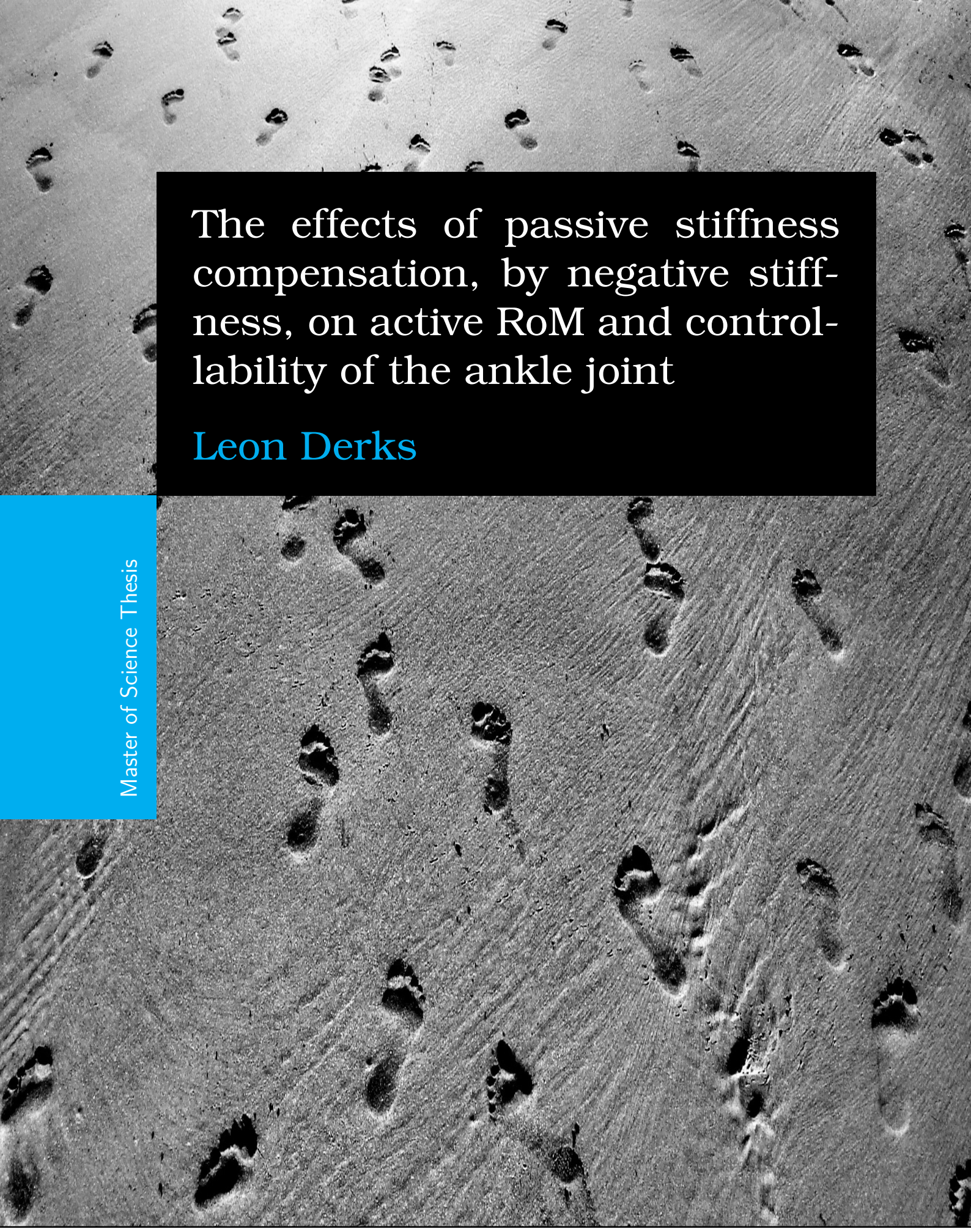

The effects of passive stiffness compensation, by negative stiffness, on active RoM and controllability of the ankle joint

Leon Derks

Master of Science Thesis

TU Delft Delft University of Technology

The effects of passive stiffness compensation, by negative stiffness, on active RoM and controllability of the ankle joint

Master of Science Thesis

For the degree of Master of Science in Biomedical Engineering at Delft University of Technology

Leon Derks

14-01-2013

Faculty of Mechanical, Maritime and Materials Engineering (3mE) · Delft University of Technology

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Preface

During my master Biomedical Engineering I developed an interest for the research and design of assisting devices for patients with an impaired motoric system. Looking for a graduation project I went by several people at the faculty. Erwin de Vlugt told me about a possible project on developing a Ankle Foot Orthosis (AFO) using something he called negative stiffness. After a talk about his ideas I knew I had found my graduation project. In this project I could combine the design and research of an assisting device for the ankle joint.

Before I started with my thesis project I did an internship and a literature study. I did a three month internship at Noppe Orthopedie, Noordwijkerhout. Noppe, part of OIM orthopedie, is a company specialized in making orthopedic devices. During my literature study I looked into different types of AFOs and into the pathophysiology of patients in need of AFOs.

Next to my internship I worked together with Freek Verbakel, also a master student, on the development of a first prototype of the negative stiffness AFO. Freek Verbakel continued developing the prototype during his graduation project. The goal of my graduation project was to test the effects of the negative stiffness AFO on Upper Motor Neuron Disease (UMND) patients. Because the prototype was still under development I performed my experiments with a virtual programmed negative stiffness AFO on "the Achilles" in the Leiden University Medical Center (LUMC).

Due to regulational limitations with "the Achilles" it was not possible for me to do research on a large group of patients. However I was still able to include a group of ten healthy subjects and two UMND patients in my experiments.

I would like to thank my direct supervisors Erwin and Jurriaan for all their help and interesting discussions during the course of the project. I would like to thank the people from the LUMC department of of Rehabilitation, especially Carel Meskers for helping with the patient measurements. I really enjoyed all the 4 o'clock Cup-a-Soup breaks with the fellow graduation students. Thank you Jeffrey for revising the report. Thank you Marleen for your help with making the figure look nice and all your support! And finally I would like my parents for their support!

Scientific Paper

The effects of passive stiffness compensation, by negative stiffness, on active RoM and controllability of the ankle joint

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Abstract

Introduction: Paresis in UMND patients leads to immobilization and disuse of the ankle joint. As a result, secondary changes, such as increased passive stiffness and plastic CNS rearrangements, aggravate the paresis. We propose an intervention that compensates the passive stiffness, enabling the remaining muscle force to initialize movement to prevent immobilization and disuse and to maximize controllability. Passive stiffness compensation is achieved by applying negative stiffness. The research question is: Does negative stiffness increase active RoM and how does this effect controllability of the ankle joint?

Methods: By applying, subject specific, negative stiffness on an electrical joint manipulator the change on active RoM and controllability of the ankle joint by different levels of stiffness compensation is assessed. Ten healthy and two UMND patients participated in this study.

Results: Active dorsiflexion RoM increased with 27.8% with the largest negative stiffness (50%) compensation ($p=0.001$) compared to the dorsiflexion RoM in normal (0%) compensation. Muscle activation did not differ between the conditions. Jerk of the ankle was 59.5% ($p<0.001$) of the normal (0%) compensation condition with 50% negative stiffness.

Conclusion: Negative stiffness compensation enabled the subjects to achieve a larger dorsiflexion RoM using the same voluntary muscle activation. To evaluate the effects of negative stiffness in walking additional experiments are needed.

Chapter 1

Introduction

Gait of Upper Motor Neuron Disease (UMND) patients is often inefficient and walking distance and velocity are limited, this has a big impact on their autonomy and quality of life [1]. Inefficient gait is related to reduced Range of Motion (RoM) of the ankle joint, especially in dorsiflexion direction [2, 3]. Lack of dorsiflexion RoM causes inability to lift the foot adequately in swing phase, resulting in low walking speed, increased energy cost and risk of falling. This gait pattern is known as drop foot [4, 5].

