

## Measurement of impaired muscle function of the gastrocnemius, soleus, and tibialis anterior muscles in spastic hemiplegia: A preliminary study

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**Abstract**—Based on the results of several electrodiagnostic and biomechanical studies, the following classification of muscle dysfunction in spastic hemiplegia is proposed: changes in muscle activation (excess symptoms, e.g., spasticity, and deficit symptoms, e.g., paresis); changes in muscle stiffness; and changes in muscle length. The clinical significance of this classification is that different types of muscle dysfunction might require specific treatment.

The authors have developed techniques to measure quantitatively each type of muscle dysfunction: free frequency repetitive movement (FFRM) and torque angle diagram (TAD). Surface EMGs of tibialis anterior, gastrocnemius, and soleus muscle are recorded during active (FFRM) and passive (TAD) ankle movements. EMG data are converted to parameters for abnormal muscle activation (excess and deficit symptoms). Parameters for muscle stiffness and muscle length are derived from the hysteresis curve of the TAD.

This article describes the measurements and the results of a validation study. For the validation study, four hypotheses were formulated: 1) in nonimpaired control subjects, parameters expressing abnormal muscle activation are low; 2) in hemiplegic subjects, differences between the affected and the unaffected sides will be found for all types of parameters; 3) after local anaesthesia of the tibial nerve on the hemiplegic side, excess symptoms will decrease, while muscle stiffness remains unchanged; and 4) despite a uniform gait pattern, between-subject differences can be detected with regard to muscle activation, stiffness, and length.

The first hypothesis was tested and confirmed in two controls; the remaining three were tested and confirmed in ten hemiplegic subjects (mean age 47.7 yrs, mean time since onset 10.7 yrs). However, the level of co-contraction of the gastrocnemius muscle was low, probably indicating that the clinical significance of this phenomenon might be limited.

The results support the validity of the proposed classification and measurements.

**Key words:** hemiplegia, measurement, spasticity.

### INTRODUCTION

The most widely accepted definition of spasticity is: “a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex, as one component of the upper motor neuron syndrome” (1). Until recently, subjective measures such as the Ashworth scale, have been widely used (2–4). However, objective measurement techniques, necessary for the evaluation of various modes of treatment, are lacking.

For the objective measurement of changes in muscle function in spasticity, electrodiagnostic and biomechanical techniques have been developed.

A wide variety of electrodiagnostic measurements has been used to quantify the level of hyperexcitability of the stretch reflex, as one component of the upper motor neuron syndrome (UMNS). Electrodiagnostic studies have contributed to the knowledge about

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neuronal circuits in the spinal cord but, unfortunately, the results correlate poorly with clinical severity. Kinesiologic electromyographic studies, measuring changes in muscle function, have shown that the following factors contribute to the impairment of muscle function: increase in stretch reflex activity, inappropriate muscle activation (e.g., co-contraction), and severity of paresis (5–7).

The enhanced stretch reflex during imposed movements can be present either in a dynamic way (only during stretch) or in a tonic way, showing EMG activity as long as passive movement is imposed (8). This results in the clinical observation of stiffness during passive movement in a joint. In UMNS there is a typical distribution of spasticity in antigravity muscles.

During voluntary movements of the knee, co-contractions (simultaneous contractions of the agonist and antagonist of movement) were noticed in the hamstring muscles (9,10). However, co-contractions were not associated with the presence of increased stretch reflexes during passive movements. Co-contractions were also found during gait analysis in some hemiplegic subjects (11,12).

In spastic hemiplegia, loss of recruitment of agonist contraction, or reduced output paresis (ROP), plays a major role in the impairment of muscle function (13). Based on these findings, motor dysfunction in UMNS can be classified as positive (excess) and negative (deficit) symptoms (14). Positive symptoms are manifestations of enhanced stretch reflex activity and co-contractions, resulting in spasticity, spontaneous spasms, and clonus. Negative symptoms are deficits in motor performance, resulting in decreased dexterity, paresis, and enhanced fatigability.

