Analyzing Anterior Knee Laxity with Isolated Fiber Bundles of Anterior Cruciate Ligament

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Abstract—Anterior cruciate ligament or ACL is damaged frequently during strenuous activities like in sports. Following injury and treatment, not only a significant percentage of patients are not able to return to their pre-injury level activity, but also they continue to have knee related complications in medium to long term period. Therefore, there is a need for better understanding of the knee. In the present study, anterior laxity of the knee that is related to the function of ACL, is analyzed during passive flexion of the joint with intact and isolated deficiency of selected fibers of the ligament.

A mathematical model of the knee was developed in the sagittal plane with ligaments represented as bundles of non-linear elastic fibers. A laxity test with 90N and 180N anteriorly directed external force on the tibia was simulated at several flexion angles. Anatomical parameters and material properties were obtained from previous studies on cadaver knees.

The results from model calculations showed agreement with experimental observations on cadaver knees. An anterior force translated the tibia anterior to the femur non-linearly through the flexion range. Isolated deficiency of anteromedial fibers of the ACL resulted in increased translations for all flexion positions and at both 90N and 180N laxity test. Isolated deficiency of the posterolateral fibers for 90N test resulted in increased translations only near full extension but for 180N it resulted in increased translations for early flexion and high flexion positions of the joint. This suggests that the posterolateral bundle may be required for situations involving large anterior tibial translations. The analysis has relevance to ACL-reconstruction and ACL rehabilitation.

Index Terms—knee biomechanics, anterior cruciate ligament injury, ACL reconstruction, ACL rehabilitation, knee laxity.

I. INTRODUCTION

CONTROVERCIES exist regarding surgical techniques for the treatment of anterior cruciate ligament (ACL) at the knee joint. No current technique recreates the complex nature of the natural ligament. The cruciate ligaments are considered as the main stabilizers of the joint in the sagittal plane [1–4]. Anterior cruciate ligament restricts anterior translation and posterior cruciate ligament restricts posterior translation of the lower bone, or tibia, relative to the upper bone, or femur. Passive laxity tests in the absence of muscle forces measure relative translations of the bones at fixed flexion positions of the joint [5]. Such tests are conducted to estimate integrity of the ligaments. For example, an increased laxity in the anterior direction, normally compared with the laxity of contralateral knee, may indicate damage to the ACL.

Contributions of the knee ligaments in stabilizing the intact or replaced joint have been studied using in vitro experiments on cadaver knees [1–3, 6] or using mathematical modelling [7]. Also, investigators have analyzed patterns of geometric changes in the ligament fiber bundles during flexion [2, 3, 8, 9]. The ACL shows a complex functional behavior mainly resulting from variations in geometry and in material properties of different fiber bundles. Such changes have influence on the knee joint mechanics [1–4, 6–10].

Clinical experience suggests that the ACL is frequently damaged while performing strenuous activities such as in sports. Literature suggests 100000–200000 sports-related ACL injuries per year in the USA alone [11]. Further, Arden et al [12] reported that less than 50% of athletes with ACL reconstruction were able to return to their pre-injury level activity. Interestingly, another clinical study showed that 94% of patients from ligament surgery continued to have knee instability even after a five-year follow-up [13]. This suggests that more understanding of the knee ligaments is needed in order to improve outcome.

Therefore, to understand the role of ACL in the knee mechanics during activity as well as in understanding mechanics of the ligament injury, appropriate requirements for ligament reconstruction and rehabilitation, more investigations are needed.

The purpose of the present study is to analyze the anterior knee laxity during flexion motion of the joint with intact and isolated deficiency of selected fibers of the ACL.

II. METHODS

A computer based mathematical model of the knee was developed in the sagittal plane. The cruciate ligaments were represented as non-linear elastic fibers. Collateral ligaments of the knee were not considered in this study as their contribution towards anterior-posterior stability is minimal [14]. Passive motion of the knee was defined in the absence of muscle forces or external loads such that selected fibers in the cruciate ligaments maintained nearly constant lengths during 0–120° flexion [7, 15, 16]. An anterior laxity test, similar to Lachman test or Drawer test [5], was simulated.
during flexion at 15° interval. In the simulation, a known anterior force was applied on the tibia while maintaining the flexion angle fixed. As a result of the applied force, the tibia translated anterior to the femur and stretched the ACL. The magnitude of translation gave the anterior laxity at that flexion angle. Anatomical parameters and material properties of the ligaments were estimated from the literature [15–20]. Further, ACL is shown to have two distinct functional bundles of fibers, classified as antero-medial (AM) and postero-lateral (PL) bundles [2, 10, 19]. Accordingly, the model analysis in the sagittal plane was performed on two sets of ligament fibers, namely, anterior and posterior fiber bundles, here referred to as AM and PM, respectively. Anterior tibial translation (ATT) was calculated with all the fibers intact, or with either the AM fibers removed / deficient (AM–D) or the PL fibers removed / deficient (PL–D). The simulation was repeated for 90N and 180N anterior laxity test. The model calculations were compared with results from similar experiments on cadaver knees available in literature [6].