The reduced RoM in these patients is a result of an interplay between failure of voluntary contraction (paresis) and increased joint stiffness. Initial disruption of the central execution of motor command causes paresis i.e. failure of voluntary contraction reducing maximal voluntary torque [6]. As a result of reduced voluntary torque the patient cannot move their ankle in full dorsiflexion and loses controllability over the joint. The initial paresis also leads to mobility reduction and disuse of the joint. Movement reduction and immobilization lead to contracture, i.e. shorter muscles, reduced number of sarcomeres and increased passive stiffness and viscosity of connective tissue [7, 8, 9, 10]. As a result of shorter muscles and the paresis the force-length relation of the muscles are changed. Paresis lowers the (optimal) force and muscle shortening leads to a decrease in the optimal muscle length [11] (Figure 1-1). In addition, disuse of the joint causes further plastic rearrangements in the Central Nerve System(CNS) resulting in aggravation of the initial paresis. To improve gait in UMND patients the downwards cycle of paresis-immobilization/disuse-paresis should be interrupted or even be reversed [6].

In current treatment strategy this is not the case. In fact the opposite is true; some of the current treatment methods aggravate immobilization and disuse. Ankle foot orthoses (AFOs) are orthotic devices that are often used to prevent drop foot [4]. The most common AFO is the rigid dorsal AFO, that fixates the foot perpendicular to the lower leg. Although some studies indicate that rigid dorsal AFOs are able to prevent drop foot [12, 13], the ankle joint is immobilized and disused [6, 14]. Another type of AFOs are externally powered AFOs [15, 16]. In an externally powered AFO an actuator moves the ankle in the desired position during gait. External powered AFOs improve walking and mobilize the joint. However, as the actuator takes over the power generating and controlling function, the central neural command and muscles are disused.

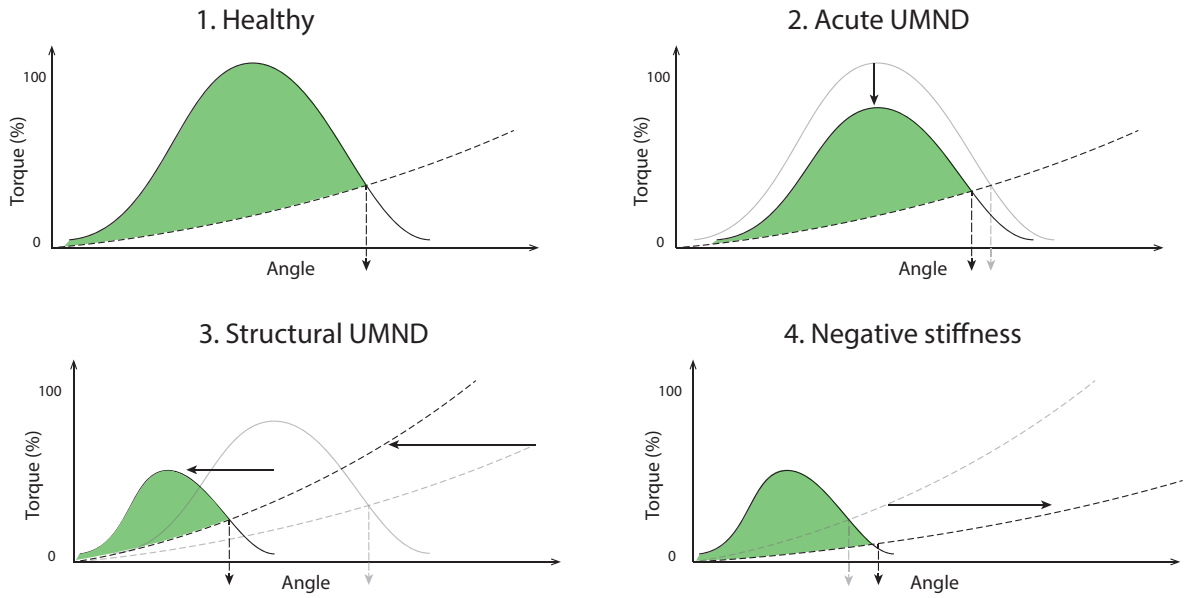


Figure 1-1: Pathophysiology. The interplay between active or voluntary contraction (solid) and passive stiffness (dotted) determines the active RoM (green area) and maximum dorsiflexion angle (dashed arrow). Figure 1. shows the healthy situation. Figure 2. shows the acute result of initial paresis after the central disruption; lower active torque (solid arrow). Figure 3. shows the structural effects of immobilization and disuse; increased passive stiffness, decrease of optimal muscle length and reduction in the number of sarcomeres (solid arrows) [11]. Figure 4. shows the assumed passive stiffness lowering effect of negative stiffness compensation, enabling a larger active RoM with the same voluntary contraction.

An effective way to prevent immobilization and disuse would be to lower joint stiffness to enable the remaining voluntary muscle force to overcome joint stiffness and allow patients to increasingly control their ankle movement (Figure 1-1 lower right). There is evidence that promoting use and mobilization does not only prevent further aggravating but can even reverse the changes in connective tissue, force-length relation and plastic CNS changes [17, 18, 7]. There are treatments that reduce the passive stiffness, e.g. night splints and physical therapy. Night splints and physical therapy force the ankle towards the limit of its RoM and stretches muscles and connective tissue to reverse the muscle shortening and lower stiffness [19, 20]. Although these treatments have shown to reduce the stiffness and increase the RoM, they are labor intensive and used in passive and not in functional situations [21].

We believe that passive stiffness compensation, by applying a negative stiffness spring, can be an effective way of reducing the passive joint stiffness and thereby increase the RoM during functional tasks. Negative stiffness [22, 23] is the opposite of positive stiffness that is natural to any kind of material. For clarity, positive passive stiffness of the ankle joint means that resisting torque increases and energy is stored with imposed rotation. A negative stiffness spring obeys the reverse relation; torque increases in the same direction as the rotation and delivers mechanical energy.

When the increased passive stiffness found in UMND patients would be compensated by negative stiffness, it would in theory, result in a lower total joint stiffness enabling the remaining voluntary muscle forces to reach a larger RoM (Figure 1-1 lower right). This is the opposite of traditional dorsal AFOs where (positive) stiffness is added and the effects volun-

tary muscle contraction are even further reduced, increasing the functional paresis.

However, it remains unclear if patients are able to generate muscle force at the enlarged RoM, because of the decrease in optimal muscle length and how a more effective muscle activation affects the controllability of the ankle joint.

The main goal of this study is to answer the question: Does negative stiffness increase active RoM and how does this effect controllability of the ankle joint? Active RoM is assessed by a task where the subject is asked to achieve their maximum voluntary RoM. Controllability is assessed by a position following task, where jerk is a measure of how smooth the movement is performed and Root Mean Squared Error (RMSE) is a measure for how good the position is followed.

Chapter 2

Methods

To test the effects of stiffness compensation on the active RoM and controllability an experiment was conducted. In this experiment negative and positive stiffness was applied virtually, explained in section 2.2. Applying the stiffness virtually makes it possible to easily handle different compensation levels. This experiment served as a first test for negative stiffness, to test the theoretical possibilities and to develop a test protocol able to evaluate the effects of negative stiffness on active RoM and controllability.

2-1 Subjects

Ten healthy (mean age 33.2, SD 13.8 years, 4 male and 6 female) and two UMND Patients (age, 44 and 38 years, both female) participated in the study. Table 2-1 shows the subjects information. In the results the healthy subjects are taken as the main group and the UMND patients are shown as two separate samples.

2-2 Instrumentation

Achilles

Subjects were seated with their hip and knee at approximately 110° and 140° flexion respectively. Ankle rotations and torques were applied by an electrically powered single axis manipulator, "the Achilles" (MOOG FCS Inc., Nieuw Vennep, The Netherlands) (Figure 2-1). A feed-back screen was placed in front of the subject. For all subjects the left foot was used for measurement. The ankle was aligned with the manipulator's axis of rotation. A positive rotation of the manipulator was defined to equal dorsiflexion of the foot. A positive torque was defined to equal torques towards plantar flexion. In other words; movement in dorsiflexion direction is a positive rotation and the passive torques towards plantarflexion experienced during this movement are considered positive torques.