Biomechanical investigations registered the torque, joint angle, and surface EMG recording of the muscles controlling the joint during either imposed linear or sinusoidal movements. These studies have shown that resistance to imposed movement is not only the result of changes in stretch reflex response, but also depends on the passive properties of the tissues, such as elastic and viscous stiffness and friction (15–17). These changes in biomechanical properties also result in increased muscle stiffness in passive movement during clinical examination. Another change in passive properties is shortening of the muscle-tendon complex, as has been demonstrated in patients with hemiplegia and in children with cerebral palsy (18–20).

Based on these findings, we propose the following classification of muscle dysfunction in UMNS:

1. changes in muscle activation: (a) positive or excess symptoms: enhanced stretch reflex activity, clonus, spontaneous spasm, and co-contraction; (b) negative or deficit symptoms: decreased dexterity, enhanced fatigability, and paresis;
2. changes in muscle stiffness;
3. changes in muscle length.

Measurement of these differences in muscle dysfunction could well be relevant for clinical practice: a shortened muscle can be treated by surgical lengthening, excess symptoms with a spasmolytic drug increasing presynaptic inhibition in the spinal cord, and increased muscle stiffness with dantrolene sodium (14).

### Objective of the Study

The aim of this study was twofold: to develop a method to quantify these three aspects of muscle dysfunction (changes in muscle activation, stiffness, and length), and to study the construct validity of the method. As many therapeutic interventions in persons with UMNS are aimed at improving walking capacity by improving ankle function, this study concentrated on muscles controlling the ankle joint.

We hypothesized that:

1. in nonimpaired control subjects, parameters expressing abnormal muscle activation are low;
2. in hemiplegic subjects, differences will be found in parameters of muscle activation (excess and deficit), muscle stiffness, and muscle length, when comparing the hemiplegic with the unaffected side;
3. after nerve-blocking on the hemiplegic side, parameters expressing excess muscle activation will decrease, while parameters expressing muscle stiffness remain unchanged;
4. notwithstanding a uniform spastic gait pattern, significant between-subject differences can be detected with regard to muscle activation, stiffness, and length.

As the method was intended for use in the evaluation of therapeutic interventions in clinical practice, the following conditions were determined: The measurements must closely resemble the normal physical examination of a patient by a physician in order to facilitate comparison of laboratory findings with physical examination data. The measurements must be applicable in a clinical environment, which sets limits to the complexity of instrumentation, the time required for

the test and the method of interpretation of the results. The measurements must be based on a biomechanical analysis of human movements in terms of torque, range of motion (ROM), and angular velocity.

## METHOD

Physical examination of muscle function in a person with spastic hemiplegia is based on observations of the quality of voluntary movement and the performance of passive motion in a joint. To objectify these clinical observations, we developed a technique to examine voluntary muscle function, the free frequency repetitive movement (FFRM) test, and a technique for measuring passive muscle function during imposed movements, which produced the torque angle diagram (TAD).

### Free Frequency Repetitive Movement

Voluntary muscle function was assessed by FFRM in the ankle in a sitting position. The subject was instructed to move the foot alternately toward plantarflexion and dorsiflexion with a maximum ROM and at a maximum speed for 30 s. Synergistic movements in the knee and hip joint were allowed. Ankle movements were recorded with a potentiometer-based goniometer. Muscle activity was recorded by surface electromyography (EMG) of the tibialis anterior muscle (TAM) and the medial head of the gastrocnemius muscle (GM). For the bipolar lead-off, the electrodes were placed in the direction of the muscle fibers, at the center of the palpable muscle belly. Rim-to-rim distance of the two electrodes was 23 mm, and the effective lead-off surface of the electrodes was 1 cm<sup>2</sup>.

The EMG was preamplified with a miniature amplifier (gain: 100×, CMRR>100 dB) mounted on the two electrodes. The differential signal was led away, high-pass filtered (at 20 Hz, 18 dB/oct) in order to remove residual movement artefacts, and amplified. This signal was fed to an oscilloscope to be checked. For recording purposes, the EMG was rectified and smoothed out (low-pass filtering at 25 Hz, 18 dB/oct) to obtain the smoothed rectified EMG (SR-EMG, equal to the integrated EMG). The SR-EMG of the TAM and the GM, together with the goniometer signal of the ankle were stored online on a microcomputer using A/D conversion (sample frequency: 120 Hz).

