Intraoperative direct mechanical stimulation of the anterior cruciate ligament elicits short and medium latency hamstring reflexes

Friemert B., Faist M., Spengler C., Gerngross H., Claes L., Melnyk M.

(*=this author deceased during the review process)

Running Head: hamstring reflexes after mechanical ACL stimulation

B. Friemert
Department of Trauma and Reconstructive Surgery, University Hospital "Rechts der Isar" of the Technical University of Munich, Ismaninger Str. 22, D-81675 Munich, Germany

M. Faist
Department of Neurology and Clinical Neurophysiology, University of Freiburg, Breisacher Str. 64, D-79106 Freiburg, Germany. Tel.: +49761-270-5333. Fax: +49761-270-5390
E-mail: faist@nz11.ukl.uni-freiburg.de

C. Spengler, H. Gerngross
Department of Surgery, German Armed Forces Hospital in Ulm, Oberer Eselsberg 40, D-89081 Ulm, Germany

M. Melnyk, L. Claes
Institute of Orthopedic Research and Biomechanics, University of Ulm, Helmholtzstrasse 14, D-89081 Ulm, Germany
Abstract

The anterior cruciate ligament (ACL) has not only a mechanical but also a sensorimotor function. Patients with injuries of the ACL frequently complain of knee instability despite good mechanical stabilization after surgical reconstruction. Compared to healthy subjects their latencies of hamstring reflexes after anterior tibia translation are considerably increased. There is evidence for the existence of a reflex arc between the ACL and the hamstrings. The aim of this study was to determine if there is a direct reflex response after an isolated mechanical stimulation of the ACL in humans.

In 10 patients who underwent arthroscopy hamstring electromyography (EMG) responses were assessed intraoperatively after applying an isolated load on the ACL. Latencies, amplitudes and integrals of the EMG responses were analyzed. In four patients, the measurements were repeated after injection of local anaesthetics into the ACL.

In all subjects, responses with mean (±SD) latencies of 42±4.4ms corresponding to a medium latency response (MLR) were found. In 7 subjects, they were preceded by responses with a short latency (SLR) of 24±2.7ms. The maximum amplitude was 8.6±7mV, the integral 0.064±0.05mV*s. The injection of local anaesthetics reduced the amplitude by 34±12% and the integral by 50±20%.

Direct mechanical stimulation of the ACL evokes considerably smaller SLRs and MLRs than anterior tibia translation during standing. It is argued that latency changes observed in patients with ACL ruptures may be rather due to changes in the sensorimotor integration of the afferent input from the knee joint than to the absence of the direct ACL reflex.

Key words:

intraoperative mechanical ligament stimulation, anterior cruciate ligament, hamstring reflex, short latency reflex, medium latency reflex
Introduction

The anterior cruciate ligament (ACL) has not only a mechanical but also a sensorimotor function. The ACL contains mechanoreceptors (Biedert et al. 1992; Raunest et al. 1998; Zimny et al. 1986), which belong to a complex proprioceptive system of the knee joint (Hogervorst and Brand 1998; Johansson et al. 1991; Krauspe et al. 1995; Miyatsu et al. 1993; Solomonow and Krogsgaard 2001). Recently it was shown that external perturbations applied to the knee evoked reflexes that significantly increased knee joint stiffness (Dhaher et al. 2004). Proprioception is impaired in the knees of patients with ACL rupture (Friden et al. 1996; Jerosch and Prymka 1996). Clinically, these patients may have symptoms of knee instability (giving way phenomenon) despite good mechanical stabilization after the surgical reconstruction of the ACL. Since most of the afferents arising from the ACL and other ligamentous structures play a role in reflexes that are integrated at the spinal level, oligosynaptic or polysynaptic pathways are involved (Nardone and Schieppati 1998; Solomonow and Krogsgaard 2001). An example of such an integrated reflex is the ACL-hamstring reflex, which is a sensorimotor unit combining the mechanical function of the ACL (to prevent anterior tibia translation) and the agonistic function of the hamstrings (to pull the tibia backwards) (Liu and Maitland 2000; Miyatsu et al. 1993; More et al. 1993). The existence of this reflex arc was described for the first time by Gruber et al (Gruber et al. 1986) and was later confirmed in experimental studies on cats (Solomonow et al. 1987) and humans (Krogsgaard et al. 2002).