Table 2-1: Subject demographics. AS = Asworth Scale [24]

Subject ID	Age	Sex	Lesion	AS
Healthy 1	24	M	-	-
Healthy 2	58	M	-	-
Healthy 3	32	F	-	-
Healthy 4	27	M	-	-
Healthy 5	24	M	-	-
Healthy 6	56	F	-	-
Healthy 7	22	F	-	-
Healthy 8	25	F	-	-
Healthy 9	28	F	-	-
Healthy 10	28	F	-	-
Patient 1	44	F	SCI	2
Patient 2	38	F	MS	1

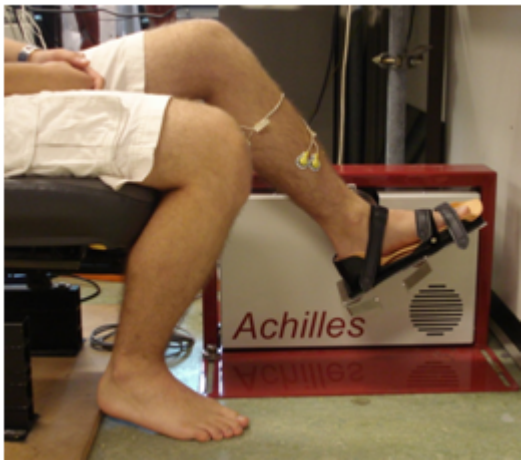


Figure 2-1: Measurement Set Up. The foot of the subject was fixated to the Achilles foot plate such that the rotation axis of the ankle was aligned with the axis of the manipulator. Joint angle, velocity, torque and EMG of plantar flexor and dorsiflexor muscle were measured.

Virtual Environment

In the experiments, additional positive and negative stiffness was applied virtually (programmed on the Achilles). The virtual stiffness was adjusted for each subject individually, based on the subject's passive torque-angle characteristic.

For every subject, first a passive stiffness identification and parameterization was performed with the Achilles, for protocol see section 2.3, and parameterized with three parameters (c1,c2,c3) describing the relation between the ankle angle θ and passive ankle reaction torque $T_{passive}$, see Eq. (2-1). The relation between ankle angle and joint torque is non-linear and torque increases exponential with ankle angle [10, 25, 26].

$$T_{passive} = c_1 \cdot e^{c_2(\theta - c_3)} \tag{2-1}$$

By taking the derivative of $T_{passive}$ with respect to θ the stiffness $k(\theta)$ is derived. The stiffness

k is a non-linear function of θ, see Eq. (2-2).

$$k(\theta) = \frac{\delta T_{passive}}{\delta \theta} = c_1 c_2 \cdot e^{c_2(\theta - c_3)} \tag{2-2}$$

The stiffness from Eq. (2-2) is used as a setting in the manipulator. The relation between the virtual spring torque, $T_{virtualspring}$ and θ is described in Eq. (2-3). In Eq. (2-3) α determines the level of compensation (negative α implies negative stiffness, positive α implies positive stiffness).

$$T_{virtualspring}(\theta) = \alpha \cdot k(\theta) \cdot (\theta - \theta_0) \tag{2-3}$$

The new torque angle characteristic around the ankle joint $T_{passivecompensated}$ becomes the sum of $T_{passive}$ and $T_{virtualspring}$, see Eq. (2-4). Figure 2-2 shows an example in case of compensation with negative stiffness (negative α).

$$T_{passivecompensated}(\theta) = T_{passive} + T_{virtualspring} \tag{2-4}$$

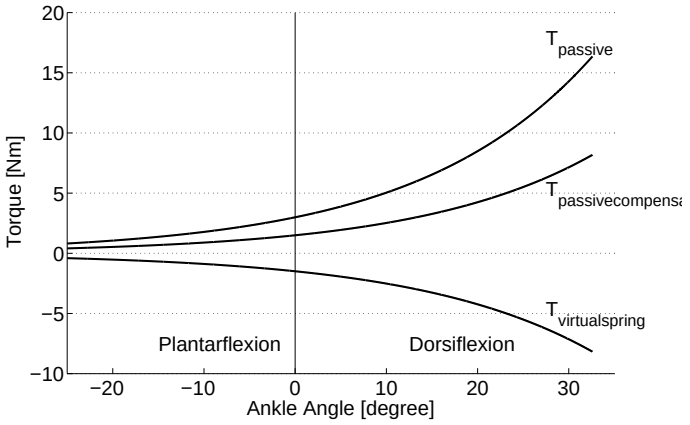


Figure 2-2: Stiffness compensation. Simulated passive stiffness compensation for α=-0.5. In the case of negative stiffness compensation the angle-torque relation, $T_{passive}$, is lowered by the torques created by the virtual spring, $T_{virtualspring}$ resulting in an altered angle-torque relation $T_{passivecompensated}$.

EMG

Muscle activation of the Tibialis anterior, Gastrocnemius lateralis and medialis and Soleus muscle was measured by differential surface electromyography (EMG) using Porti system (TMS International B.V., Oldenzaal, The Netherlands). Electrodes were placed following guidelines developed by SENIAM [27]. Inter electrode distance was 20 mm. The EMG signals were sampled at 2000 Hz, online filtering was done with a low pass filter, with a cutoff frequency of 540 Hz. The signals were filtered offline by a high pass filter (3th order butterworth) at 20 Hz, rectified and low pass filtered (3th other butterworth) at 5 Hz to obtain a linear envelope. The filtered EMG data from the Gastrocnemius lateralis and medialis and Soleus muscle were summed to obtain the muscle activity of the Triceps Surae.

2-3 Protocol

The experimental protocol was made up of two sections; an identification part and a measurement part. The identification part was needed to assess the subject's passive characteristics that are used to set the virtual springs settings for the measurements. In the measurement part, the effects of five different virtual spring conditions on active RoM and controllability were tested.

Identification

At the start of the experiment, the subject's passive characteristics were assessed with the Achilles manipulator. The torque-angle relation was needed to determine the negative stiffness spring settings; c1, c2 and c3 from Eq. (2-2).

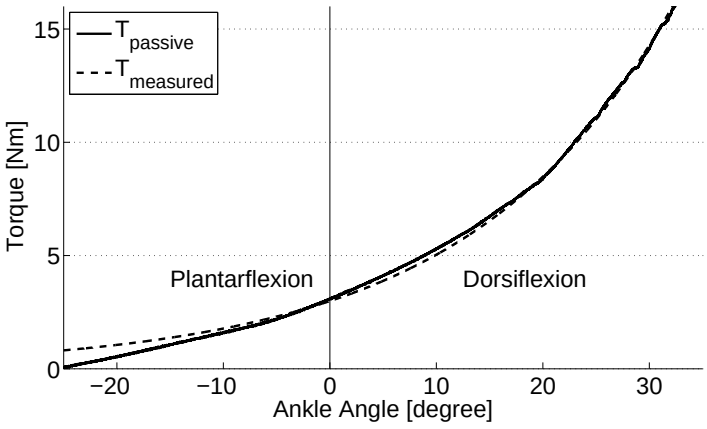


Figure 2-3: Identification. Example of measured ($T_{measured}$) (dotted) and modeled ($T_{passive}$) (solid) passive relation between ankle angle and torque. The modeled torque was obtained by minimization of least square error between the measured and the modeled torque. All relations are modeled from zero torque to full dorsiflexion.

First the passive RoM was determined by a slowly increasing torque ranging from -10Nm (plantarflexion) to 15Nm (dorsiflexion) in 80 seconds. The torque was applied and rotation was measured with the Achilles manipulator.

With the passive RoM known the characteristics are obtained by dictating the position with the manipulator and measuring reaction torques. When the position is dictated the velocity is fixed and undesired accelerations are prevented. The ankle was moved passively with the Achilles manipulator, from full plantar flexion to full dorsiflexion of the passive RoM with a fixed velocity of 3.75 deg/s, below the reflex treshold found by Lorentzen et al. [28].

EMG control was applied to check if the passive RoM was not affected by stretch induced muscle activity. The EMG levels were checked immediately after the trial.

The obtained torque-angle characteristic was the result of the passive tissue stiffness to be compensated. The torque-angle characteristic was parameterized using the model described in Eq. (2-1) of which parameters (c1, c2, c3) were quantified by minimization of the least square error between the measured ($T_{measured}$) and modeled passive torque ($T_{passive}$), see Eq. (2-5) and Figure 2-3.

$$e = \sum (T_{measured} - T_{passive})^2 \tag{2-5}$$

Conditions

Different levels of stiffness compensation were applied by the virtual rotational stiffness spring on the Achilles manipulator, described in section 2.2. The subjects performed the active RoM measurement with five different levels of compensation(-25%, -50%, 0%, +25%, +50%).