Off-line, the movement was divided into cycles to calculate the movement frequency. A cycle was defined

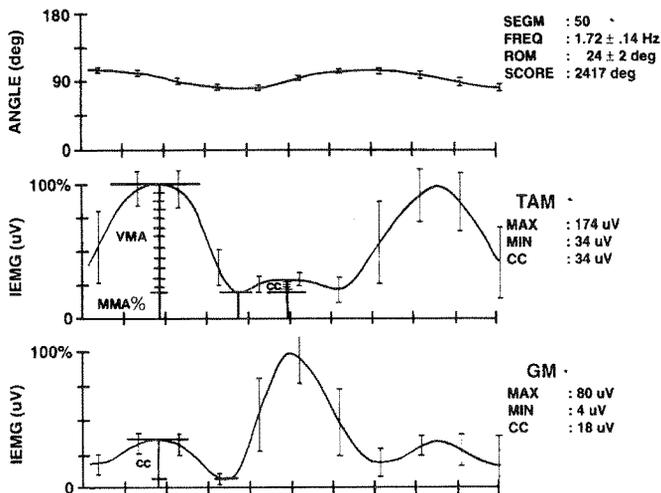
as the period between two dorsidirectional crossings of the signal with the median angle of the movement. The SR-EMG was filtered through a digital low-pass filter at a frequency of 4 times the movement frequency of the ankle (4th order Butterworth filter with no phase lag). Furthermore, as shown in **Figure 1**, the cycles were ensemble averaged for all signals in order to obtain the average movement and muscle activation patterns, together with the standard deviation, as a function of 0–100 percent of the cycle phase (21). In the plots, when the maximum value of the SR-EMG exceeds 40  $\mu$ V, it was normalized to a percentage of the recorded maximum SR-EMG value of each muscle.

### Torque Angle Diagrams

In order to assess the passive behavior of the ankle joint, a standardized movement was imposed. It was decided that this movement should be sinusoidal, as this is very similar to functional movements, such as gait, in contrast to isokinetics. Moreover, during constant velocity movements, very high decelerations and accelerations occur at the reversal of movement direction, which trigger the tendon stretch reflex in persons with UMNS.

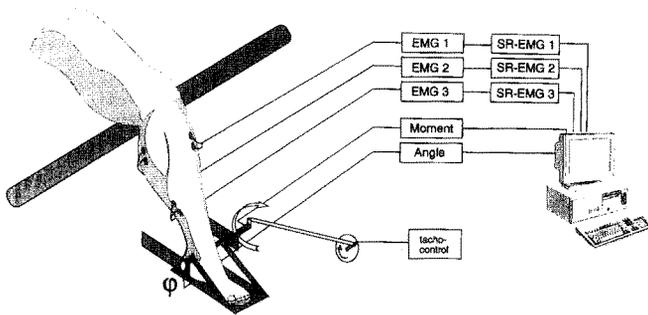
For this study, a special dynamometer capable of driving the ankle joint was constructed (**Figure 2**); its frequency and ROM could be programmed. The foot of the subject was fixed in an exchangeable shoe, firmly secured to the footplate of the dynamometer. The axis of rotation of the footplate could be adjusted, in order to obtain alignment with the closest approximation of the single axis of rotation of the ankle (22). The alignment was checked by minimizing the shift of the tibia during ankle movement through the full ROM. The footplate was rotated by an electrical motor with tachocontrol, via a crank-linkage mechanism. On one side, a torque transducer, based on strain gauges (23), was interposed between the crank and the footplate. On the other side of the axis, a potentiometer-based goniometer measured the angular position of the footplate. The SR-EMG of the TAM, the medial head of the GM and the soleus muscle (SM) were also recorded using the same instruments used for the FFRM. The signals of torque, angle, and SR-EMG were stored for 3–5 cycles (depending on the frequency of the movement).