Beard et al (Beard et al. 1994) measured the latency of hamstring reflex contractions after mechanically induced tibia translation in healthy control subjects and patients. They found that the latency was nearly twice as long in ACL-deficient legs. They postulated that the longer latency was caused by the loss of the ACL-hamstring reflex. Similar results were reported by Bruhn (Bruhn 1999), although they measured an increase in latency (approximately 6 ms) between injured and healthy subjects that was far less marked.
However, not only the receptors in the ACL but also the other receptors in the ligaments, the capsule and the knee-stabilizing muscles are excited by tibia translation. Friemert et al. (Friemert et al. 2005) were for the first time able to show that mechanically induced tibia translation in standing subjects evokes a complex multiphasic reflex response which consists of a short latency response (SLR) and a medium latency response (MLR). The SLR with a latency of approximately 20 ms was interpreted as the hamstring stretch reflex and, therefore, is mediated by Ia afferents (Faist et al. 1999; Friemert et al. 2005). The mechanisms underlying the generation of the MLR component of these complex signal responses have thus far not been studied in knee joints. Corna et al. (Corna et al. 1995) were the first who provided evidence that at least part of the MLR is mediated by secondary muscle spindle (group II) afferents, Similar findings have been reported by Schieppati and Nardone (Nardone and Schieppati 1998), Nardone and Schieppati (Nardone and Schieppati 1998), Bove et al (Bove et al. 2003), and by Grey et al (Grey et al. 2001), who investigated MLRs during locomotion. As far as we know, there are no studies looking at the influence of knee joint afferents on the SLR and the MLR.

In particular, it is unclear whether SLR and/or MLR components can be ascribed to the direct reflex pathways that link the ACL and the hamstrings. Therefore, the first aim of this study was to determine if there is a direct reflex response after an isolated mechanical stimulation of the ACL. The second question is whether such a reflex responses consists of different components corresponding to SLR and MLR.

**Patients and Methods**

**Patient population**

Our study included thirteen men with no history of neurological disorders (mean age: 24.6 ± 5.5 years, height: 179 ± 5 cm). All patients underwent arthroscopy for knee problems (7 x plica syndrome, 1 x meniscus, 2 x cartilage defects and 3 x for purely diagnostic purposes
due to anterior knee pain syndrome). No subject had a history of additional orthopedic disorders. In three cases, it was impossible to analyze the data due to a defective trigger in one case and massive artefacts in two cases. We were thus able to analyze the data obtained from ten patients. The patients gave their written informed consent in accordance with the Declaration of Helsinki. The study was approved by the ethics committee of the University of Ulm (No. 216/2002).

*Anesthesia*

All surgical procedures were performed under general anesthesia (total intravenous anesthesia, TIVA, with remifentanil 0.5-1.0 µg/kg and propofol 50-150 µg/kg/min, intubation). In the doses used here it is unlikely that the excitability of the monosynaptic reflex arc or neuromuscular function were affected (for details, see (Kerz et al. 2001)). Muscle relaxants (precurarization with rocuronium bromide 5 mg, then suxamethonium chloride 1-1.5 mg/kg) were used only at the induction of anesthesia. The half-time of this relaxation was ten minutes. The time between the induction of anesthesia and the beginning of measurements was 54.5 ± 9 minutes on average (positioning of the leg and the electrodes, sterile cleaning of the operating field, installation of the arthroscopy instruments, arthroscopic procedure and preparation of the measurement system). No further relaxants were used during anesthesia. Since they were only used in the initial part of the test, it is unlikely that they influenced neuromuscular excitation at the time when the measurements were performed (for details, see Beers and Camporesi 2004; Chang et al. 1994; Shiraishi et al. 1992; Suzuki et al. 1998). Last but not least neuromuscular function was assessed before the tests were started (2 Hz stimulation frequency, train-of-four ratio > 0.8 (Gatke et al. 2002)). Therefore, medication effects on muscular responses are unlikely.
**Measurement system**

