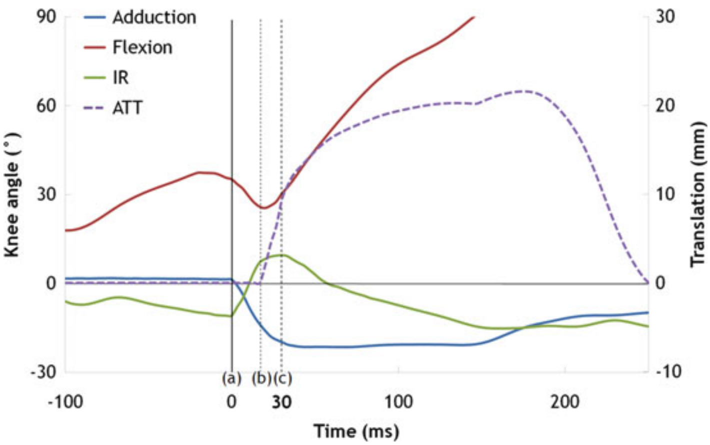



**Fig. 10.5** Time sequences of knee joint angles (*left axis*) and anterior tibial translation (r*ight axi*s) in the soccer case. Time 0 (*a*) indicates IC and the dotted vertical lines (*b*) and (*c*) indicate the time point 20 and 30 ms after IC, respectively

(Fig. 10.5). The knee was flexed 35° at IC, with initial extension (26° of flexion) until 20 ms after IC, after which flexion angle continued to increase. The knee abduction angle was neutral at IC, but had increased by 21° 30 ms later. The knee was externally rotated 11° at IC, but abruptly rotated internally by 21° during the first 30 ms, and then changed its direction to external rotation after this. In addition, anterior tibial translation was able to be detected; it started to occur at 20 ms after IC, where the knee was the most extended, and by 30 ms after IC approximately 9 mm of anterior translation had occurred. The translations plateaued by 150 ms and then shifted back to a reduced position between 200 ms and 240 ms after IC.

## 10.3 Timing of Noncontact ACL Injuries

It has not been possible to determine the exact timing of ACL injury from video analysis based on simple visual inspection [7–9]. However, this may be possible by using the MBIM technique, by assessing abnormal joint configurations, sudden changes in joint angular motion, and timing of GRFs. The extracted knee kinematics during ACL injuries using MBIM technique showed that sudden increase of valgus and internal rotation angle occurred within the first 40 ms after IC. These periods also correspond to the average peak vertical GRF in these cases. Moreover, in the case recorded using HD cameras, abrupt anterior tibial translation reached 9 mm in 30 ms after IC, which corresponds to the maximum anterior translation in intact knees [30,31]. Based on these results, together with the previous studies showing that the ACL was strained shortly (approximately 40 ms) after IC in simulated landing [19,32], it seems likely that the injury occurs within 40 ms for the majority of these cases.

## 10.4 Mechanisms for Noncontact ACL Injury

As already mentioned, valgus collapse in combination with external rotation (i.e., knee in, toe out) has been frequently identified as an ACL injury mechanism in simple visual inspection of injury videotapes. However, it has been discussed as to whether this kinematics actually represents the cause for ACL injuries or simply is a result of the ACL being torn [9,23]. Our results using the MBIM technique showed that immediate valgus motion occurred within 40 ms after IC. The abrupt internal rotation also occurred during the first 40 ms after IC, and then external rotation was observed, which seems to have occurred after the ACL was torn. In addition, anterior tibial translation started a little later after IC and increased abruptly until when the injury might have occurred. The discrepancy between the previous studies and our results could be that the abrupt internal rotation and anterior tibial translation observed using the MBIM technique analysis are likely not easily detected from visual inspection alone; the external rotation that occurs afterwards is more pronounced and therefore easier to observe. The internal-to-external rotation sequence with anterior tibial translation has also been reported previously. In a recent cadaver study, the application of pure compressive loads led to anterior tibial translation and internal tibial rotation of up to 8°, followed by a sudden external rotation of 12°[31]. The combination of internal tibial rotation and anterior tibial

