In vivo biomechanics of cruciate ligament injuries

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CHAPTER

Evaluation of knee biomechanics with three-dimensional modeling techniques.

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In orthopaedic surgery, the central pathology motivating the study of biomechanics is osteoarthritis (OA). The stiffness, pain and loss of movement attributed to OA affect nearly 27 million people in the United States and increase its total health care costs by $186 billion each year.[20] The knee, the principal large joint to be targeted by OA, results in disabling knee symptoms in an estimated 10% of the UK population older than 55, a quarter of whom are severely disabled.[28] Despite decades of research, OA continues to be viewed as an irreversible degradation of the joint. Upon diagnosis, the patient is resigned to either palliative medical care or – if conservative management fails – surgical replacement of the diseased joint. Current orthopaedic practice and its attempts at preventing OA are to a large extent based on the widely accepted assumption that abnormal joint kinematics, with consequent abnormal loading within the joint, initiate detrimental processes such as the progressive degeneration of articular cartilage and the subchondral bone next to it. The biomechanics of the various musculoskeletal articulations are therefore studied so that treatment protocols could be developed that create optimal joint environments: the creation of an optimal biomechanical joint environment, whether it is done through reconstructing a ruptured ligament or modifying the joint alignment by bracing or osteotomy is believed to avert long-term complications of OA.

The predicament is that any research, biomechanics or not, directed at OA and its prevention is complicated by the fact that the pathogenesis of OA is likely multifactorial and remains unclear to a large extent. The long-held belief that knee OA is a straightforward "wear and tear" disease of cartilage has been abandoned by many experts.[2] Instead, the metabolic and structural changes that are seen in the osteoarthritic joint are currently viewed as the adaptive response of the synovial joint to a variety of genetic, constitutional, or biomechanical insults.[6] Nevertheless, it remains widely accepted that knee joint instability is an important risk factor in the aetiopathogenesis of the disease[11,12,30] – an assumption bolstered, for example, by the success of the instability-inducing transection of the anterior cruciate ligament (ACL), a well-established animal model to study OA.[7,24] Analogous to the study of osteoarthritic changes following ACL deficiency in dogs, sheep or horses, the biomechanical examination of patients that are seen in the orthopaedic practice with an instability-inducing injury, such as rupture of the ACL or posterior cruciate ligament (PCL), has been compelling to OA researchers for several reasons. Rupture of one of the cruciate ligaments is a common and acute injury (annually one in every 1750 people between 15 and 25 years old could become ACL-deficient[15] and up to 40% of patients evaluated for knee hemarthrosis have an associated PCL injury[10]), and is associated with rapid cartilage degeneration and a very high risk for developing knee OA in the affected knee.[30] In other words, patient recruitment is relatively easy, the time of disease onset could be clearly determined, and the contralateral knee could be used as an intra-subject control.[19]

Even though OA is now widely believed to result in part from local mechanical factors acting within the context of systemic susceptibility, very little is known about the

biomechanical response of healthy articular cartilage to physiologic loading or about the extent of the mechanical alteration in the knee joint once one of the cruciate ligaments is ruptured. Even less is known about the impact of surgical reconstruction on the biomechanics of articular cartilage. In addition, most research has been directed predominantly at the tibiofemoral joint. In contrast, little is attention has been paid to the patellofemoral joint – the knee compartment with often the predominant clinical complaints following cruciate ligament injury. Finally, few quantitative data have been reported on the effect of cruciate ligament deficiency on the soft tissue structures surrounding the knee joint – structures critical for the maintenance of the knee joint homeostasis. We believe that these critical gaps in knowledge are due to the difficulty in analyzing with an acceptable accuracy the in vivo biomechanics of the both the tibiofemoral and patellofemoral joint with its corresponding cartilage layers and soft tissue structures.

Historically, the joint kinematics have been studied using cadaveric specimens. For example, in our laboratory a robotic testing system is used which applies various external loads on a human cadaver knee and measures the corresponding changes in knee kinematics as well as in situ forces in the native ACL and reconstruction grafts (Figure 1). Knowledge obtained from these studies has been vital for our current approach to the treatment of