A commercially available hook probe (Wolf®, Knittlingen, Germany) with a modified (rounded) tip was attached posteriorly and distally to the ACL (figure 1). In a few cases little synovia between the ACL and the posterior cruciate ligament had to be removed, to allow the hood to be placed correctly. No patient had severe adhesions or hyperplasia of the synovial membrane so that it was possible in all cases to place the hook in the appropriate position. A sterile thin wire rope was attached to the hook probe. A dynamic force of 300 N (see preparatory work below) was applied to the ACL. A load cell (0-2000 N, Biovision, Wehrheim, Germany) was placed between the wire rope and the weight in order to control the force applied to the ACL. Furthermore, it was used as a trigger signal (onset of force application to the ACL) to determine the onset latency of the EMG signals. Video recordings were made of all measurements.

**Measurements**

The patients were positioned for arthroscopic knee surgery on the operating table. A commercially available tourniquet (Ulrich Company, Ulm, Germany) was placed around the thigh as proximal as possible in order to cut off the blood supply during surgery. It was, however, not inflated before and during the measurements. The leg was then placed loosely on a leg holder. The EMG electrodes were positioned on the medial and lateral hamstrings, and the reference electrode was placed on the iliac spine (see below). The electrodes were taped to the skin in such a way that contact with the rinsing solution was avoided during arthroscopy. Once the skin had been cleaned in a standard manner and the leg draped, a diagnostic arthroscopy was performed to determine whether the patient had an intact ACL and could be included in the study. A medioventral approach was used to insert the hook probe into the knee. Immediately before the measurements were made, measurements at rest were performed to rule out adverse effects of the diagnostic arthroscopy on the EMG
recordings. A weight of 1kg was placed on the load cell, which was then checked again and calibrated to zero. Once the measuring system was attached to the ligament, 5 measurement series each consisting of 10 trials were performed. During each trial the predetermined force of 300 N (for calculation, see preparatory work) was applied. Four patients then received an injection of a local anesthetic (2 ml of 0.5% Scandicaine®) in the proximal and distal ends of the ACL. In this local anesthetic group (LA group), three further series of ten trials were performed five to ten minutes after the injection. During all measurements, the tibia was fixed manually to prevent anterior tibia displacement.

**Electromyography**

The surface EMG was recorded using pairs of disk electrodes (Arbo Ag/AgCl sensor, Tyco Healthcare, Neustadt, Germany; diameter: 0.5 cm, distance: 2 cm), which were placed over the muscle bellies of the medial and lateral hamstrings. The reference electrode was positioned on the iliac crest. The skin was shaved and cleaned with alcohol. Raw EMG data were collected at a sampling rate of 5 kHz, pre-amplified (x1000) and recorded with commercially available evaluation software (Daisy Lab Biovision, Weilheim, Germany). For further analysis, all EMG signals were band-pass filtered (35-700 Hz; analogue on the amplifier, Biovision, Weilheim, Germany) with an 8th order Butterworth filter to eliminate artefacts associated with the leg holder, which showed a frequency of 15-20 Hz in preliminary experiments. Then the signals were rectified and averaged. It has to be noted that, in comparison to a 6th order filter (10Hz), an 8th order high-pass filter (35Hz) may increase the latency assessed for the SLR by approximately 2 ms.

**Preparatory work**

As part of the preparatory work for this study, two sheep knee joints with intact capsulo-ligamentous structures were used to determine the forces to be applied. A miniarthrotenomy
was performed in order to mimic the experimental set-up described above for the human experiment. To compare the applied force used in the present study to previous studies, it was shown that a weight of 1.5 kg and a height of 30 cm was required to generate a force of 300 N. Solomonow et al (Solomonow et al. 1987) suggested that a minimum force of 130 N was needed to elicit a hamstring reflex. The tensile strength of an intact cruciate ligament has been reported to be 1725 ± 660 N in humans (Noyes and Grood 1976) and 1118 ± 244 N in sheep (Durselen et al. 1996). The two types of cruciate ligaments thus have similar tensile strengths. Once the two measurement series had been completed, the cruciate ligaments of the sheep knee joints were exposed and no damage to the ACLs could be seen. Thus, the intraoperative measurements are far below the thresholds to cause damage to the ACL.