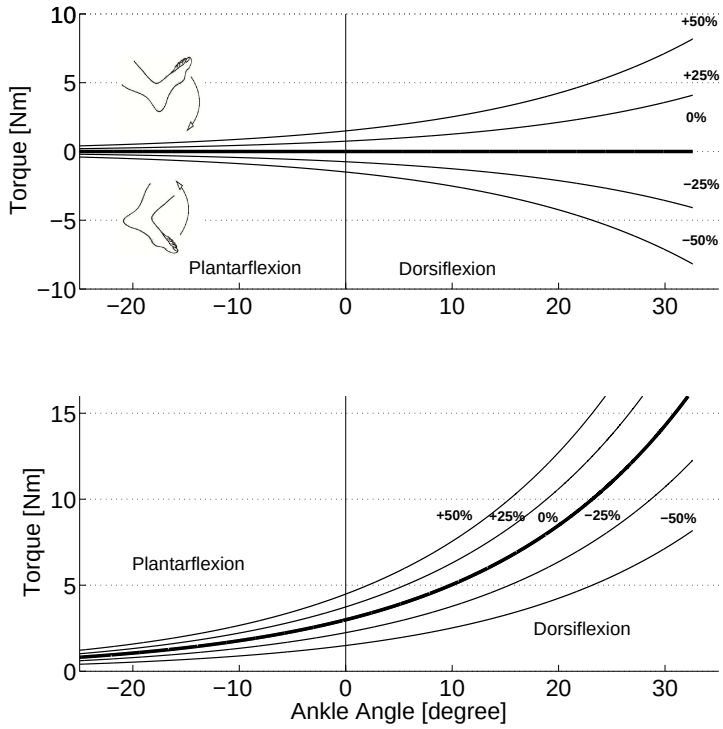


Figure 2-4: 5 Conditions. Typical example of applied virtual stiffnesses, $T_{virtualspring}$, for the 5 conditions **(Top)**. Positive stiffness implies positive torques (directed towards plantarflexion) and negative stiffness result in negative torques (directed towards dorsiflexion). Theoretic total torque $T_{passivecompensated}$ (Eq. (2-4)) for the 5 conditions are shown **(Bottom)**. In condition 0% no virtual torques are applied, see top figure, thus $T_{passivecompensated}$ in the 0% condition equals the measured passive stiffness.

Where no compensation (0%) is the subjects normal situation, negative (-25%, -50%) stands for compensation by a negative stiffness spring and positive (+25%, +50%) is restriction by a positive spring (Figure 2-4). For the healthy subjects the positive stiffness conditions simulates an increased passive stiffness component as found in UMND patients and treatment with stiffness increasing dorsal AFOs. Because simulation of increased stiffness was not needed and due to increased fatigability in UMND patients [6], the patients only performed three conditions (-25%, -50%, 0%).

Measurement 1: Active RoM

After the stiffness identification, the effect of passive stiffness compensation on the active RoM (especially dorsiflexion) was assessed. The five different levels of compensation (-25%, -50%, 0%, +25%, +50%) were applied randomly and the subject was asked to perform full dorsiflexion and plantar flexion repeatedly in 30 seconds trials. During the experiment it was checked if every trial consisted of at least three full cycles through the RoM, otherwise the trial was redone. Visual feedback of the ankle position and maximum plantar- and dorsiflexion achieved was given on a screen to motivate the patient to achieve a larger RoM (Figure 2-5).

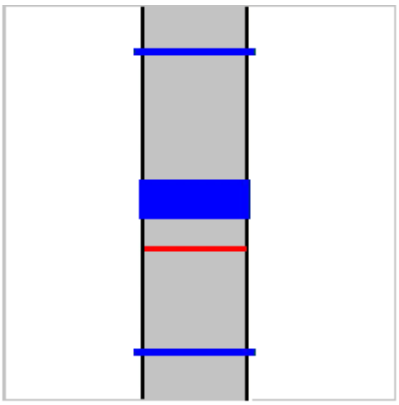


Figure 2-5: Active RoM screen. Feedback screen during Active RoM. The blue square in the centre represents the ankle position where the foot is perpendicular to the lower leg (0°).The thin blue lines are the maxima of dorsi- and plantarflexion achieved during that trial, these lines move when a larger angle is achieved. The red line shows the current position.

Measurement 2: Controllability

The aim of the last trials was to determine the controllability over the ankle movement for the five conditions (-50%, -25%, 0%, +25%, +50%). Controllability of the ankle joint is an important measure in a functional task e.g. walking. Since it was not possible to walk with negative stiffness in this protocol, ankle controllability was tested by performing a position following task illustrated by following a road, during 40 seconds per trial. On the screen, a sine-shaped road with an increasing amplitude and a red dot were displayed (Figure 2-6). The red dot represents the subject's ankle position on the Achilles. The subject can control the vertical position of the red dot on the screen with the Achilles foot plate, comparable to the throttle of a car. For every condition, the road was normalized to the largest dorsiflexion angle achieved in the active RoM trial. In other words, the subject should be able to reach the largest amplitude of the road in every condition, such that the controllability of the ankle in the active RoM was assessed. Before the measurement trial, the subject performed one practice trial to get familiar with the task after that the different conditions were applied in random order.

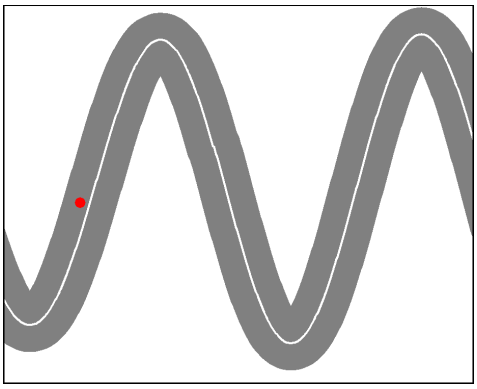


Figure 2-6: Controllability screen. Feedback screen during Controllability task. The screens displays the road, the red dot represents the current ankle position. The task during the trial was to keep to red dot on the road as good as possible.

2-4 Data Analysis

In this section the data analysis of the passive stiffness, active RoM and controllability task are elaborated.

Passive stiffness

For the passive angle-torque relation one property, K_{10} the passive stiffness at 10° dorsiflexion, was taken. Passive stiffness K was assessed as described in Eq. (2-6). Where K is a quasistatic stiffness [29] and ΔT is the passive torque increment during a ankle rotation ($\Delta\ \theta$). Quasistatic stiffness was calculated at 10° dorsiflexion by taking the slope of the regression curve to fit 6 data points around 10° [29].

$$K_{10} = \frac{\Delta T}{\Delta \theta} \tag{2-6}$$

Active RoM

The active RoM data was divided into movement cycles through the RoM, from plantar flexion to dorsiflexion. For analysis, the cycles were roughly indicated manually and thereafter the minima (plantar flexion) and maxima (dorsiflexion) were found for every cycle automatically. After defining the cycles, the maximum dorsiflexion, maximum plantar flexion and the total RoM per cycle were derived. Per trial the mean and standard deviation of these three parameters were taken.

To make sure changes in RoM were not a result of altered muscle activation between conditions, EMG data was observed. Next to EMG data torque generated by the subject is also analyzed.

The torque generated by the subject $T_{subject}$ contains both actively and passively generated torque, the active torques are generated by muscles and passive torques by passive tissues. $T_{subject}$ was obtained by subtracting the torque from the virtual environment ($T_{virtual}$) from

the total torque (T_{total}). T_{total} was obtained by deriving the accelerations from the measured velocity data and multiplying them with the total inertia, see Eq. (2-7)

$$T_{total} = (I_{virtual} + I_{plate} + I_{foot}) \cdot \ddot{\theta} \tag{2-7}$$

$$T_{total} = T_{subject} + T_{virtual} \tag{2-8}$$

In which $I_{virtual}$ is the virtual inertia (0.1 kg.m^2), I_{plate} the inertia of the plate (0.0036 kg.m^2) and I_{foot} the inertia of the subject's foot around the ankle joint. I_{foot} depends on the mass and distance of the centre of mass to the point of rotation of the subject's foot, see Eq. (2-9).

$$I_{foot} = m_{foot} \cdot l_{foot}^2 \tag{2-9}$$

$$m_{foot} = 0.014 \cdot m_{total} \tag{2-10}$$

$$l_{foot} = 0.0652 \cdot l_{total} \tag{2-11}$$

Both m_{foot} and l_{foot} were calculated by applying standard anthropometric measures [30] to total body mass and length, see Eq. (2-10) and Eq. (2-11).