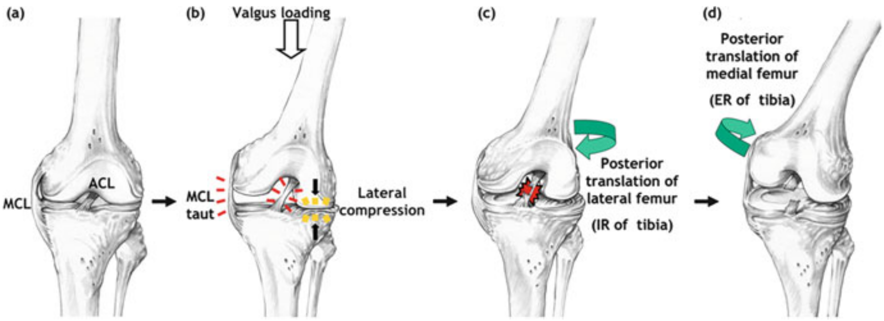



**Fig. 10.6** The proposed noncontact ACL injury mechanism. (**a**) An unloaded knee. (**b**) When valgus loading is applied, the MCL becomes taut and lateral compression occurs. (**c**) This compressive load causes a lateral femoral posterior displacement, probably due to the posterior slope of lateral tibial plateau, and the tibia translates anteriorly and rotates internally, resulting in ACL rupture. (**d**) After the ACL is torn, the primary restraint to anterior translation of the tibia is gone. This causes the medial femoral condyle to also be displaced posteriorly, resulting in external rotation of the tibia

translation is probably caused by the joint surface geometry. The concave geometry of the medial tibia facet combined with the slightly convex lateral tibia facet may cause the lateral femoral condyle to slip back. This may also explain why ACL-injured patients tend to have greater posterior lateral tibial plateau slopes than uninjured controls [33–35].

Combining the results obtained using the MBIM technique with previous findings, the following hypothesis for the mechanism of noncontact ACL injury is proposed (Fig. 10.6): (1) when valgus loading is applied, the MCL becomes taut and lateral compression occurs. (2) This compressive load causes a lateral femoral posterior displacement, probably due to the posterior slope of lateral tibial plateau, and the tibia translates anteriorly and rotates internally, resulting in ACL rupture. (3) After the ACL is torn, the primary restraint to anterior translation of the tibia is gone. This causes the medial femoral condyle to also be displaced posteriorly, resulting in external rotation of the tibia. This external rotation may be exacerbated by the typical movement pattern when athletes plant and cut, where the foot typically rotates externally relative to the trunk.

## 10.5 Hip's Role in ACL Injury Mechanisms

Lower extremities act as a kinetic chain during dynamic tasks and the control of hip motion largely affects the knee motion. Researchers have studied the relationships between hip biomechanics and ACL injury. In terms of risk factor of ACL injury in hip biomechanics, Decker et al. [36] reported that, in drop landing, energy absorption at hip joint and hip flexion angle at IC were less in females than in males. Schmitz et al. [37] reported that, in single-leg landing, energy absorption at hip and total hip flexion displacement were smaller in female, whereas peak vertical GRF was larger in female. Yu et al. [38] also reported that hip flexion angular velocity at IC was negatively correlated with peak vertical GRF in stop-jump task. When it comes to ACL injury mechanisms, Heshemi et al. [39] reported that, in a cadaver study, a restricted flexion of the hip at 20° combined with low quadriceps and hamstring force levels in simulated single-leg landing were found to be conductive to ACL injury. A video analysis has shown that ACL-injured subjects' hip flexion and abduction angle was constant during 100 ms after IC, whereas uninjured control subjects' hip flexion increased by 15° in cutting/landing maneuvers [40]. Our study using MBIM technique also showed that hip kinematics was constant during 40 ms after IC at abducted, flexed, and internally rotated position, which seems to play a significant role in the mechanism of ACL injury. In this regard, Hashemi et al. [41] have proposed a mechanism called "hip extension, knee flexion paradox," i.e., mismatch between hip and knee flexion in landing is the cause of ACL injury. In normal condition, both the knee and hip flex together in landing, whereas in unbalanced landing, the knee is forced to flex but hip is forced to extend, and tibia will undergo anterior translation, which will increase the risk of ACL injury.