**Data analysis**

The EMGs of both, the medial and the lateral hamstring muscles were used for analysis. Latencies were assessed as the time from the beginning of force application (trigger) to the first onset of the reflex response. The onset of the short latency response (SLR) was determined semi-automatically using the trigger signal from the load cell as the beginning of ACL stimulation. The first significant rise in EMG activity (defined as average amplitude plus ± 5 SD of the preloading baseline activity) was identified as the SLR. Then, the exact onset of this SLR was assessed by visual inspection on the computer screen using a cursor which was set at the beginning of the EMG deflection. Earliest responses with a latency of more than 30 ms (medial hamstrings: three cases; lateral hamstrings: two cases) were defined as pure medium latency responses (MLRs). In case of superimposed signal configuration we used our evaluation algorithm to differentiate MLR from SLR. Briefly, the duration of the hamstring SLR can be estimated quite accurately by multiplying the time between the onset of the SLR and the first SLR peak with the empirical factor of 3.28 as shown in detail in our previous paper (Friemert et al. 2005). The end of the MLR was
defined 30 ms after the onset of the MLR. The maximum amplitude and the integral from the SLR and MLR were determined. In addition the total duration of the reflex response was assessed. The end of the reflex response was defined as the point where the EMG returned to baseline for at least 10 ms. Due to artefacts which frequently occurred after the MLR response the total reflex duration could not be assessed reliably in all cases. An example of such artefacts is illustrated in figure 2a in the single sweep of the lateral hamstrings. The nature of these artefacts is most probably mechanically due to the weight connected with the rope and the leg holder used. In the LA group, we also measured the integrals and maximum amplitudes over a time window of 100 ms after the trigger signal (with and without local anesthetic) in order to assess the effects of local anesthesia. Strong suppression after local anesthesia made a separate analysis of SLRs and MLRs impossible. For this reason, only overall EMG activity was quantified and compared. Mean values, standard deviations and the range of values were calculated using SPSS® software.

Results

Reflex responses

All patients showed reflex responses after mechanical stimulation of the ACL. In seven patients, the first onset was observed after 23.9 ± 2.7 ms (range: 18 to 26 ms) in the medial hamstrings and was classified as an SLR. Detailed individual results of all subjects are given in table 1. All these seven patients showed a second reflex response (MLR) with an average latency of 40.9 ± 4.3 ms (range: 35 to 46 ms) after the end of the first reflex response. A typical raw EMG response is shown in figure 2a, the averaged data of 50 responses are shown in figure 2b. In three patients, the first onset was detected after 44 ± 1.2 ms (range: 43 to 45 ms), which corresponds to an MLR. These latencies are similar to the MLR latencies of those patients with a preceding SLR. The mean MLR latency of all subjects was 41.5 ± 4.4 ms (range: 35 to 45). Similar results were obtained for the lateral hamstring muscles,
although it was possible to analyze only eight out of ten subjects for technical reasons (see tables 1 and 2).

Amplitude/duration/integral

The mean amplitude of the SLR was 8.6 ± 7.1 mV (range: 1.9 to 16.8 mV) and of the MLR 8.9 ± 11.2 mV (range: 1.2 to 36.4 mV). The mean duration of the SLR was 17.1 ±3.7 ms (range 10.8 to 21.1 ms). The MLR integral was calculated on the basis of a 30 ms interval. The integral of the SLR showed a value of 0.064 ± 0.05 mV*s (range: 0.011 to 0.16 mV*s), the integral of the MLR was 0.17 ± 0.22 mV*ms (range: 0.014 to 0.68 mV*s). The mean duration of the overall reflex response was 74.4 ± 15 ms (range: 55 to 112 ms). There were no significant differences between the mean results for the lateral hamstrings and those for the medial hamstrings (table 1).

Local anesthesia

A 34% ± 12% (range: 30% to 54%) decrease in the EMG amplitude and a 50% ± 20% (range: 35% to 76%) decrease in the integral was observed in the LA group (figure 3).

