

# The Effects of Robotic-Assisted Locomotor Training on Spasticity and Volitional Control

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**Abstract**—We studied the effects of Robotic-Assisted Locomotor (LOKOMAT) Training on spasticity and volitional control of the spastic ankle in persons with incomplete Spinal Cord Injury (SCI). LOKOMAT training was performed 3 days/week during a 1-hr period including set-up time with up to 30 minutes of training during a single session. The training was provided for 4 weeks and subjects were evaluated before and after 1, 2, and 4 weeks of training. Spasticity was characterized in terms of neuromuscular abnormalities associated with the spastic joint. A system identification technique was used to quantify the effects of LOKOMAT training on these neuromuscular abnormalities. The effect of LOKOMAT training on volitional control was determined by measuring isometric maximum voluntary contraction (MVC) of ankle extensor and flexor muscles. Our results indicated that the reflex stiffness, abnormally increases in SCI, was significantly reduced (up to 65%) following 4-weeks of LOKOMAT training. Similarly, intrinsic (muscular) stiffness, which also abnormally increases in SCI, decreased significantly (up to 60%). MVCs were increased substantially (up to 93% in extensors and 180% in flexors) following 4-week training. These findings demonstrate that LOKOMAT training is effective in reducing spasticity and improving volitional control in SCI.

**Keywords**—locomotion, robotic, voluntary movement, spasticity, stiffness, reflex, kinematics, ankle, spinal cord injury

## I. INTRODUCTION

Approximately 200,000 individuals in the US have sustained a spinal cord injury with an incidence of up to 10,000 new cases every year. Of these, approximately 50% have motor incomplete lesions, which result typically in impaired voluntary muscle activation and increased fatigue. In addition, virtually all patients with SCI display some form of spasticity, including exaggerated stretch reflexes and involuntary muscle spasms that disrupt daily activity, cause shorter life span, and have physical, emotional and social costs. An effective treatment of spasticity could therefore have many benefits. Furthermore, the underlying causes for the loss of volitional muscle control and the origins of spastic behaviors are incompletely understood.

A specific type of intervention that has been shown to greatly enhance ambulation following SCI is body weight supported treadmill training (BWSTT), in which patients are

unloaded over a treadmill and manual assistance is provided to simulate walking activity [1-5]. This specific intervention is, however, physically demanding and time consuming, often requiring three therapists to perform effectively[1]. Recent advances in technology have prompted the use of robotic devices to assist therapists in the rehabilitation of patients with neurological injury (reviewed in [6]). Colombo and colleagues have developed a driven gait orthosis that operates by computer interface with a motorized treadmill (LOKOMAT, Fig. 1, [7]). Use of the LOKOMAT is similar to BWSTT but provides swing and stance assistance through a motorized exoskeleton. Actuators at bilateral hip and knee joints programmed by dual personal computers and a current controller allow the LOKOMAT to mimic a physiological gait pattern that will provide the necessary afferent input to improve locomotion.

The merging results have shown that the LOKOMAT training can improve over-ground speed, gait endurance, temporal/spatial characteristics of gait, and improved temporal patterns EMG activities [8-9]. However, the effects of such training on abnormal neuromuscular properties associated with spastic hypertonia are completely unknown, due to lack of a quantitative objective tool.

Accordingly, we used the novel system identification technique to characterize the effects of LOKOMAT on neuromuscular abnormalities associated with spasticity. We also quantified the effects of this intervention on volitional control by measuring isometric maximum voluntary contractions.

## II. EXPERIMENTAL PROTOCOL

### A. Robotic-Assisted Locomotor (LOKOMAT) Training

Locomotor training utilizing the LOKOMAT is accomplished by 4 DC motors aligned at the hip and knee joints moving the in a physiological gait pattern timed to the treadmill belt speed. Control of the LOKOMAT consists of a host personal computer (PC), a target PC, and a current controller. The operator controls the LOKOMAT via a user interface that is programmed in LabView, with subject safety ensured by an accessible panic switch and monitoring by therapists (for full description, please see [7]).



Figure 1: Robotic-assisted locomotor (LOKOMAT) training apparatus (from Hocoma website).

Subjects were fitted in the overhead harness /counterweight system and unloaded by a counterweight. Subjects were fitted into the LOKOMAT by stabilizing the trunk and pelvis, aligning the hip and knee joints at the motor axes, and stabilizing the legs with fitted cuffs (Fig. 1). The foot was controlled with adjustable elastic restraints attached from the metatarsal heads and heels to the shank segment of the exoskeleton, maintaining the ankles in neutral ( $90^\circ$ ) position.