Off-line, the signals were divided into two phases, dorsal and plantarflexion, each ensemble averaged. In this way, an X-Y plot of the angle versus the net torque could be generated, showing a typical curve of hyster-



**Figure 1.**

Report of a free frequency repetitive movement test of the ankle of a control subject: illustration of the parameters. TAM=tibialis anterior muscle; GM=gastrocnemius muscle; SEGM=number of segments of movement; FREQ=frequency; ROM=range of motion; Score=product score; CC=co-contraction; VMA=voluntary muscle activity; MMA=minimal muscle activity. X-axes: One and a half cycles of movement (dorsiflexion–plantarflexion–dorsiflexion). Y-axes: top, the ankle movement in degrees; mean values of 50 cycles are calculated, 1 SD plotted. Middle, the integrated (smoothed rectified) EMG of the TAM is represented. The mean maximal EMG activity of that muscle (174  $\mu$ V) is used for normalization to 100 percent. Bottom, the IEMG activity of the medial head of the GM is shown.



**Figure 2.**

The ankle dynamometer with surface EMG registration of the tibialis anterior, soleus, and gastrocnemius muscles.

esis (**Figure 3**). The SR-EMG of the muscles was also expressed as a function of the joint angle for each separate phase of the movement. The net torque was corrected for the gravitational influence of footplate and foot.

## Procedures

The subject was seated in a stable chair, with 90° of flexion in hip and knee. The FFRM in the ankle was performed, as described, after which TAD was measured with the ankle dynamometer. After fixation of the foot, the ankle axis was carefully aligned. The ROM was defined as the range limited by the net torque values of 8 Nm (dorsiflexion) and -5 Nm (plantarflexion). The frequency of the imposed sinusoidal movement was set at a maximum angular velocity of 30 °/s. The procedure was repeated after adjustment of the sitting position of the subject to 110° flexion of the hip and full extension of the knee.

The tests were performed three times at weekly intervals: in the first session, measurements were performed on the hemiplegic side, in the second, on the hemiplegic side after local anaesthesia of the tibial nerve (TN), and in the third on the unaffected (nonhemiplegic) side.

Local anaesthesia of the TN was achieved with 10 ml xylocain 2 percent solution in the fossa poplitea after localization with a stimulation-needle (Labaz®). The innervation of the GM and SM was blocked to eliminate any involuntary muscle activity. The success of the nerve-blocking could be determined by loss of sensitivity in the heel. All measurements were performed between 1400 and 1600 hours.

## Converting the Measurements into Parameters

The following parameters were defined (see **Table 1**):

### Muscle Activation

Positive or excess signs include reflex release phenomena and inappropriate muscle activation. In a TAD procedure, the GM and SM are stretched during passive dorsiflexion, so the increase of the mean level of EMG activity in  $\mu$ V during the second half of dorsiflexion is used as a parameter of dynamic stretch reflex activity (DSR). In the first half of the dorsiflexion movement, the GM and SM are relaxed, so the mean level of EMG activity in  $\mu$ V is used as a parameter for tonic stretch reflex activity (TSR) of the GM and SM (**Figure 3**).

Inappropriate muscle activation can also be traced during FFRM. In voluntary movements, the threshold to elicit a stretch reflex is elevated, compared with imposed movements (24), but reciprocal excitation (a tonic activation of the antagonist of movement) is recorded in some subjects with UMNS (25). TSR of a

**Table 1.**

Parameters.

**Free Frequency Repetitive Movement Test:**

Excess parameters

MMA = Minimal Muscle Activity % = [minimal (MIN) EMG activity/maximal (MAX) EMG activity] · 100.

CC = Co-contraction in  $\mu\text{V}$ : Maximal EMG activity of the antagonist of movement - minimal EMG activity.

RC = Reciprocal Coefficient %: (the increase of EMG activity of the antagonist/the increase of EMG activity of the agonist of movement) · 100.

Deficit parameters

FREQ = Frequency (in Hz)

ROM = Range Of Motion (in degrees)

PS = Product Score: (FREQ) · (ROM) during 30 seconds.

VMA = Voluntary Muscle Activity in  $\mu\text{V}$ : Maximum (MAX) - Minimum (MIN).

ROP = Reduced Output Paresis %: ratio (VMA hemiplegic side/VMA unaffected side) · 100.

**Torque Angle Diagrams**

Excess parameters

DSR = Torque-Angle-Diagram Dynamic Stretch Reflex: mean EMG activity during the stretching phase of a muscle.

TSR = Torque-Angle-Diagram Tonic Stretch Reflex: mean EMG activity during the shortening phase of a muscle.