To be able to mediate EMG and torque data over the cycles, time normalization was applied [31]. Time normalization arranges each dorsiflexion movement cycle from 0% (full plantar flexion) to 100% (full dorsiflexion) with a fixed sample size, making it possible to compare the cycles.

For every subject total active RoM, dorsiflexion RoM and plantar flexion Rom of their own normal active (0%, no compensation) trial were taken as the 100% level. The results of the trial with other conditions were taken relative and compared to this 100% level.

Controllability task

Controllability of the ankle was rated by two measures; Root Mean Squared Error (RMSE) and jerk. RMSE is a measure to quantify the difference between the desired position p_{road} and the actual position of the subject $p_{subject}$, see Eq. (2-12). The smaller the RMSE the better the road is followed by the subject.

$$RMSE = \sqrt{\frac{\sum (p_{road} - p_{subject})^2}{n}} \tag{2-12}$$

Jerk (rad/s3) is the rate of change of acceleration, obtained by deriving the acceleration with respect to time. Jerk is used as a measure of movement smoothness, the lower the jerk the smoother the movement. The jerk of every trial is rectified and then the mean per trial was taken as the measure for each trial.

2-5 Statistical analysis

Difference across the five different conditions was tested using a repeated measures ANOVA (with post-hoc Bonferonni testing) for the healthy subjects. As the two patients were considered as two seperated samples they are not included in the statiscal analysis. Level of stiffness compensation was taken as the fixed within-subject factor to assess outcomes variables i.e. total RoM, dorsiflexion RoM, plantarflexion RoM, jerk and RMSE. All statistical testing was performed using SPSS 20.0, SPSS Inc. at an α of 0.05.

Chapter 3

Results

All the subjects were able to understand and perform the tasks in the identification and measurement trials. In the patient measurements some difficulties were caused by fatigability. In the active RoM trials this resulted in less cycles performed per trial and in the controllability trials it proved difficult to follow the road the full 40 seconds of the trial. It was still possible to obtain data for all the patient trials.

3-1 Passive measurement

Table 3-1 shows the identified characteristics of the passive RoM and passive stiffness for the healthy subjects and the two patients. Figure 3-1 shows all the obtained passive angle-torque relations.

Table 3-1: Passive Measurement. Passive RoM, dorsiflexion, plantarflexion and stiffness K_{10} for both healthy and patients, divided by sex

Male	**Healthy**	**-**	**-**
Passive RoM (deg)	$69.53° \pm 5.1$	-	-
Passive DF (deg)	$26.12° \pm 6.1$	-	-
Passive PF (deg)	$43.42° \pm 2.9$	-	-
K_{10} (Nm/deg)	0.39 ± 0.13	-	-
Female	**Healthy**	**Pat. 1**	**Pat. 2**
Passive RoM (deg)	$89.29° \pm 10.1$	$87.40°$	$98.69°$
Passive DF (deg)	$25.76° \pm 7.2$	$28.06°$	$30.50°$
Passive PF (deg)	$62.20° \pm 14.1$	$59.34°$	$68.18°$
K_{10} (Nm/deg)	0.37 ± 0.17	0.31	0.29

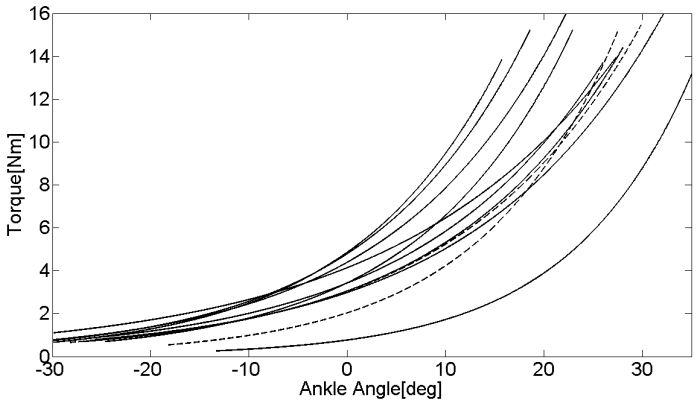


Figure 3-1: Passive angle-torque relation. All passive angle-torque relations in dorsiflexion direction. For 10 healthy (solid) and 2 patients (dotted). The plotted lines are the modeled $T_{subject}$ angle-torque relation, based on the $T_{measured}$ as explained in Eq. (2-5) and Figure 2-3. All relations are plotted from Torque=0 Nm till maximum dorsiflexion.

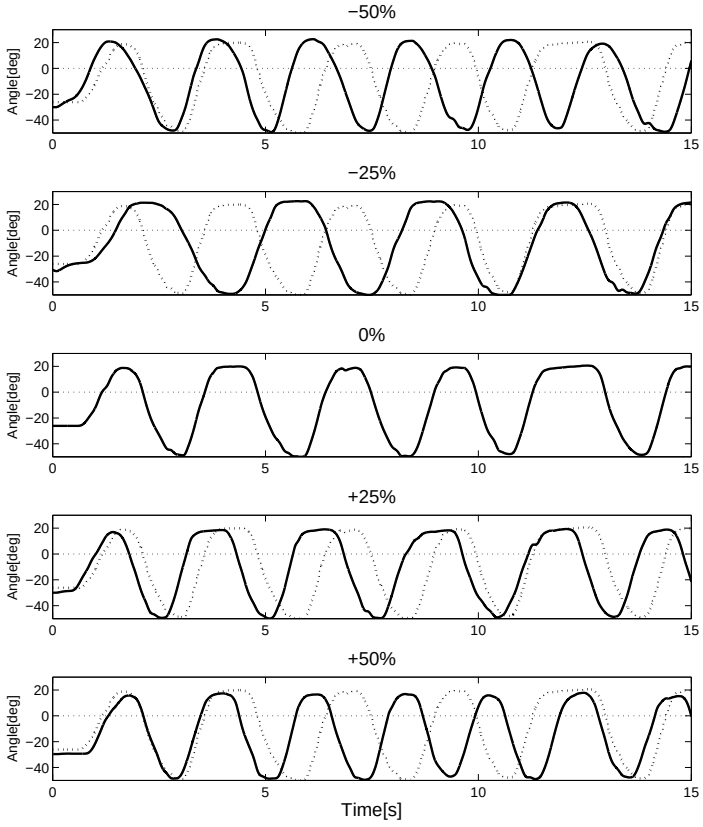


Figure 3-2: Raw active RoM. Typical raw active RoM rotations for first 15 seconds of the five conditions for subject 3. In every graph the results of the 0% condition is shown (dotted) for comparison. During the active RoM trials the subjects moved their ankle voluntary and tried to achieve maximum dorsi- and plantar flexion.

3-2 Active RoM measurement

Figure 3-3 shows the averaged total active RoM (75.71±10.9), dorsiflexion RoM (22.31±6.3) and plantarflexion RoM (53.40±12.9) for the normal (0% compensation) active trial across all healthy subjects and the two patients.

Figure 3-4 shows mean (±SD) of the normalized dorsiflexion and active RoM results for the five conditions, for healthy subjects and separate the two patients. The patients only performed three conditions (0%, -25% and -50%).

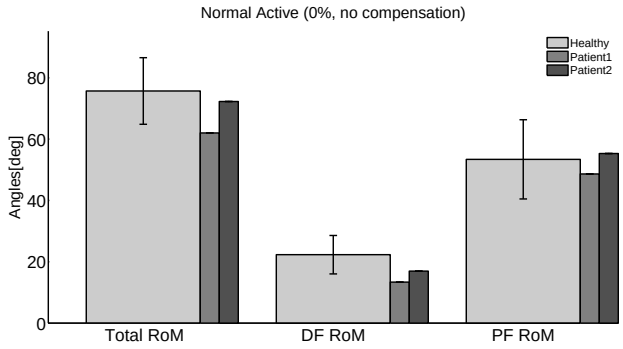


Figure 3-3: Normal (0% compensation) results. Active RoM, dorsiflexion (DF) and plantar flexion (PF) and standard deviation, for both healthy (main group) and patients (two samples). Active RoM, DF and PF were obtained by taking the mean of the dorsiflexion and plantarflexion peaks for the active 0% trial. For an example of raw data see Figure 3-2.