Discussion

The purpose of this study was to investigate the ACL-hamstring reflex by direct isolated mechanical stimulation of the ACL. We were able to show that muscular responses were elicited corresponding to SLRs and MLRs with mean latencies of 23 ms and 42 ms, respectively. The significant decrease of the responses after the injection of a local anesthetic provides further evidence for a direct involvement of the ACL in the reflex pathway.

There are presently only two studies in the literature that assessed the latency of the ACL-hamstring reflex in humans by direct stimulation. Dyhre-Poulsen et al (Dyhre-Poulsen and Krogsgaard 2000) were able to show a latency of the muscular response of 95 ms with the
knee flexed at 30°. Unlike in our study they stimulated the ACL electrically using electrodes inserted into the cruciate ligament. They used current amplitudes of twice the patient’s individual sensory threshold. However, it remains unclear which receptors or neural pathways were stimulated by this technique, although the authors concluded that the muscular responses had been elicited by the stimulation of group II and III afferents. It should be kept in mind that group III fibers also transmit the sensation of pain. In addition, in their study five out of eight patients received a spinal anesthetic. Although the measurements were performed six to eight hours after the induction of anesthesia, an effect of this procedure on latency cannot be entirely ruled out. In addition, most patients complained of postoperative pain in the knee joint, which may have additionally influenced the reflex responses with longer latencies as was shown by Andersen et al (Andersen et al. 2000). The anesthesia used in the present study is unlikely to have relevantly affected neuromuscular transmission or central excitability at least as far as the SLR is concerned (see methods). Tsuda et al (Tsuda et al. 2003) examined cruciate ligament grafts in patients who had undergone surgery. Following electrical stimulation, they were able to show latencies of 120 ms. This value approximately corresponds to the time window described by Dyhre-Poulsen and Krogsgaard (Dyhre-Poulsen and Krogsgaard 2000). These long latencies now raise the question of whether the short latencies measured in the present study are realistic.

In the cat, Solomonow et al (Solomonow et al. 1987) reported an SLR that was attributed to the high mechanical load according to Dyhre-Poulsen et al (Dyhre-Poulsen and Krogsgaard 2000). The fact that the mechanical stimulus evokes a considerably larger response indicates that this stimulus is more effective than the one applied electrically. In accordance with the recommendations of Solomonow et al., we used such a high mechanical load (300 N). As described by Freeman and Wyke (Freeman and Wyke 1967), the ACL contains Golgi corpuscles with afferents showing a conduction velocity of 75 m/s, which approximately corresponds to the conduction velocity of group Ia afferents. In a subject with a height of
185 cm, the distance from the knee joint to the lumbar segments L3-L4 and back to the mid-thigh is approximately 120 cm (Gomez-Barrena et al. 1996). Accordingly, the conduction time can be estimated at around 16 ms. Assuming that a synaptic connection accounts for a delay of 1 ms and 3 ms at the motor end-plate (Solomonow and Krogsgaard 2001), our reflex latencies appear to be realistic. The injection of lidocaine into the ACL led to a 50% decrease in the integral and a 34% decrease in the amplitudes. These findings support the view that the ACL is involved in these muscular responses. In a recently published study, Friemert et al (Friemert et al. 2005) reported a latency of 21 ms for the SLR after mechanical anterior tibia translation. In accordance with the results obtained by Faist et al (Faist et al. 1999) for the tendon jerk reflex, this finding was interpreted as a reaction associated with the hamstring stretch reflex mediated by group Ia muscle spindle afferents. Our intraoperative measurements showed a mean maximum amplitude of 8.6 mV and a mean integral of 0.064 mV*s. In order to compare the present data with effects observed after the stimulation of all knee structures, we analyzed the data obtained in the experiments for our previous study (Friemert et al. 2005) in the same way using an 8th order Butterworth high-pass filter (35 Hz). We found a mean maximum amplitude of 234 mV and a mean integral of 7 mV*s for those experiments performed in a standing position. This implies that the responses measured during the standing position are 30-fold larger than those obtained intraoperatively during lying (or 100-fold larger if the integrals are considered). At the same time, the background EMG only shows a 5-fold increase. It is unlikely that the different positions alone induce such a strong suppression of the SLR and MLR responses. Thus, the results of the present study strongly suggest that only a minor part of the reflex response is mediated by the direct ACL-hamstring reflex pathway. In patients with ACL ruptures, the latencies of hamstring responses after anterior tibia translation were nearly twice as long as in normal subjects (Beard et al. 1994; Bonfim et al. 2003). According to the present results it appears unlikely that these results are directly attributable to the impaired ACL-hamstring reflex.
This view was already suggested by Hogervorst and Brand (Hogervorst and Brand 1998). A possible explanation are changes in the sensory-motor integration, i.e. that the longer latencies are secondary to a change in afferent information, which may cause inhibitory or excitatory effects on different interneurones or changes in the activity of afferents. Johansson et al (Johansson et al. 1991) suggested that the gamma motor neurons may play a crucial role in transmitting information from the cruciate ligament which may alter the entire regulation of muscular activity and consequently could also affect the latency. Johansson et al (Johansson et al. 1990) and Sojka et al (Sojka et al. 1989) were able to show that afferents from the anterior/posterior cruciate ligament have a direct effect on the gamma motoneuron system. They recorded electrical potentials from primary muscle spindle afferents under various stretch load conditions. The study also included tests in which loads of 5 to 40 N were applied to cruciate ligaments. Compared with the unloaded legs, the loaded legs showed a marked increase in the activity of the muscle spindle afferents. Lundberg et al (Lundberg et al. 1978) found that alpha motoneurons responded only weakly to very high electrical stimulations in the posterior articular nerve (PAN), whereas 93% of the gamma cells reacted to similar stimulations (Johansson et al. 1986). This could explain why a cruciate ligament rupture is not associated with the absence but with a latency shift of the reflex response (Beard et al. 1994). The large differences in reflex size suggest that the direct ACL-hamstring reflex after intraoperative isolated stimulation is only a small component of the overall response after tibia translation during standing (Friemert et al. 2005). In conclusion, the direct ACL-hamstring reflex pathway seems to be comparably weak and should not cause relevant latency shifts after ACL ruptures. Therefore, the latency changes that were observed in patients with ACL ruptures may be the result of changes in the sensorimotor integration of the afferent input from the knee joint.