Muscle stiffness:

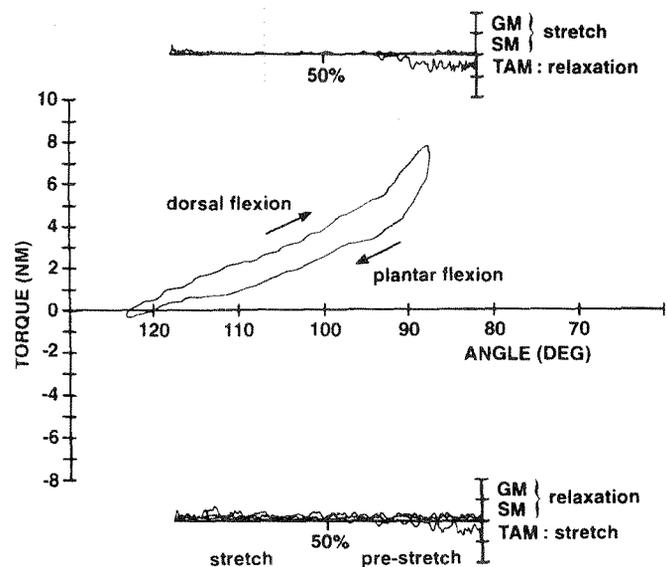
STIFF = The shift of the hysteresis curve over the axis of torque in Nm.

Muscle length:

LENGTH = The shift of the hysteresis curve over the axis of angle in degrees.

muscle is reflected as continuous muscle activation during stretching (26). To quantify the TSR level, the average minimal muscle EMG activity of a muscle is used, indicating the impairment of relaxation of a muscle. This is converted into the parameter minimal muscle activity (MMA), expressing the minimal level of EMG activity as a percentage of the maximal EMG activity during voluntary movement (**Figure 1**).

Co-contraction of the antagonist is probably also a sign of a self-induced stretch reflex, and can be traced during FFRM (9,27,28). The increase in EMG activity of a muscle at the moment of lengthening is defined as co-contraction (CC in  $\mu\text{V}$ ; see **Figure 1**). A relative parameter is the reciprocal coefficient: (the increase in

**Figure 3.**

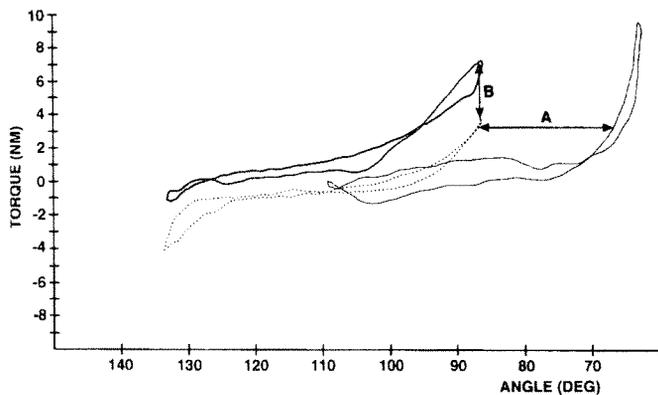
Torque angle diagram of the ankle during passive dorsal-plantar flexion with simultaneous registration of surface EMG activity on the hemiplegic side. GM=gastrocnemius muscle; SM=soleus muscle; TAM=tibialis anterior muscle. Top: registration of surface EMG during dorsiflexion: stretch of the GM and SM, relaxation of the TAM; Bottom: registration of surface EMG during plantar flexion: relaxation of the GM and SM, stretch of the TAM. EMG activity is represented in the direction of action on the torque.

EMG activity of the antagonist/the increase in EMG activity of the agonist of movement) · 100.

Negative or deficit signs of muscle activation include loss of dexterity, paresis, and enhanced fatigability. When the hemiplegic side is compared with the unaffected side, these properties of muscle function are expressed as the performance during FFRM. This is defined as product score in degrees: frequency of movement (Hz) · ROM (degrees) during a period of 30 s. The increase in EMG activity in  $\mu\text{V}$  of the agonist of movement is expressed as the voluntary muscle activity (VMA) in  $\mu\text{V}$  (**Figure 1**). The level of ROP is defined as the ratio (VMA hemiplegic side/VMA unaffected side) · 100.