There are some possible causes of hip/knee mismatch: (1) in sagittal plane, upright or backward-leaning trunk position at IC makes center of mass posterior to the knee, and increased GRF may encourage more knee flexion than hip flexion and relatively act to extend the hip. (2) In the other plane, insufficient hip abductor/external rotator strength or activation would lead to adducted/internally rotated position of the hip, causing knee valgus. (3) Large hip internal rotation at IC seen in our video analysis could also be an explanation; ACL-injured patients could have limited range of motion in internal rotation [42], and hip joint may be locked at a large internally rotated position. As a matter of fact, hip dysplasia has also been reported to be a risk factor of ACL injury [43]. It has also been reported that decreased range of internal femoral rotation results in greater ACL strain [44].

For these reasons, it seems that hip joint is relatively locked at IC and cannot absorb energy from GRF, and knee joint is exposed to larger force, which leads to ACL injury. Therefore, it is important that prevention efforts should focus not only on knee joint but also on hip joint.

## 10.6 Tips for ACL Injury Prevention Based on the Proposed Mechanisms

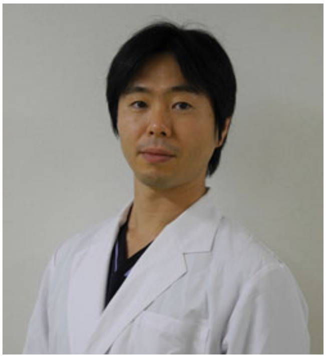
Based on the mechanisms clarified using MBIM technique, prevention strategy for ACL injury can be proposed as follows: (1) as the kinematics when ACL injury is happening is knee valgus and internal rotation with the hip being locked, it is important to acquire a good cutting and landing technique with knee flexion avoiding knee valgus and foot internal rotation and with hip flexion to absorb energy from GRF, avoiding hip internal rotation. (2) As ACL injuries occur approximately 40 ms after IC, it is likely that a “feedback” strategy, i.e., ACL prevention program focusing on training after landing, cannot prevent ACL injury; it takes at least 150–200 ms to react after landing at risk. Prevention efforts should focus on a “feed-forward” strategy before landing, i.e., training muscular pre-activation and neural control during the pre-landing phase.

## References


1. Oiestad BE, Engebretsen L, Storheim K, Risberg MA (2009) Knee osteoarthritis after anterior cruciate ligament injury: a systematic review. Am J Sports Med 37(7):1434–1443
2. Caraffa A, Cerulli G, Projetti M, Aisa G, Rizzo A (1996) Prevention of anterior cruciate ligament injuries in soccer. A prospective controlled study of proprioceptive training. Knee Surg Sports Traumatol Arthrosc 4(1):19–21
3. Gilchrist J, Mandelbaum BR, Melancon H, Ryan GW, Silvers HJ, Griffin LY, Watanabe DS, Dick RW, Dvorak J (2008) A randomized controlled trial to prevent noncontact anterior cruciate ligament injury in female collegiate soccer players. Am J Sports Med 36 (8):1476–1483
4. Mandelbaum BR, Silvers HJ, Watanabe DS, Knarr JF, Thomas SD, Griffin LY, Kirkendall DT, Garrett W Jr (2005) Effectiveness of a neuromuscular and proprioceptive training program in preventing anterior cruciate ligament injuries in female athletes: 2-year follow-up. Am J Sports Med 33(7):1003–1010
5. Myklebust G, Engebretsen L, Braekken IH, Skjolberg A, Olsen OE, Bahr R (2003) Prevention of anterior cruciate ligament injuries in female team handball players: a prospective intervention study over three seasons. Clin J Sport Med 13(2):71–78
6. Olsen OE, Myklebust G, Engebretsen L, Holme I, Bahr R (2005) Exercises to prevent lower limb injuries in youth sports: cluster randomised controlled trial. BMJ 330(7489):449
7. Boden BP, Dean GS, Feagin JA Jr, Garrett WE Jr (2000) Mechanisms of anterior cruciate ligament injury. Orthopedics 23(6):573–578
8. Krosshaug T, Nakamae A, Boden BP, Engebretsen L, Smith G, Slauterbeck JR, Hewett TE, Bahr R (2007) Mechanisms of anterior cruciate ligament injury in basketball: video analysis of 39 cases. Am J Sports Med 35(3):359–367
9. Olsen OE, Myklebust G, Engebretsen L, Bahr R (2004) Injury mechanisms for anterior cruciate ligament injuries in team handball: a systematic video analysis. Am J Sports Med 32(4):1002–1012
10. Krosshaug T, Andersen TE, Olsen OE, Myklebust G, Bahr R (2005) Research approaches to describe the mechanisms of injuries in sport: limitations and possibilities. Br J Sports Med 39 (6):330–339
11. DeMorat G, Weinhold P, Blackburn T, Chudik S, Garrett W (2004) Aggressive quadriceps loading can induce noncontact anterior cruciate ligament injury. Am J Sports Med 32 (2):477–483
12. Yu B, Garrett WE (2007) Mechanisms of non-contact ACL injuries. Br J Sports Med 41(Suppl 1):i47–i51
13. McLean SG, Andrish JT, van den Bogert AJ (2005) Aggressive quadriceps loading can induce noncontact anterior cruciate ligament injury. Am J Sports Med 33(7):1106; author reply 1106–1107
14. McLean SG, Huang X, Su A, Van Den Bogert AJ (2004) Sagittal plane biomechanics cannot injure the ACL during sidestep cutting. Clin Biomech (Bristol, Avon) 19(8):828–838
15. Hewett TE, Myer GD, Ford KR, Heidt RS Jr, Colosimo AJ, McLean SG, van den Bogert AJ, Paterno MV, Succop P (2005) Biomechanical measures of neuromuscular control and valgus

