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Reducing anterior tibial translation by applying functional electrical stimulation in dynamic knee extension exercises: Quantitative results acquired via marker tracking

Chiun-Fan Chen [a,f], Yi-Hsuan Kuo [b], Jer-Junn Luh [c], Yu-Jen Chen [c], Shih-Wei Chen [a], Te-Son Kuo [a,b,d], Jin-Shin Lai [e,f,*]

[a] Department of Electrical Engineering, National Taiwan University, Taiwan
[b] Graduate Institute of Biomedical Electronics and Bioinformatics, National Taiwan University, Taiwan
[c] School of Physical Therapy, National Taiwan University, Taiwan
[d] Institute of Biomedical Engineering, National Taiwan University, Taiwan
[e] Department of Physical Medicine and Rehabilitation, National Taiwan University Hospital, Taiwan
[f] Health Science and Wellness Center, National Taiwan University, Taiwan

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ABSTRACT

Background: Pain that accompanies anterior cruciate ligament deficiency during dynamic knee extension exercises is usually caused by excessive anterior tibial translation, which can be restricted if the anterior cruciate ligament was intact.
Methods: A functional electrical stimulator is incorporated with a training device to induce hamstring contractions during certain degrees of knee extension to replicate effects similar to those generated by an intact anterior cruciate ligament and to reduce anterior tibial translation. By using a camera that tracks markers placed on bony prominences of the femur and tibia, the anterior tibial translations corresponding to various settings were determined by customized image processing procedures.
Findings: In the electrical stimulation sessions, the knee extensions with electrical stimulation feedback induced significantly ($n = 6$, $P < .05$) less anterior tibial translation over the range of 20 to 50° when compared to those using the standard isokinetic shank restraint. Likewise, the knee extensions with an anti-shear device that blocks tibia displacement mechanically also induced significantly ($n = 6$, $P < .05$) less anterior tibial translation, but over a different range of knee extension (30 to 70°).
Interpretation: Despite the fact that both the electrical stimulator and the anti-shear device assisted in reducing anterior tibial translation, the tendency of the curves generated with the functional electrical stimulation was generally more similar to those generated when using the standard isokinetic shank restraint.

1. Introduction

Rupture of anterior cruciate ligament (ACL) in the knee joint is a common sports injury that causes knee instability and quadriceps atrophy (Ageberg, 2002; Ingersoll et al., 2008; Van Grinsven et al., 2010; Williams et al., 2005). Rehabilitation is generally recommended to avoid further deterioration of the quadriceps regardless of whether reconstruction surgery is recommended (Ageberg et al., 2008; Andersson et al., 2009; Frobell et al., 2010; Meunier et al., 2007). Notwithstanding the fact that dynamic knee extension exercises (i.e., both open-kinetic-chain and closed-kinetic-chain exercises) have been regarded as good rehabilitation approaches for ACL deficiencies (Fleming et al., 2005; Heijne and Werner, 2007; Kozánek et al., 2011; Mesfar and Shirazi-Adl, 2008; Morrissey et al., 2002; Tagesson et al., 2008), pain associated with instability (Kvist, 2005; Marti et al., 2004), i.e., the pain generally caused by anterior tibial translation (ATT), which induces primary (e.g., mechanical collision) and secondary (e.g., inflammatory synovitis) injuries, makes such training methods difficult or unsuitable in early intervention of clinical practice. This suggests a need to look further into the ACL to determine whether a certain device or system was appropriate for replicating the mechanism of this tough and fibrous ligament to reduce ATT during dynamic exercises.

The ACL originates from the medial side of the lateral femoral condyle and inserts into the tibial plateau (Amis and Dawkins, 1991; Arnoczky et al., 1983). The origin and insertion of this ligament assist in restricting ATT and medial rotation of the tibia in relation to the femur. A partial or complete tear of the ACL leads to unrestrained ATT and dysfunctions that include a partial or complete loss of neuromuscular reflexes (e.g., the ligamento-muscular protective reflex) (Chu et al., 2003; Dyhre-Poulsen and Krogsgaard, 2000; Krogsgaard et al., 2002; Solomonow, 2006; Solomonow and Krogsgaard, 2001; Solomonow et al., 1987). The lack of a normal protective reflex lessens sensorimotor control of the hamstring when tension is

* Corresponding author at: 7, Chung-Shan S. Rd., Taipei, Taiwan.
E-mail address: jslai@ntu.edu.tw (J.-S. Lai).

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applied to a deficient ACL (Shelburne et al., 2005; Yanagawa et al., 2002). Hence, a possible method to restore the normal functions may be achieved by intentionally activating hamstring muscles during knee extension. In this study, a feedback system is implemented by incorporating functional electrical stimulation (FES) into an isokinetic training device. The FES was applied according to the concept of the "Smart ACL Brace" (Solomonow, 2006), which served as an "active" device that stimulated the hamstring at certain degrees of knee extension to partially reproduce normal functions triggered through the neuromuscular protective reflex. The FES was compared with a Johnson anti-shear (JAS) device (CSMi, Stoughton, MA, USA), which "passively" blocks tibia displacement during dynamic knee extensions. The purpose of the study was to compare the active and passive approaches with the standard approach that applies only a standard isokinetic shank restraint, while the results are quantified by tracking ATT to determine whether the FES or JAS device reduces ATT during the dynamic knee extension exercises. Knee extension torque was also monitored to observe the extent of reciprocal inhibition of the quadriceps caused by the application of FES to the hamstring (Harrison and Zytnicki, 1984; Laughman et al., 1983).