Acknowledgements: This work was supported by the Fraunhofer grant (FV InSan I 0102-V-1105).
References


**Figure Legends**

**Figure 1:**
Experimental setup and the measurement system that was used to apply a force to the ACL during arthroscopy. A hook probe with a modified tip was placed around the ACL and grasped the entire ligament. A sterilely wrapped wire rope, a load cell/trigger and a weight were then connected. Data from the load cell/trigger and EMG data were recorded on a computer and analyzed off-line.

**Figure 2:**
A: Single (rectified and non-rectified) EMG sweeps of the medial (first and second rows) and the lateral hamstrings (third and fourth rows) and load cell/trigger signal (bottom row). In the single sweep of the lateral hamstrings typical artefacts occurring after the MLR can be seen. B: Averaged EMG data (n=50 trials) of the same subject. First row = rectified EMG recorded from the medial hamstrings; second row = rectified EMG recorded from the lateral hamstrings; third row = trigger signal.

\( \downarrow = \text{triggerpoint} \quad \downarrow = \text{SLR onset} \quad \downarrow = \text{MLR onset} \)

**Figure 3:**
EMG sample (mean of 30 trials) obtained from a patient with local anesthesia. Figure 3a shows the EMG signal and the trigger before the injection. This subject is one of those who only showed an MLR. Figure 3b shows the EMG signal and the trigger after the injection. There is virtually no reflex response. First row = rectified EMG recorded from the medial hamstrings; second row = trigger signal.
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Table 1: Individual results and mean values. Missing values for SLR indicate that there was only an MLR response; lat=lateral; med=medial; ham=hamstring; SD=standard deviation; n=number of subjects.
Table 2: Pattern of SLR and MLR responses for each subject.

+ = response, - = no response, tech = no EMG signal for technical reasons.
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\[\text{\small \uparrow} = \text{triggerpoint} \quad \text{\small \uparrow} = \text{SLR onset} \quad \text{\small \downarrow} = \text{MLR onset}\]
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