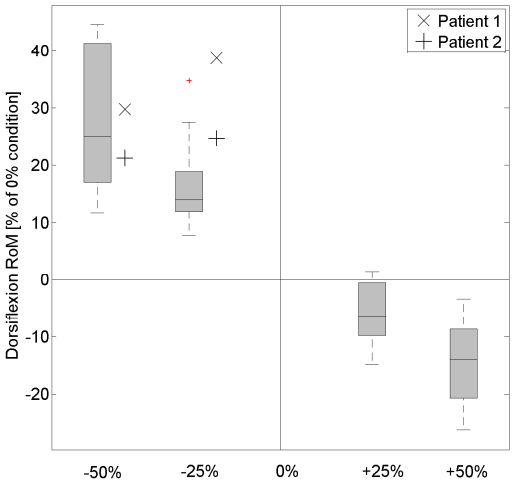


Figure 3-4: Dorsiflexion results. Dorsiflexion RoM (mean±1 SD) for the five different conditions. Scaled in percentage to normal (0%) dorsiflexion RoM. Positive percentages on the Y axis indicate an increase in RoM compared to the 0% RoM. Again the results of patients are displayed as two seperate samples.

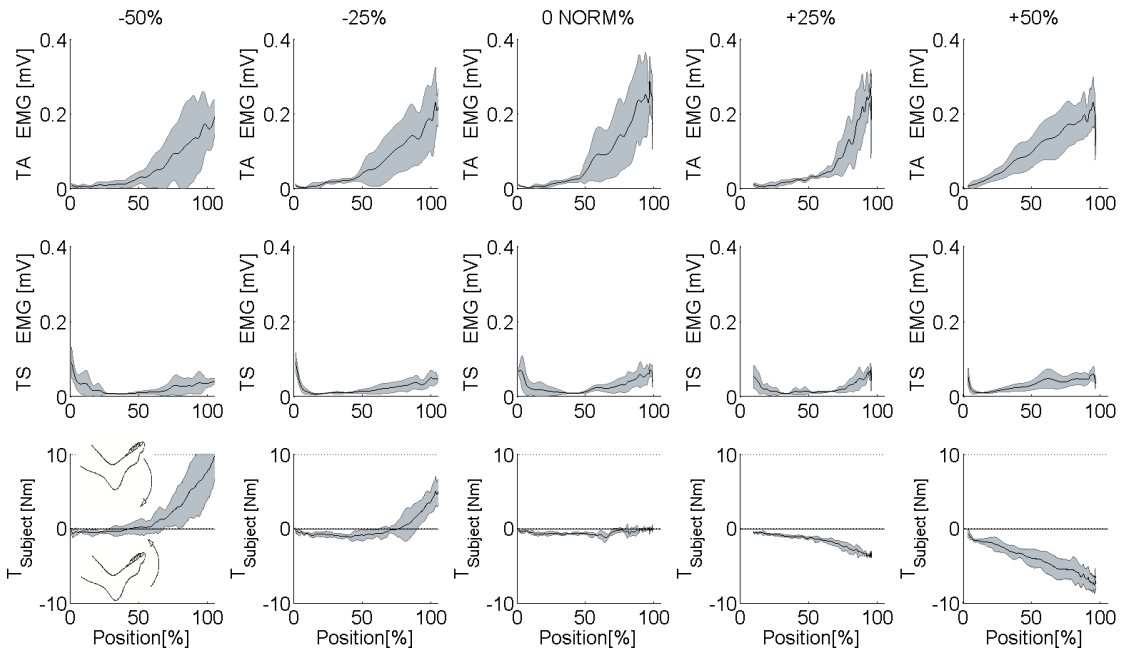
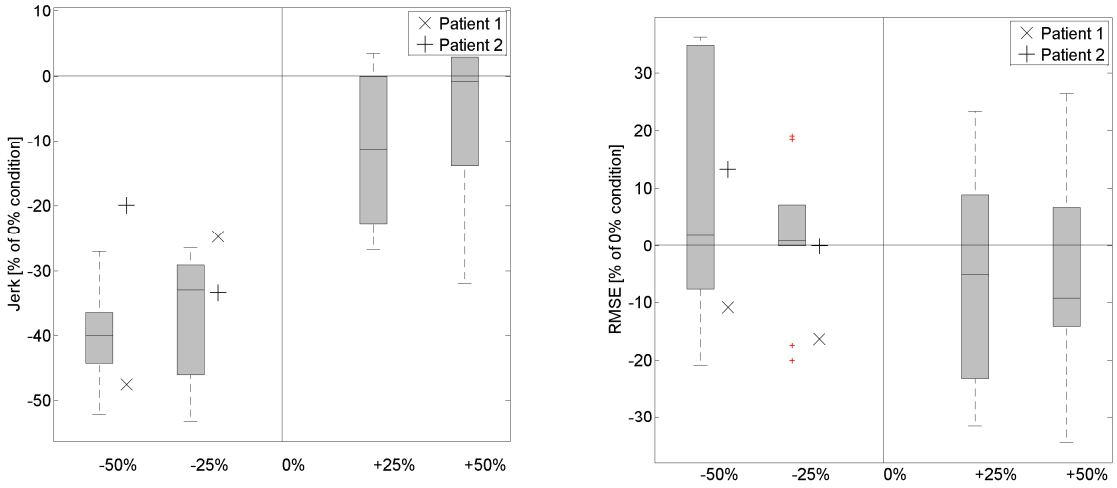


Figure 3-5: EMG and Torque. Typical example of EMG and $T_{subject}$ for all 5 conditions during active RoM task. Tibialis Anterior (TA) activity (Top,blue). Triceps Surae (TS) activity (Middle,green) and $T_{subject}$ (Bottom,red). $T_{subject}$ is the sum of actively and passively generated torque. Position ranges from full plantarflexion (0%) and full dorsiflexion (100%).

Maximum dorsiflexion (F=56.58, p<0.001) and active RoM(F=23.38, p<0.001) differed between the five conditions. Maximum plantar flexion was not significantly different between the conditions (F=1,33, p>0.27). For all subjects (healthy and the two patients) the maximum active RoM and dorsiflexion RoM increased when negative stiffness was applied and decreased with positive stiffness. Across the healthy subjects the largest active dorsiflexion(+27.81% of normal active dorsiflexion, p=0.001) and RoM (+8.46%, p=0.02) increase was achieved in the 50% negative stiffness compensation condition. The largest decrease in dorsiflexion(-14.87%, p=0.003) and active RoM (-2.99%, p=0.006) was observed in the 50% positive stiffness condition (Figure 3-4).

Figure 3-5 shows a typical example of the time normalized EMG and torque data during dorsiflexion movement for five active conditions. There are no major differences in the muscle activation between the conditions for both the Tibialis Anterior and the Triceps Surae. The torque delivered by the subjects ($T_{subject}$) does not differ between the conditions up to around 60% of the cycle. After 60% the delivered torque are clearly different. For the negative stiffness conditions, the torques increases strongly in the direction against the rotation. In the positive stiffness conditions the torque also changes after 60%, only now in the direction of the rotation.



(a) Jerk. Jerk results of controllability task for the five conditions, normalized to (0%) trial. The jerk is used as a measure of the smoothness of the movement, the lower the jerk the smoother the movement.

(b) RMSE. RMSE (Root Mean Squared Error) results of controllability task. RMSE is used as a measure of precision of the following movement.

Figure 3-6: Controllability results

3-3 Controllability task

All healthy subjects were able to reach the amplitude of the road completely for all conditions. For patients however, fatigability was a limiting factor. The jerk and RMSE scores were normalized; the scores in the normal (no compensation) trial were taken as 100% and the other conditions were normalized to this trial.