loading of the knee predict anterior cruciate ligament injury risk in female athletes: a prospective study. Am J Sports Med 33(4):492–501
16. Quatman CE, Hewett TE (2009) The anterior cruciate ligament injury controversy: is "valgus collapse" a sex-specific mechanism? Br J Sports Med 43(5):328–335
17. Mazzocca AD, Nissen CW, Geary M, Adams DJ (2003) Valgus medial collateral ligament rupture causes concomitant loading and damage of the anterior cruciate ligament. J Knee Surg 16(3):148–151
18. Shin CS, Chaudhari AM, Andriacchi TP (2009) The effect of isolated valgus moments on ACL strain during single-leg landing: a simulation study. J Biomech 42(3):280–285
19. Withrow TJ, Huston LJ, Wojtys EM, Ashton-Miller JA (2006) The effect of an impulsive knee valgus moment on in vitro relative ACL strain during a simulated jump landing. Clin Biomech (Bristol, Avon) 21(9):977–983
20. Speer KP, Spritzer CE, Bassett FH 3rd, Feagin JA Jr, Garrett WE Jr (1992) Osseous injury associated with acute tears of the anterior cruciate ligament. Am J Sports Med 20(4):382–389
21. Matsumoto H (1990) Mechanism of the pivot shift. J Bone Joint Surg (Br) 72(5):816–821
22. Matsumoto H, Suda Y, Otani T, Niki Y, Seedhom BB, Fujikawa K (2001) Roles of the anterior cruciate ligament and the medial collateral ligament in preventing valgus instability. J Orthop Sci 6(1):28–32
23. Ebstrup JF, Bojsen-Moller F (2000) Anterior cruciate ligament injury in indoor ball games. Scand J Med Sci Sports 10(2):114–116
24. Cochrane JL, Lloyd DG, Buttfield A, Seward H, McGivern J (2007) Characteristics of anterior cruciate ligament injuries in Australian football. J Sci Med Sport 10(2):96–104
25. Krosshaug T, Nakamae A, Boden B, Engebretsen L, Smith G, Slauterbeck J, Hewett TE, Bahr R (2007) Estimating 3D joint kinematics from video sequences of running and cutting maneuvers–assessing the accuracy of simple visual inspection. Gait Posture 26(3):378–385
26. Krosshaug T, Bahr R (2005) A model-based image-matching technique for three-dimensional reconstruction of human motion from uncalibrated video sequences. J Biomech 38(4):919–929
27. Koga H, Nakamae A, Shima Y, Iwasa J, Myklebust G, Engebretsen L, Bahr R, Krosshaug T (2010) Mechanisms for noncontact anterior cruciate ligament injuries: knee joint kinematics in 10 injury situations from female team handball and basketball. Am J Sports Med 38 (11):2218–2225
28. Koga H, Bahr R, Myklebust G, Engebretsen L, Grund T, Krosshaug T (2011) Estimating anterior tibial translation from model-based image-matching of a noncontact anterior cruciate ligament injury in professional football: a case report. Clin J Sport Med 21(3):271–274
29. Krosshaug T, Slauterbeck JR, Engebretsen L, Bahr R (2007) Biomechanical analysis of anterior cruciate ligament injury mechanisms: three-dimensional motion reconstruction from video sequences. Scand J Med Sci Sports 17(5):508–519
30. Jakob RP, Staubli HU, Deland JT (1987) Grading the pivot shift. Objective tests with implications for treatment. J Bone Joint Surg (Br) 69(2):294–299
31. Meyer EG, Haut RC (2008) Anterior cruciate ligament injury induced by internal tibial torsion or tibiofemoral compression. J Biomech 41(16):3377–3383
32. Shin CS, Chaudhari AM, Andriacchi TP (2007) The influence of deceleration forces on ACL strain during single-leg landing: a simulation study. J Biomech 40(5):1145–1152
33. Brandon ML, Haynes PT, Bonamo JR, Flynn MI, Barrett GR, Sherman MF (2006) The association between posterior-inferior tibial slope and anterior cruciate ligament insufficiency. Arthroscopy 22(8):894–899
34. Stijak L, Herzog RF, Schai P (2008) Is there an influence of the tibial slope of the lateral condyle on the ACL lesion? A case-control study. Knee Surg Sports Traumatol Arthrosc 16 (2):112–117
35. Hashemi J, Chandrashekar N, Mansouri H, Gill B, Slauterbeck JR, Schutt RC Jr, Dabezies E, Beynnon BD (2010) Shallow medial tibial plateau and steep medial and lateral tibial slopes: new risk factors for anterior cruciate ligament injuries. Am J Sports Med 38(1):54–62

