

## INFLUENCE OF MUSCLE PRE-ACTIVATION AND KNEE JOINT ANGLE ON AXIAL TIBIO-FEMORAL SHOCK TRANSMISSION

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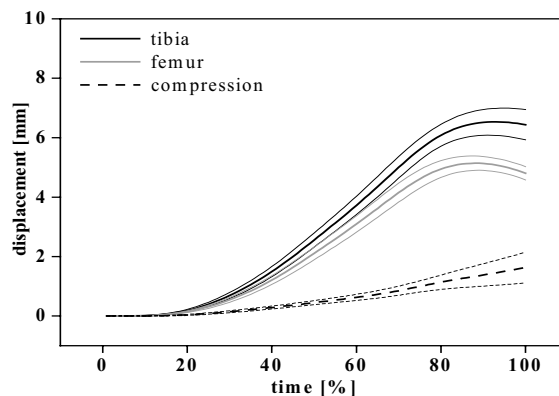
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### INTRODUCTION

A number of cadaver studies has shown a shock attenuating capacity of intra- and periarticular tissues in axial impact loading [1,2]. This shock reduction was mainly explained by deformation. In vivo reductions in shock transmission in walking or running were primarily explained by variations in leg stiffness due to modified joint angles or muscle activity [3,4]. The interdependence of joint angles and muscle activation in locomotor activities makes it difficult to analyze determining factors of shock transmission during locomotion. The purpose of this study was to analyze the role of muscle pre-activation and knee angle on shock transmission in controlled situations.

### METHODS

Four male (35-47 years, 70-88kg) healthy subjects volunteered for this study. They were positioned supine with three different goniometric controlled knee angles (0°, 20°, 40°). The forefoot was strapped on the metal plate of an impact device. The hip angle (40°) and ankle angle (90°) were constant. Three muscle activation levels were defined in respect to prior maximal voluntary contractions (0%, 30%, 60% MVC) at each angle condition. Using EMG (Biovision®) the activation was measured at both gastrocnemii (GM, GL), both vasti (VM, VL) and the semitendinosus (ST). In each of the nine different angle-activation conditions ten impacts were initiated under the subjects' heel by means of the pneumatic impactor. The force was measured using a one dimensional transducer (Kistler®). Tibial and femoral shocks were measured using three dimensional accelerometers (Kistler®,  $m < 0.0025\text{kg}$ ). This abstract focuses on the longitudinal components only. The sensors were attached to Apex® pins (diameter 3.0mm, length 60mm) inserted under local anesthetic approx. 1.5cm into the right tibia and femur. The insertion locations were medial and approximately 5-7cm below (tibia) and 2-3cm above (femur) the knee joint space. After attaching the sensors their axes were aligned with the anatomical segment axes using a correction algorithm based upon reflective markers recorded by a movement analysis system (ProReflex®). The shock transmission through the knee joint was calculated by the ratio RAT of the acceleration maxima at the tibia (ACCtib) and femur (ACCfem) ( $\text{RAT} = \text{ACCtib}/\text{ACCfem} \cdot 100$ ). The linear displacement of tibia and femur was estimated by double integrating the acceleration time history and the knee compression (KOM) by the difference of tibial and femoral displacement. The sampling rate of all analog data was 1000Hz. An ANOVA ( $p < 0.05$ ) was carried out to identify significant differences of the mentioned parameters between knee angle or activation conditions.



**Figure 1:** Time normalized time history of mean ( $\pm$ sd) knee compression and femoral and tibial displacement of one subject achieved at 0° knee angle and 0% MVC level ( $n=10$ ).

### RESULTS AND DISCUSSION

In the described study conditions the average peak acceleration at the tibia varied between 2 and 4g and at the femur between 1.5 and 2.5g. With increasing muscle activation levels the tibia acceleration decreased significantly under all knee angles which was also true for the femur at 0° and 20°. ACCtib increased significantly with increasing angles at all MVC levels while ACCfem showed no systematic trend in all subjects. The average shock transmission over all trials was 59.7%. In the 0° and 20° knee angle condition three of the four subjects showed a significant increase in RAT of about 10% with increasing muscle activation levels while one subject showed no substantial change. In the 40° condition increasing muscle activation had a smaller effect on RAT compared to 0° and 20°.

Figure 1 shows the average segmental displacement for one subject at 0° knee angle and 0% MVC. In all knee angle conditions the relative axial movement of tibia and femur (KOM) was significantly decreasing with increasing muscle activation. Apparently the muscle forces pre-load the joint's peri- and intraarticular structures. Considering the facts that RAT was increasing while KOM was decreasing with higher muscle activity and that cadaver studies [1,2] have shown a shock attenuating effect of passive structures it could be assumed that the deformation of peri- and intraarticular tissue also has an effect on shock transmission in vivo. This effect could be substantially influenced by muscle activation.

### REFERENCES

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