The amount of jerk differed between the conditions ($F=49{,}55, p<0.001$) (Figure 3-6a). Jerk is significantly smaller in both conditions with negative stiffness than in the normal 0% condition (for both $p<0.001$). In the 25% negative stiffness condition jerk was $63.18\pm9.9\%$ of the normal (0%), for 50% negative stiffness this was $59.51\pm6.8\%$. There is no significant difference between the two negative stiffness conditions ($t=1.58$, $p=0.146$).

There is no significant difference between the 5 conditions in RMSE score ($F=1{,}151$, $p>0.31$) (Figure 3-6b).

Chapter 4

Discussion

The overall aim of this study was to test the effects of different passive stiffness conditions on active RoM and controllability of the ankle joint. Passive stiffness conditions are achieved by applying different positive and negative stiffness springs. The springs were applied virtually on an electrically powered joint manipulator.

Effects of negative stiffness compensation on active RoM

In this study we found that passive stiffness compensation, by negative stiffness, significantly increases the total active RoM by enlarging the dorsiflexion RoM (Figure 3-4). The increase in dorsiflexion and not in plantarflexion was expected because negative stiffness is programmed to compensate stiffness in dorsiflexion direction. The largest effect was seen with the largest compensation level. EMG activity, of the muscles, seems not influenced by stiffness condition (Figure 3-5 top and middle). Assuming muscle activity is directly coupled to active torque, this indicates that the active generated torque by the muscles is also not different in the five conditions. From this it seems that the in Figure 1-1 presumed effect of negative stiffness occurs; with the same voluntary active torque a larger (dorsiflexion) RoM was achieved when negative stiffness is applied to reduce the passive stiffness.

Effects of positive stiffness on active RoM

Positive stiffness reduces total active RoM, by reducing the dorsiflexion RoM, of subjects, however the effect is not as large as the effect of negative stiffness on dorsiflexion RoM (Figure 3-4). The active RoM in every condition is determined by the intersection between the active and passive angle-torque relation as shown in Figure 1-1. The passive relation differs for the five conditions (Figure 2-4). Because of the nonlinearity, the same torque level results in different increase in RoM between the five conditions. Figure 3-5 indicates that the active angle-torque relation is not different for the conditions, however the slope of the

active anlge-torque relation does influence the active RoM as it determines the intersections between the active and passive curves.

Influence of stiffness conditions on torque interplay around ankle joint

$T_{subject}$ is the sum of active and passive torques generated around the subjects ankle, however it is not possible from $T_{subject}$ to distinguish the actively (muscles) and passively (passive tissue) generated torque. Figure 3-5 gives an indication on how the torque interplay around the ankle is influenced by the different stiffness conditions. $T_{subject}$ (Figure 3-5 bottom) is clearly influenced by the different stiffness conditions, especially near full dorsiflexion. At full dorsiflexion, the ankle cannot rotate further and torques around the ankle joint are in equilibrium. In other words, T_{total} from Eq. (2-8) equals zero and $T_{subject}$ and $T_{virtual}$ are equal and opposite in direction. This explains the change in $T_{subject}$ in the different conditions, as $T_{virtual}$ is changed in every condition.

Because muscle activity seems not influenced by the stiffness condition (Figure 3-5 top and middle), the changes in $T_{subject}$ are likely to be caused by passive torques generated by passive tissues. The increased passive torque in negative stiffness conditions could have a positive long term effect on the viscoelastic properties of the ankle. Comparable to night splints and physical therapy the increased passive torque stretches muscles and connective tissue to reverse the muscle shortening and lower stiffness. However in the case of negative stiffness in a more active way, including afferent and efferent neural control.

Effects of different stiffness conditions on controllability of the ankle joint

Controllability with different stiffness compensation conditions was tested by a road following task. Jerk was used as a measure of smoothness of the movement. RMSE is a measure of how precise the road was followed.

Negative stiffness results in lower jerk (Figure 3-6a) for both patients and healthy subjects. The low jerk with negative stiffness indicates that negative stiffness smoothens movement during the road following task. Although potentially, negative stiffness could lead to instabillity as the total stiffness of the joint is lowered. However, the lower jerk indicate this is not the case during the position following task.

The RMSE scores do not give a clear consensus on the effect of different stiffness conditions in the road following task precision (Figure 3-6a). Learning effects could have influenced the RMSE scores for both healthy subjects and patients. In the case of patients fatigability also influenced the performance during the controllability task.

Another factor influencing both jerk and RMSE are the strategies subjects use to follow the road. Some subject tend to “catapult” their foot between the peaks of the sine. This strategy theoretically leads to low jerk, especially in the negative stiffness conditions, where the external torque is in the same direction as the dorsiflexion movement. When catapulting between peaks the focus is not on precisely following the road at the slopes between the peaks, having a negative effect on the RMSE.

Patient Measurements

Because the patient group in this study is very small (n=2) it is not possible to interpret their results in the same way as the results of a patient group. However some remarking results and findings are discussed.

Patient passive RoM and Stiffness

The two participating patients were clinically labeled as patients with an increased joint resistance caused by increased passive stiffness, rated with Ashworth scale 1 and 2, see Table 2-1. However their passive RoM was not smaller than the passive RoM of the healthy subjects, see Table 3-1, contrary to the expectations from their Ashworth scores. De Vlugt et al. reported a relation between the Asworth scores and passive RoM [10], the higher the Asworth score, the smaller the passive RoM, this is not the case in the current study.

There is also no clear increase in the passive stiffness at 10 degrees dorsiflexion, see Table 3-1 and Figure 3-1. Chung et al. found a clear increase in the passive stiffness of stroke patients compared to healthy controls [29]. The comparable passive characteristics between patients and healthy subject in the current study indicate that increased joint resistance seems not caused by increased passive stiffness for these two patients. Another component that can increase the joint resistance is hyperactivity of the stretch reflex [32, 33, 34]. Because reflexes are velocity dependent and in the passive RoM experiments velocity was low, reflexes were not apparent in our passive RoM experiment.

There are studies indicating a difficulty to discriminate between passive stiffness and hyperactive reflexes in a clinical setting [35, 28]. Studies using quantitative methods claim to be able to discriminate between the components involved in passive joint resistance. However in these studies there is no consensus about the dominance of one of the resistance components. Some studies report increased passive stiffness in UMND patients [10, 29, 25] while another study found the hyperactive reflexes to be the cause of the resistance [36]. The difficulties in distinghuishing between joint resistance components could explain the difference in the patient's clinical label and the experimental outcome in the current study.

Despite their "normal" passive stiffness, compensation of the patient passive stiffness, by negative stiffness, still resulted in a larger active RoM. Because of the paresis the maximal voluntary activation of patients is reduced and because of the decrease in optimal muscle length the forces at higher angles are lower. Passive stiffness compensation enlarges the functional length of the remaining muscle activation (Figure 1-1).

Patient active RoM

The total normal (no compensation, 0% condition) active RoM of the two patients is comparable to the normal active RoM of the healthy subjects. However there is a small shift towards plantar flexion (Figure 3-3), confirming the patients trouble to reach normal dorsiflexion. This shift towards plantar flexion in active RoM is also observed in other studies and is a result of the paresis and shift in active force-length relation. This leads to asymmetric contraction around the joint, i.e. plantarflexors contract proportionately more than dorsiflexors[8, 37].

Clinical implications

The finding that passive stiffness compensation, by negative stiffness, increases the active RoM and possibly smoothens the movement might implicate that applying negative stiffness could lead to improved ability of UMND patients to perform functional tasks, e.g. walking. Although in this study no walking tests were performed, the outcomes can have an interesting contribution in the discussion about walking in UMND patients. There are studies stating that the increased stiffness in the ankle joint of UMND patients is a positive adaption mechanism to the paresis allowing functional movements on a simpler level of organization [38, 39]. However, the current findings show that negative stiffness enables the same muscular activation to achieve a larger active RoM. This might indicates that lowering the stiffness will enable functional movement on a higher level of organization, because muscles and higher neural tracks are involved. The ability to perform functional tasks, initialized by the remaining muscle force may promote mobilization and use of the ankle joint and neuromuscular system.