LOKOMAT training was performed 3 days/week during a 1-hr period including set-up time with up to 30 minutes of training during a single session. Treadmill speed was started as low as 1.0 kmph and increased up to 2.5 kmph as tolerated. Total training time, speed, distance, and amount of unloading were recorded during each session. Subjects were encouraged to stepping behavior “as much as possible”, with relative perceived exertion, blood pressure, and heart rate assessed every 10 minutes. Training was provided for 4-weeks.

### B. Experimental Setup

Ten incomplete SCI subjects with different degrees of spasticity were examined. The joint stretching device was used to operate as a position control servo driving ankle position to follow a command input. Subjects were seated and secured in an adjustable, experimental chair with the ankle strapped to the foot rest and the thigh and trunk will be strapped to the chair. The seat and foot rest will be adjusted to align the ankle axis of the rotation with axis of the force sensor and the motor shaft (Fig. 2).

Joint position, velocity and torque were recorded by a potentiometer, tachometer & torque transducer, respectively. Electromyograms (EMGs) from tibialis anterior (TA) and gastrocnemius (GS) were recorded using bipolar surface electrodes. These signals were filtered at 230 Hz to prevent aliasing, and sampled at 1 kHz by a 16 bit A/D converter.

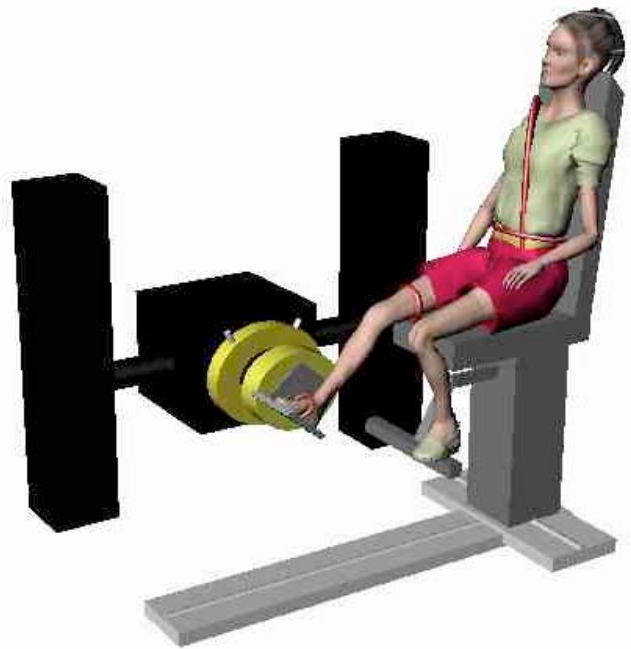


Figure 2: Experimental setup

### C. Operating Conditions

A series of pseudorandom binary sequences with the amplitude of 0.03 rad and a switching-rate of 150ms were used to perturb the ankle at different positions from  $60^\circ$  plantarflexion to  $25^\circ$  dorsiflexion, at  $5^\circ$  intervals with knee  $60^\circ$  flexion. A  $90^\circ$  angle of the ankle joint was considered to be the neutral position (NP) and defined as zero. These experiments were conducted under passive condition on the more spastic side. Plantarflexion was considered negative by convention.

MVCs were determined by having subjects contract the ankle muscles maximally toward dorsiflexion and plantarflexion at  $90^\circ$  flexion; torque and EMGs were sampled for 5 s.

## III. ANALYSIS METHODS

### A. Parallel-cascade Identification Technique

Intrinsic and reflex contributions to the ankle stiffness dynamics were separated using a parallel-cascade identification technique [10-11].

Intrinsic stiffness dynamics were estimated by determining the Impulse Response Function (between position and torque). Reflex stiffness dynamics were modeled as a differentiator, in series with a delay, a static nonlinear element and then a dynamic linear element. Reflex stiffness dynamics were estimated by determining the impulse response function, between velocity as the input and the reflex- torque as the output, using Hammerstein identification methods.

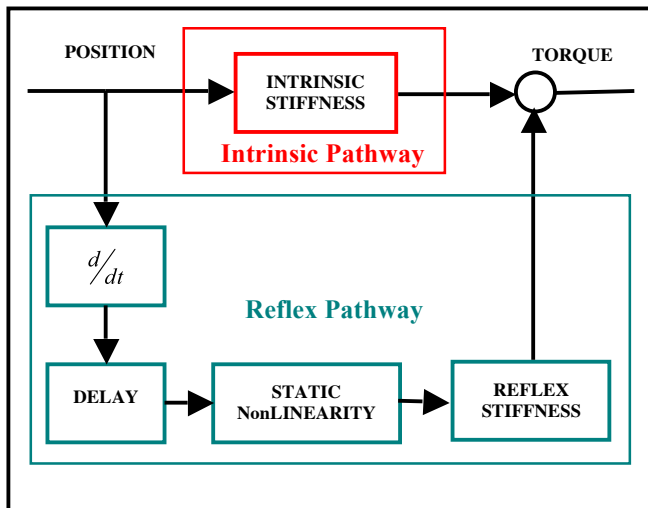


Fig. 3: The parallel-cascade system identification Model

### B. Volitional Control

The volitional control was quantified by measuring MVCs of ankle dorsiflexors and plantarflexors. The trial was repeated 3 times and the measurements were averaged.