III. RESULTS

Table 1 gives values of anterior tibial translation (ATT) resulting from 90N anterior force applied on the tibia for different flexion positions of the joint. Model calculations are compared with experimental measurements on 14 cadaver knees shown as mean values with standard deviation by Kondo et al [6].

Fig.1a compares the model calculations and experimental measurements with the AM fibers removed from the ACL (AM–D). 90N laxity test was performed during 0–120° flexion. Similarly, Fig.1b compares the model calculations and experimental measurements with the PL fibers removed from the ACL (PL–D).

Fig.2a and Fig.2b give the calculated values for ATT during flexion with all the fibers intact as well as with selected fibers removed to represent deficiency of antero-medial (AM–D) or postero-lateral (PL–D) bundles, respectively, for the laxity test at 90N and 180N anterior force.

IV. ANALYSIS

From table 1, the model results for simulated test with 90N anterior force on tibia show values of tibial translations very similar to those reported by Kondo et al [6] from in vitro experiments conducted on 14 cadaver knees. At each flexion position, the model calculations for tibial translation are close to the experimental mean values and are within the reported standard deviation. The tibial translation or the anterior laxity first increased from low to mid flexion range and then decreased in higher flexion.

The effect of ligament fiber deficiency is shown in Fig.1a and Fig.1b, respectively, corresponding to selective removal of AM of PL fibers.

In Fig. 1a, the model calculations for AM–D underestimate the experiment, possibly due to large specimen-to-specimen variations as also depicted by the standard deviation in table 1. However, the calculated and the measured values show similar trends. When compared with ATT in table 1 for the intact ligament, deficiency of the AM fibers resulted in much increased laxity at all flexion positions of the joint.

In Fig. 1b for PL–D, the calculated and measured values are very similar, except near full extension. However, the calculated and measured values show similar trends. When compared to the intact ligament, deficiency of the PL fibers resulted in little or no change in ATT, except near full extension where the model predicted increased laxity.

These observations agree with other anatomical studies suggesting that the anteromedial bundle of ACL is the primary restraint against anterior tibial translation and the posterolateral bundle provides contributions near full extension [18].

Further, Fig.2a and Fig.2b suggest that compared to the intact ligament, the ATT increased uniformly over the flexion range for both AM–D and PL–D situations as the external load was increased from 90N to 180N. In addition to the observations corresponding to the deficiency of the AM fibers, the model calculations suggest that the deficiency of the PL fibers too may require attention particularly near extension or during situations involving large ATT, as experienced in some sports activities. Such increased values of ATT may help in explaining the instability experienced by the patients with partial tears of the ACL.

V. CONCLUSION

Model calculations during a simulated knee laxity test showed reasonable agreement with experimental measurements from literature. The analysis suggests that antero-medial and posterior-lateral bundles of the ACL provide significantly different contributions towards resisting anterior forces on the tibia during knee motion. The postero-lateral bundle may be required for situations involving large anterior tibial translations. The analysis has relevance to ACL-reconstruction and ACL rehabilitation.
Fig. 1a. ATT plotted over the flexion range corresponding to 90N laxity test with isolated deficiency of the AM fibers of ACL. The model calculations are compared with experimental measurements of Kondo et al [6].

Fig. 1b. ATT plotted over the flexion range corresponding to 90N laxity test with isolated deficiency of the PL fibers of ACL. The model calculations are compared with experimental measurements of Kondo et al [6].

Fig. 2a. ATT plotted over the flexion range. The model calculations for the intact and the ML-deficient ACL are given for 90N and 180N laxity test.
Fig. 2b. ATT plotted over the flexion range. The model calculations for the intact and the PL–deficient ACL are given for 90N and 180N laxity test.