2. Methods

2.1. Experimental setup and system configuration

Six male volunteers (age: 24–32 years old, weight: 69–85 kg, height: 1.75–1.82 m) with no records of knee instability or surgery were recruited in this study. The participants were given informed consent according to guidelines approved by the institutional research ethics committee (NTUH-REC no.: 201002029D) prior to all the experiments. All subjects participated in two different sessions, where knee exercises were either performed with FES feedback or with a JAS device. The same knee exercises with only the shin pad worn were also performed by the same subjects in both sessions to allow comparisons between the effects of only the shin pad and the ATT limiting accessory (i.e., either the FES or JAS device). A system including an isokinetic training device (CYBEX NORM, CSMi, USA) was configured (block diagram provided in Supplementary Data) to operate with the shin pad (the standard isokinetic shank restraint) and with or without FES or with the JAS device without FES during knee flexion and extension exercises. The system was designed to trigger FES at an appropriate range of knee extension and demonstrate mechanical data with images acquired during exercise.

The subjects were restrained to the isokinetic training device in a seated position to perform knee flexion and extension exercises. The training device was set at an angular velocity of 60° per second (default value of the isokinetic training device) and each subject performed five consecutive knee flexions and extensions for each session. Subjects were asked to produce maximal contractions and the extension that induced the greatest peak torque was selected for analysis. Position, torque and image signals related to the knee joint were acquired from the dynamometer of the training device and a camera (SI-520, VION, Taipei, Taiwan), where the position and torque signals were used as the basis of FES control. The lens of the recording camera was set parallel to the medial side of the moving knee in order to capture images of markers placed on the bony prominences of the knee joint. The image signals (720×480, NTSC) were encoded by a video converting interface (C-200 plus, Compro, Taipei, Taiwan) at a rate of 30 frames per second while the torque signals were digitalized at a sampling rate of 2000 Hz by using a data acquisition device (USB-6211, National Instruments, Austin, TX, USA). The processed data were transferred to a personal computer loaded with a LabVIEW (National Instruments) graphical user interface (GUI). A custom-made FES device (details are provided in Supplementary Data) that referenced a previous circuit design (Chen et al., 2012) was controlled via the GUI with respect to the data acquired from the training device to induce co-contraction of the hamstring and the quadriceps. Specific timings of the electrical stimulation were based upon earlier studies on the strain of ACL at different degrees of knee extension (Beynnon et al., 1992; Draganich and Vahey, 1990; Hirokawa et al., 1991; Li et al., 2005; Renstrom et al., 1986). Stimulation pulses that are voltage-controlled, charge-balanced and biphasic with slow reversal (Merrill et al., 2005) were generated by a microcontroller (MSP430, Texas Instruments, Dallas, TX, USA), level shifters, power amplifiers and isolation transformers in the FES device. Commands indicating the stimulation intensity and frequency were received by the FES from the computer through a serial port (RS-232). The stimulation intensity was controlled between a maximum voltage V_{max} (approximately 12 V) that induced a painless contraction and a minimum voltage V_{min} (approximately 6 V) that initiated at least a twitch while the frequency was set at around 30 Hz to generate a tetanic pulling force. The intensity was also modulated according to the peak torque T_{peak} and instantaneous torque T_i corresponding to the knee extension of each subject, where T_{peak} was determined by the isokinetic training device in a rehearsal that took place days before the actual experiments of all participants. In the case when T_i is smaller than T_{peak}, the output voltage V_o is

$$V_o = \frac{T_i}{T_{peak}} \times (V_{max} - V_{min}) + V_{min}. \quad (1)$$

On the other hand, when T_i is greater than or equal to T_{peak}, the output voltage V_o is

$$V_o = V_{max} \quad (2)$$

(please note that we limited the output voltage V_o to be less than or equal to V_{max} for safety reasons). Based upon the foregoing parameters, stimulation pulses (active pulse width: 200 μs; charge balancing pulse width: 800 μs; intensity inversely proportional) were delivered to the hamstring (i.e., the semitendinosus and the biceps femoris) via disposable self-adhesive electrodes (SW5320151A, Unomedical Ltd., Singapore, dimensions: 3.8 cm × 5.1 cm) to generate contractions that imitate the protective reflex.

2.2. ATT measurements via marker tracking

Markers in the form of circular stickers were tracked throughout the sessions to measure the amount of ATT. As illustrated in Fig. 1, a red marker (diameter: 1 cm) was placed on the immediate surface over the medial epicondyle of the femur; a yellow marker (diameter: 1 cm) was placed on the immediate surface over the medial condyle of the tibia; and a green marker (diameter: 1.5 cm) was placed on the surface 2 cm posterior to the tibial tuberosity. Note that the dimensions of the markers were determined by their adherence to the skin surface at their respective locations. Error associated with skin movement was minimized, since we ensured that the shifting of marker locations (relevant to the bony prominences) was limited during repeated knee flexions and extensions performed before the experiments. The NI Vision Assistant (National Instruments) was used to detect the color differences between each marker and its surrounding skin (Fig. 2). Color thresholds were applied to the planes of hue–saturation–value to delineate the contours of the markers. High-level operations on blobs in binary images were then performed by removing small objects and filling holes. Arithmetic and logical operations were also performed to mask with the original image. The center of each marker was then determined to make further calculations of ATT. Details for deriving the amount of ATT were based upon the diagram in Fig. 3, where the red, yellow and green markers were labeled as A, B and C (or A', B' and C'),