## IV. RESULTS

### A. Passive Stiffness Gain vs. Ankle Position

Figure 4 shows  $K_p$  and as a function of ankle position before and 1, 2 and 4 weeks after using Lokomat training for a patient.  $K_p$  did not change during the first week but it decreased by  $\sim 35\%$  and  $\sim 60\%$  after 2 and 4 weeks of using Lokomat training, respectively. This reduction happened at dorsiflexed positions where intrinsic stiffness usually increases abnormally in spastic subjects.

### B. Reflex Stiffness Gain vs. Ankle Position

Figure 5 shows the reflex stiffness gain ( $G_R$ ) as a function of ankle position before and 1, 2 and 4 weeks after using Lokomat training for a patient.  $G_R$  was continuously reduced and declined by  $\sim 65\%$  after 4 weeks of LOKOMAT training. Similar to the intrinsic stiffness, this reduction mainly happened from middle plantarflexion to full dorsiflexion. This reduction was consistent in the LOKOMAT group but the percentage of changes was different.

### C. Maximum Voluntary Contraction (MVC)

Our results showed that MVCs of ankle flexors and extensors increased significantly up to 180% and 93%, respectively.

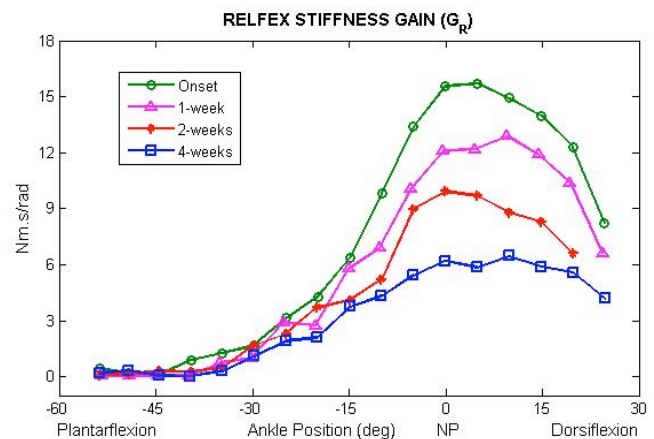


Fig. 4: Intrinsic stiffness ( $K_p$ ) versus ankle position before and after 1,2, and 4 weeks of LOKOMAT training.

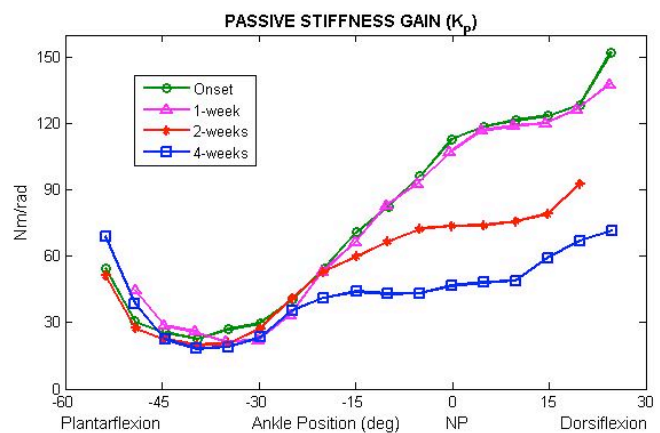


Fig. 5: Reflex stiffness ( $G_R$ ) versus ankle position before and after 1,2, and 4 weeks of LOKOMAT training.

## V. DISCUSSION AND CONCLUSIONS

The parallel cascade identification technique has been successfully used to characterize the effect of LOKOMAT training on ankle stiffness and its reflex and intrinsic components.

Both reflex and intrinsic stiffness, increased abnormally in SCI, were significantly reduced following 4-weeks LOKOMAT training. These improvements could be at least partially due to stretching the ankle (from foot drop to the neutral position) during the training, which can modify the ankle neutral position and as a result change its reflexive and intrinsic properties.

Maximum voluntary contraction of ankle extensors and flexors were improved significantly following 4-weeks LOKOMAT training. Further investigation is needed to find whether this improvement is because of a direct result of the training or is due to a decrease in spasticity.

These findings demonstrate that LOKOMAT Training has a potential to modify the responses of various spinal pathways, which can modify neuromuscular properties and their recovery. Thus, LOKOMAT training can be considered as an effective physical intervention in SCI.

## ACKNOWLEDGEMENT

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