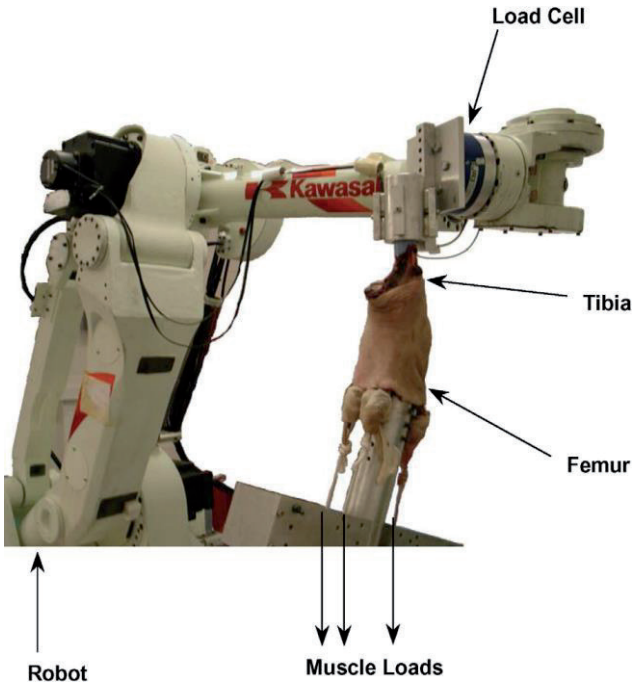

Figure 1. The robotic testing system with pulleys for the application of muscle loads.

various articular pathologies. However, due to the complexity of muscle loading patterns, the simulation of the human joint function under physiological loads remains difficult in in-vitro conditions. Furthermore, factors such as graft healing and postoperative rehabilitation cannot be addressed in-vitro. Out of this context, the quest for an accurate, minimally invasive method to analyze the in vivo joint biomechanics was initiated.

In brief, measuring the in vivo joint biomechanics in six degrees-of-freedom (6DOF) with an acceptable accuracy has been proven to be technically challenging. Multiple video cameras have been used to track the three-dimensional (3D) motions of reflective markers fixed to the skin.[8,29] Due to the relative motion between the skin and the underlying bones, as well as the difficulty in identifying external landmarks on the tested joint, there is a certain degree of inaccuracy associated with this technique.[4] One solution that has been proposed to reduce the artifact associated with non-rigid body movement of points placed on the skin during gait analysis was based on uniformly distributing a cluster of points on the limb segment.[1] To entirely eliminate the effect of skin motion, reflective markers have been fixed directly to knee or ankle bones using intracortical pins.[21,35] Another technique, roentgen stereophotogrammatric analysis (RSA), was developed to measure the motion of roentgen opaque markers embedded within bone, which are captured using dual X-ray images.[23,33] The accuracy of kinematic measurements greatly improved. Unfortunately the enhanced precision could only be attained through disruption of the joint, limiting the clinical implementations. Another highly accurate but intrinsically invasive analysis technique which has greatly enhanced our understanding of in vivo joint contact forces was the placement of instrumented joint prostheses that measure the in vivo dynamic loads experienced by for example the shoulder,[5] hip,[17] or knee[9] during daily activities. Unfortunately, the technique is obviously limited to patients scheduled for joint arthroplasty surgery.

Recently, computed tomography (CT) and magnetic resonance (MR) imaging techniques have been introduced into in vivo musculoskeletal joint biomechanics studies.[16,26,31] These techniques offer 3D quantification of in vivo morphology and positions of the joint without using invasive markers – qualifications of particular value in the examination of the human spine with its complicated anatomy[13,27] or for the study of the complex glenohumeral and thoracoscapular motion patterns in the shoulder.[14] Within the imaging equipment however, joint motion is restricted, thereby limiting the flexibility to study various functional activities.

Fluoroscopy, the radiology technique that allows still or moving digital images of internal structures on a monitor or TV screen, has been used extensively for the analysis of in vivo knee joint and total knee arthroplasty (TKA) kinematics,[3,18,32,34,36] due to its relative accessibility, easiness to operate, and low radiation dosage compared to traditional X-rays.