In the long term the effects of negative stiffness compensation can have a broader function than only improving performance of functional tasks. The effects of a larger active RoM on the neuromuscular system can clarify more about the pathophysiology of UMND patients. It could give more insight in the roles that passive stiffness and hyperactive stretch reflexes play in joint resistance of UMND patients. A larger RoM enables more use of the ankle joint, entailing that muscle spindles will have more input. Hyperactive response to muscle spindle input could result in undesired reflexes, having a negative effect. However, increased input on muscle spindles could also have a positive effect through facilitating reversing of plastic CNS rearrangements, a positive effect. From the lower jerk in negative stiffness conditions reported in this study, it seems that a larger RoM does not necessarily evoke undesired stretch reflexes. Undesired stretch reflex would lead to a less smooth “spastic” movement pattern.

Future Research

This study served as a first test with negative stiffness compensation. The positive results of negative stiffness on active RoM raises interest for future research. Two factors that should be the focus in future research are; walking experiments and the use of a larger group of patients. Experiments on walking are important because the results from the current study do not shed light on the effects of stiffness compensation on important factors in walking, such as stability.

There are multiple factors involved in the present symptoms of UMND diseases, e.g. site, origin and severity of the lesion and also duration since the lesion. The two participating patients both did not have an increased passive stiffness. However when a larger group of patients would be measured, it is likely that a wider variety of symptoms would be observed. Therefore it is necessary to test future experiments on a larger group of patients.

Chapter 5

Conclusion

This study shows that negative stiffness enables a larger active dorsiflexion RoM than in the situation without compensation, using the same muscular activation. Compensation of passive stiffness gives voluntary muscle contraction a larger functional length. For UMND patients this means that despite a paresis, self initialized movement of the ankle becomes possible. By enabling muscle force to initialize movement, immobilization and disuse can be prevented. Mobilizing and using the ankle joint has a possible positive effect on viscoelastic properties of connective tissue and on reversing plastic rearrangements in the CNS. The possible positive effects of a larger active RoM may be the foundation of rehabiliation therapy of UMND patients focusing on enlarging the RoM during active functional tasks.

In this study negative stiffness is not unambiguously related to a positive effect on ankle joint controllability. Negative stiffness has a positive effect on the smoothness of movement, indicating positive effects during functional tasks.

We can conclude that negative stiffness has a positive effect on active RoM, especially in dorsiflexion. The positive but premature results of the present study encourage more research on controllability and stability during walking.

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Appendix A

Reflection and recommendations

The study performed served as a first experiment with negative stiffness, to test the possibilities and to develop a test protocol to evaluate the effects of negative stiffness on active RoM and controllability. This section reflects on the experimental protocol and gives recommendations for follow-up research.

The first goal for the study was to test the effects of negative stiffness compensation on a group of patients. As regulational limitations restrained this, the focus of the experiment became to measure the on healthy people. However, two patients measurements were done. While doing the measurements on the patients it became clear that due to the limitations of these patients the "healthy" protocol was a big challenge for them.
For the patients a short protocol with short trials is needed, as fatigability was the biggest limitation during the experiments.

Looking back to the controllability task, it seems that the current road following task might not be the best method to evaluate the controllability. As already mentioned in the discussion of the scientific paper the subject strategy and learning effects seems to influence the outcomes more than the different stiffness conditions. For example tests using a single ramp to achieve a certain position with the task to limit the overshoot, might give better results.

I think the protocol functioned well as the effects of negative stiffness on active RoM are clearly shown. However there is much more to be tested in the similar protocols and finally in more functional settings. I believe follow up research should focus on testing on more patients and in parallel develop a prototype of an negative stiffness AFO, making experiments on walking with negative stiffness possible.

Appendix B

Forms

B-1 Subject information form

B-2 Protocol Checklist

B-1 Subject information Form:

Proefpersoongegevensformulier

Deze informatie is vertrouwelijk en wordt niet beschikbaar gemaakt voor derden.

Persoonsinformatie

Voor- en achternaam	:…….
Adres	:…….
Postcode en woonplaats	:…….
Telefoon	:…….
Geboortedatum	:…….
Geslacht	: m / v
Lengte	:…… cm
Gewicht	:…… kg
Voorkeursbeen	: rechts / links

Ik verklaar hierbij bovenstaande informatie naar eerlijkheid te hebben ingevuld.

Datum:

Handtekening:

B-2 Protocol CheckList:

PP save NAME:……/Been in Achilles:…….

Voorbereiden zonder proefpersoon

- **Zet stoel goed**
- Open c: AchillesGUI07_Leon\RobinGui.m
- Init de Achilles
- Bepaal volgorde van actieve RoM metingen → random
- Bepaal volgorde van wegvolg metingen → random
- Standaard volgorde voor zowel actief als wegvolgtaak **VOLGORDE**
  - *1)Act RoM* ……. …….
  - *2)Negk.25* ……. …….
  - *3)Negk.50* ……. …….
  - *4)Posk.25* ……. …….
  - *5) Posk.50* ……. …….
- ProtocolName=LeonPatientMeting
- SessionName= Patient_#
- Zet trials in geode volgorde:
  - 1) A_Leon_startPOS
  - 2) B_Leon_EMG
  - 3) Ca_leon_pasROM
  - 4) Cb_leon_pasPOS
  - 5 tm 9 in volgorde bepaald
  - *10) oefen file voor wegvolg taak*
  - 11 tm 15 in volgorde bepaald

Voorbereiden met proefpersoon

EMG plakken

- Vraag proefpersoon in dorsale richting aan te spannen
  - Bepaal positie Tibialis Anterior en markeer met pen
- Vraag proefpersoon in plantaire richting aan te spannen
  - Bepaal positie Soleus en markeer met pen
  - Bepaal positie Gastronemicus lateralis en medialis en markeer met pen
- Maak de gemarkeerde plekken schoon met scrub en alcohol
- Plak EMG plakkers tegen elkaar aan en maak kabeltjes vast

Proefpersoon in stoel positie bepalen

- Maak voet proefpersoon goed vast in voetenbakje **inclusief extra opzetstukje**
- Bepaal de kniehoek van de proefpersoon → 140° → schuif stoel tov Achilles

Begin protocol

90 graden stand

- Vraag de proefpersoon zijn enkel in 90° te houden → meet na met gradenboog en vraag deze positie vast te houden
- **Run Trial 1. A_Leon_startPOS**

EMG check

- **Instructie Proefpersoon:** Beweeg je voet rustig heen en weer van maximaal dorsaal naar maximaal plantair.
- **Run Trial 2. b_Leon_EMG**

- **Check figure** → Als EMG niet goed zit opnieuw plakken en EMG check opnieuw

Identificatie
- **Instructie Proefpersoon:** Het apparaat gaat u straks op en neer bewegen probeer zoveel mogelijk te ontspannen.
- **RUN Run Trial 3. Ca_Leon_PASROM**
- **Check figure for EMG activity**
- **Instructie Proefpersoon:** Het apparaat gaat u straks opnieuw op en neer bewegen probeer zoveel mogelijk te ontspannen. Het duurt even voordat de trial begint.
- **RUN Run Trial 4. Cb_Leon_PASPOS**
- **Check figure for EMG activity**
- **Check figure for FIT**
- **Check figure for Negative SPRING**
-

SetLimits
- **Patient voet los halen uit de Achilles**
- Run trial Cc_SetLimits
- Druk op spatie → verzet onder stop
- Druk op spatie → Verzet boven stop
- Druk op spatie
-

Actieve RoM:
- **Instructie Proefpersoon:** U gaat nu 5 keer zelf zo hoog en laag mogelijk proberen te komen. U ziet straks op het beeldscherm een rode balk in beeld wat u huidige positie voorstelt, en 2 blauwe balken wat u maximale bereikten posities zijn. Beweeg op en neer en probeer de blauwe balkjes constant te verleggen, lukt dit niet meer blijf dan nog steeds op en neer bewegen.
- **RUN Trial 5 t/m 9.**
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WegVolg Taak:
- **Instructie Proefpersoon:** U gaat nu 5 keer een weg volgen, we gaan het eerst een keer oefenen. Een rood blokje presenteert de positie van u voet en probeer zoveel mogelijk in het midden van de weg te blijven.
- **RUN 10 OEFEN FILE SinePosition**
- **RUN Trial 11 t/m 15.**