36. Decker MJ, Torry MR, Wyland DJ, Sterett WI, Richard Steadman J (2003) Gender differences in lower extremity kinematics, kinetics and energy absorption during landing. Clin Biomech (Bristol, Avon) 18(7):662–669
37. Schmitz RJ, Kulas AS, Perrin DH, Riemann BL, Shultz SJ (2007) Sex differences in lower extremity biomechanics during single leg landings. Clin Biomech (Bristol, Avon) 22 (6):681–688
38. Yu B, Lin CF, Garrett WE (2006) Lower extremity biomechanics during the landing of a stop-jump task. Clin Biomech (Bristol, Avon) 21(3):297–305
39. Hashemi J, Chandrashekar N, Jang T, Karpat F, Oseto M, Ekwaro-Osire S (2007) An alternative mechanism of non-contact anterior cruciate ligament injury during jump-landing: in-vitro simulation. Exp Mech 47:347–354
40. Boden BP, Torg JS, Knowles SB, Hewett TE (2009) Video analysis of anterior cruciate ligament injury: abnormalities in hip and ankle kinematics. Am J Sports Med 37(2):252–259
41. Hashemi J, Breighner R, Chandrashekar N, Hardy DM, Chaudhari AM, Shultz SJ, Slauterbeck JR, Beynnon BD (2011) Hip extension, knee flexion paradox: a new mechanism for non-contact ACL injury. J Biomech 44(4):577–585
42. Gomes JL, de Castro JV, Becker R (2008) Decreased hip range of motion and noncontact injuries of the anterior cruciate ligament. Arthroscopy 24(9):1034–1037
43. Yamazaki J, Muneta T, Ju YJ, Morito T, Okuwaki T, Sekiya I (2011) Hip acetabular dysplasia and joint laxity of female anterior cruciate ligament-injured patients. Am J Sports Med 39 (2):410–414
44. Beaulieu ML, Oh YK, Bedi A, Ashton-Miller JA, Wojtys EM (2014) Does limited internal femoral rotation increase peak anterior cruciate ligament strain during a simulated pivot landing? Am J Sports Med 42(12):2955–2963



**Hideyuki Koga, M.D., Ph.D.,** Department of Joint Surgery and Sports Medicine, Tokyo Medical and Dental University, Tokyo, Japan.