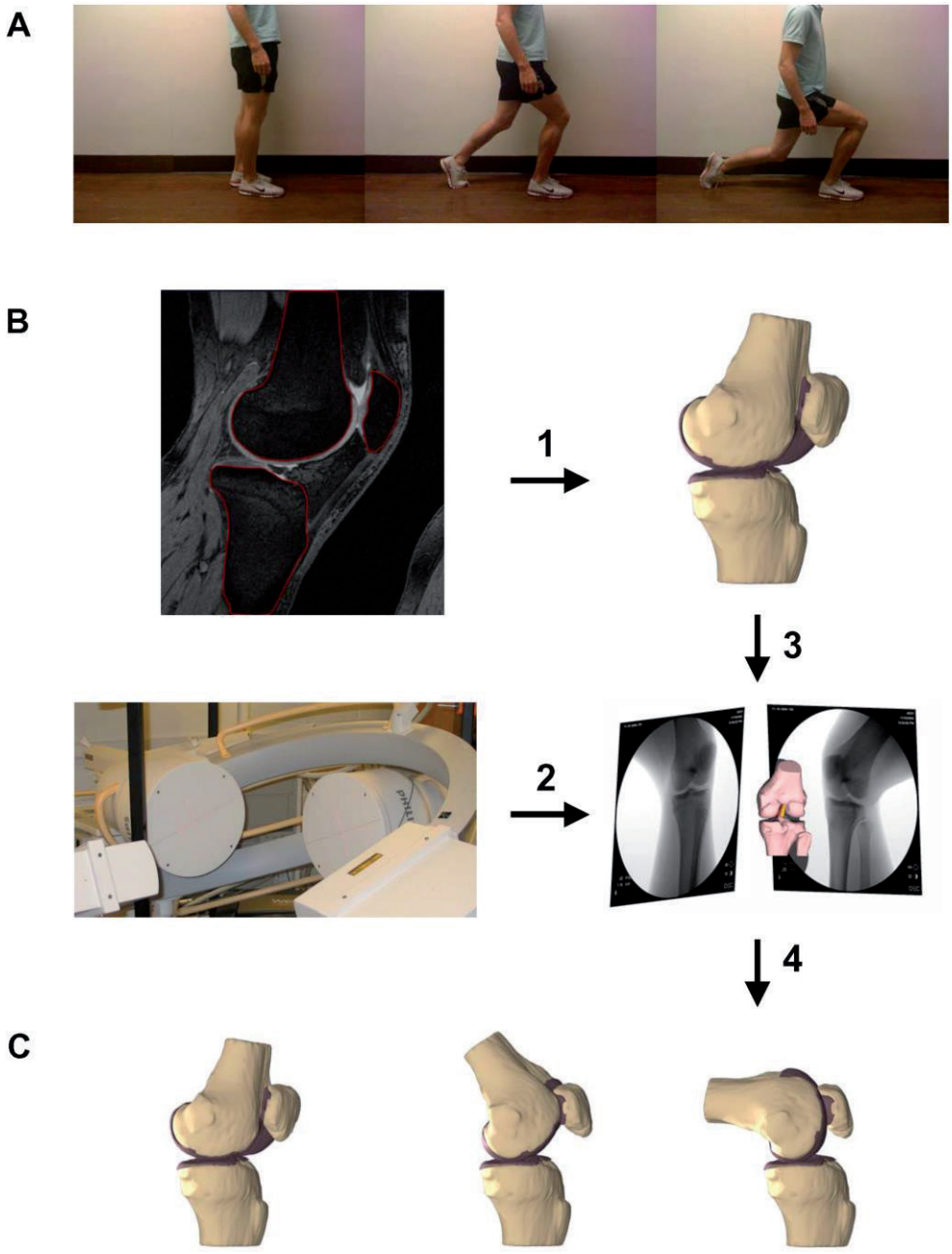

Figure 2. A concise overview of the methodology. The kinematics during a single-leg lunge (**A**) were analyzed using a combined MR and dual fluoroscopic imaging technique (**B**), in which an MR-based 3D model (1) and fluoroscopic images (2) are combined and manipulated in six degrees-of-freedom (3), resulting in a series of knee models (4) which accurately reproduce the performed in vivo activity (**C**).

The widespread availability of mobile digital fluoroscopic systems with dynamic imaging capabilities and adjustable configurations ("C-arms") has placed this type of motion analysis now within reach of many research groups. Both single- and double-plane fluoroscopic systems are utilized with excellent results. For the examination of tissue responses under in vivo loading conditions, single-plane fluoroscopy provides experimental flexibility and relatively large viewing volumes, besides the lower radiation and cost.[188] Although 3D model matching could theoretically be achieved using a single image, certain studies have found that the use of only a single image may not result in the same accuracy in the out-of-plane degrees-of-freedom compared to the in-plane motion.[18,22] To eliminate this critical source of error, our laboratory has added the additional fluoroscope, thereby creating a dual fluoroscopic imaging system for the analysis of in vivo knee joint motion in all degrees-of-freedom.

Over the past decade, the research of joint biomechanics in our laboratory has gone through an evolution from in-vitro simulation to in vivo measurements. The overall goal of our project was to develop a non-invasive methodology that would not only be capable of capturing joint kinematics in all degrees-of-freedom, but would also be sufficiently accurate for the detailed analysis of subtle articular contact changes during physiologic loading. Furthermore, the methodology would incorporate unmodified and commercially available imaging hardware, so that – once automated algorithms for the various processes are refined – the in vivo analysis of joint biomechanics could be employed in routine clinical settings. Our approach was to combine the benefits of fluoroscopy (i.e. in vivo joint motion) and MR imaging (i.e. joint anatomy), while reducing the innate limitations of the techniques in isolation (Figure 2).

REFERENCES

1. Andriacchi, T. P.; Alexander, E. J.; Toney, M. K.; Dyrby, C.; and Sum, J.: A point cluster method for in vivo motion analysis: applied to a study of knee kinematics. J Biomech Eng, 120(6): 743-9, 1998.
2. Aspden, R. M.; Scheven, B. A.; and Hutchison, J. D.: Osteoarthritis as a systemic disorder including stromal cell differentiation and lipid metabolism. Lancet, 357(9262): 1118-20, 2001.
3. Banks, S. A., and Hodge, W. A.: Accurate measurement of three-dimensional knee replacement kinematics using single-plane fluoroscopy. IEEE Trans Biomed Eng, 43(6): 638-49, 1996.
4. Benoit, D. L.; Ramsey, D. K.; Lamontagne, M.; Xu, L.; Wretenberg, P.; and Renstrom, P.: Effect of skin movement artifact on knee kinematics during gait and cutting motions measured in vivo. Gait Posture, 24(2): 152-64, 2006.
5. Bergmann, G.; Graichen, F.; Bender, A.; Kaab, M.; Rohlmann, A.; and Westerhoff, P.: In vivo glenohumeral contact forces--measurements in the first patient 7 months postoperatively. J Biomech, 40(10): 2139-49, 2007.
6. Brandt, K.; Lohmander, L.; and Doherty, M.: The concept of osteoarthritis as failure of the diarthrodial joint. In Osteoarthritis, pp. 70-74. Edited by Brandt, K.; Doherty, M.; and Lohmander, L., 70-74, Oxford, Oxford University Press, 1998.
7. Brandt, K. D.; Myers, S. L.; Burr, D.; and Albrecht, M.: Osteoarthritic changes in canine articular cartilage, subchondral bone, and synovium fifty-four months after transection of the anterior cruciate ligament. Arthritis Rheum, 34(12): 1560-70, 1991.
8. Chao, E. Y.: Biomechanics of the human gait. Frontiers in Biomechanics, in Zweifach B, New York, NY: Springer-Verlag: 225-219, 1986.
9. D'Lima, D. D.; Steklov, N.; Patil, S.; and Colwell, C. W., Jr.: The Mark Coventry Award: in vivo knee forces during recreation and exercise after knee arthroplasty. Clin Orthop Relat Res, 466(11): 2605-11, 2008.
10. Fanelli, G. C.; Giannotti, B. F.; and Edson, C. J.: The posterior cruciate ligament arthroscopic evaluation and treatment. Arthroscopy, 10(6): 673-88, 1994.
11. Felson, D. T. et al.: Osteoarthritis: new insights. Part 1: the disease and its risk factors. Ann Intern Med, 133(8): 635-46, 2000.
12. Fithian, D. C.; Paxton, L. W.; and Goltz, D. H.: Fate of the anterior cruciate ligament-injured knee. Orthop Clin North Am, 33(4): 621-36, v, 2002.
13. Fujii, R.; Sakaura, H.; Mukai, Y.; Hosono, N.; Ishii, T.; Iwasaki, M.; Yoshikawa, H.; and Sugamoto, K.: Kinematics of the lumbar spine in trunk rotation: in vivo three-dimensional analysis using magnetic resonance imaging. Eur Spine J, 16(11): 1867-74, 2007.
14. Graichen, H.; Stammberger, T.; Bonel, H.; Karl-Hans, E.; Reiser, M.; and Eckstein, F.: Glenohumeral translation during active and passive elevation of the shoulder - a 3D open-MRI study. J Biomech, 33(5): 609-13, 2000.

15. Griffin, L. Y. et al.: Noncontact anterior cruciate ligament injuries: risk factors and prevention strategies. J Am Acad Orthop Surg, 8(3): 141-50, 2000.

16. Hirsch, B. E.; Udupa, J. K.; and Roberts, D.: Three-dimensional reconstruction of the foot from computed tomography scans. J Am Podiatr Med Assoc, 79(8): 384-94, 1989.

17. Hodge, W. A.; Fijan, R. S.; Carlson, K. L.; Burgess, R. G.; Harris, W. H.; and Mann, R. W.: Contact pressures in the human hip joint measured in vivo. Proc Natl Acad Sci U S A, 83(9): 2879-83, 1986.

18. Komistek, R. D.; Dennis, D. A.; and Mahfouz, M.: In vivo fluoroscopic analysis of the normal human knee. Clin Orthop, (410): 69-81, 2003.

19. Kozanek, M.; Van de Velde, S. K.; Gill, T. J.; and Li, G.: The contralateral knee joint in cruciate ligament deficiency. Am J Sports Med, 36(11): 2151-7, 2008.

20. Lawrence, R. C. et al.: Estimates of the prevalence of arthritis and other rheumatic conditions in the United States. Part II. *Arthritis Rheum,* 58(1): 26-35, 2008.

21. Lafortune, M. A.; Cavanagh, P. R.; Sommer, H. J., 3rd; and Kalenak, A.: Three-dimensional kinematics of the human knee during walking. J Biomech, 25(4): 347-57, 1992.

22. Li, G.; Wuerz, T. H.; and DeFrate, L. E.: Feasibility of using orthogonal fluoroscopic images to measure in vivo joint kinematics. J Biomech Eng, 126(2): 314-8, 2004.

23. Lundberg, A.; Goldie, I.; Kalin, B.; and Selvik, G.: Kinematics of the ankle/foot complex: plantarflexion and dorsiflexion. Foot Ankle, 9(4): 194-200, 1989.

24. Marshall, K. W., and Chan, A. D.: Arthroscopic anterior cruciate ligament transection induces canine osteoarthritis. J Rheumatol, 23(2): 338-43, 1996.

25. Moro-oka, T. A.; Hamai, S.; Miura, H.; Shimoto, T.; Higaki, H.; Fregly, B. J.; Iwamoto, Y.; and Banks, S. A.: Dynamic activity dependence of in vivo normal knee kinematics. J Orthop Res, 26(4): 428-34, 2008.

26. Nakagawa, S.; Kadoya, Y.; Todo, S.; Kobayashi, A.; Sakamoto, H.; Freeman, M. A.; and Yamano, Y.: Tibiofemoral movement 3: full flexion in the living knee studied by MRI. J Bone Joint Surg Br, 82(8): 1199-200, 2000.

27. Ochia, R. S.; Inoue, N.; Renner, S. M.; Lorenz, E. P.; Lim, T. H.; Andersson, G. B.; and An, H. S.: Three-dimensional in vivo measurement of lumbar spine segmental motion. Spine, 31(18): 2073-8, 2006.

28. Peat, G.; McCarney, R.; and Croft, P.: Knee pain and osteoarthritis in older adults: a review of community burden and current use of primary health care. *Ann Rheum Dis,* 60(2): 91-7, 2001.

29. Reinschmidt, C.; van Den Bogert, A. J.; Murphy, N.; Lundberg, A.; and Nigg, B. M.: Tibiocalcaneal motion during running, measured with external and bone markers. Clin Biomech (Bristol, Avon), 12(1): 8-16, 1997.

30. Roos, H.; Adalberth, T.; Dahlberg, L.; and Lohmander, L. S.: Osteoarthritis of the knee after injury to the anterior cruciate ligament or meniscus: the influence of time and age. Osteoarthritis Cartilage, 3(4): 261-7, 1995.

31. Sheehan, F. T.; Zajac, F. E.; and Drace, J. E.: Using cine phase contrast magnetic resonance imaging to non-invasively study in vivo knee dynamics. J Biomech, 31(1): 21-6, 1998.

32. Stiehl, J. B.; Komistek, R. D.; Dennis, D. A.; Paxson, R. D.; and Hoff, W. A.: Fluoroscopic analysis of kinematics after posterior-cruciate-retaining knee arthroplasty. J Bone Joint Surg Br, 77(6): 884-9, 1995.

33. van Dijk, R.; Huiskes, R.; and Selvik, G.: Roentgen stereophotogrammetric methods for the evaluation of the three dimensional kinematic behaviour and cruciate ligament length patterns of the human knee joint. J Biomech, 12(9): 727-31, 1979.

34. Varadarajan, K. M.; Moynihan, A. L.; D'Lima, D.; Colwell, C. W.; and Li, G.: In vivo contact kinematics and contact forces of the knee after total knee arthroplasty during dynamic weight-bearing activities. J Biomech, 41(10): 2159-68, 2008.

35. Westblad, P.; Hashimoto, T.; Winson, I.; Lundberg, A.; and Arndt, A.: Differences in ankle-joint complex motion during the stance phase of walking as measured by superficial and bone-anchored markers. Foot Ankle Int, 23(9): 856-63, 2002.

36. Zihlmann, M. S.; Gerber, H.; Stacoff, A.; Burckhardt, K.; Szekely, G.; and Stussi, E.: Three-dimensional kinematics and kinetics of total knee arthroplasty during level walking using single plane video-fluoroscopy and force plates: a pilot study. Gait Posture, 24(4): 475-81, 2006.