**Muscle Stiffness**

The S-shape of the hysteresis curve in a TAD is determined by the stiffness of the TAM and the GM or SM. When there is no EMG activity present in the muscles, the shift of the hysteresis curve over the axis



**Figure 4.**

Torque angle diagrams of the ankle of a hemiplegic patient. Thin line=unaffected side; thick line=affected side; dotted line=affected side after local anaesthesia of the Tibial Nerve. No EMG activity was recorded. A=difference in muscle length (TAD-LENGTH); B=difference in muscle stiffness (TAD-STIFF).

of torque in Nm (STIFF) is to be considered as the difference in stiffness (**Figure 4**: shift B).

#### Muscle Length

This parameter can also be derived from a comparison of the TADs of the hemiplegic and the unaffected side. The shift of the hysteresis curve over the angle axis represents the decrease in muscle length. By fitting the neutral position of the S-shape on the angle axis, the level of muscle shortening can be measured in degrees (LENGTH, see **Figure 4**: shift A). For a left-right comparison of the curves, absence of EMG activity is required. All parameters are summarized in **Table 1**.

#### Validation Study

Two nonimpaired control subjects (male, 36 yrs; female, 45 yrs) were measured by means of FFRM and TAD.

Ten subjects with a spastic hemiplegia were included in the study. Inclusion criteria were: 1) voluntary movement of the lower limb using flexor and extensor synergies, as on stage 3 on the Fugl-Meyer Motor Assessment Scale (29); 2) involuntary extensor synergy in supine position in the hemiplegic lower limb; 3) equinus position of the foot in barefoot walking during the swing phase; 4) ability to walk outdoors; 5) a minimum of 12 mo since the onset of the spastic hemiplegia. The use of a walking aid and/or an

**Table 2.**

Subject characteristics.

SUB	DIAGN	AGE	SEX	DUR	ASHW
1	STROKE	55	M	12	3
2	STROKE	67	M	10	3
3	CC	26	M	18	2
4	STROKE	38	M	10	2
5	STROKE	59	M	8	2
6	CC	28	M	10	2
7	STROKE	66	F	10	3
8	STROKE	56	F	6	2
9	CP	25	F	BIRTH	3
10	STROKE	57	M	12	3

SUB=subject; DIAGN=diagnosis; STROKE=first ischemic or hemorrhagic stroke of a cerebral hemisphere; CC=contusio cerebri; CP=cerebral palsy; DUR=duration since onset in years; ASHW=Ashworth score.

ankle-foot orthosis was allowed. Full consent, according to the declaration of Helsinki, was obtained from the subjects. Their characteristics and medical diagnoses are summarized in **Table 2**. Their mean age was 47.7 yrs (range 25–67), and the average time from onset to the first session was 10.6 yrs (range 6–18).

## RESULTS

### Characteristics of the Apparatus and Measurement Procedure

After a period of training, the staff were easily able to perform both FFRM and TAD measurements within 30 min.

#### Free Frequency Repetitive Movement

**Table 3** gives a summary of the results of measurements of 15 sessions (5 sessions a day at 1-wk intervals) in the 2 controls. As plantarflexion of the ankle in a sitting position occurs with the aid of gravity, the VMA levels of the GM are low. No learning effects were noticed. The left side showed a lower product score than the right side (the subjects were dextral), but the VMA of the TAM was slightly higher. Differences between the right and the left side are also indicated in the table.

#### Torque Angle Diagram

Initially, measurements were carried out to establish the accuracy of the instrument. With an empty footplate, after correcting for the effect of gravity, a flat

**Table 3.**  
Free frequency repetitive movement data from nonimpaired control subjects.

Variable	Right Side		Left Side		P	
	Mean	(SD)	Mean	(SD)		
<b>SUB A</b>						
FREQ	Hz	2.55	0.35	2.31	0.15	**
ROM	°	31.07	3.77	32.27	6.13	NS
PS	°	4268.53	619.73	4127.47	564.35	NS
TAM.VMA	μV	261.47	29.66	273.00	22.87	NS
ROP	%	97.06	9.16			
TAM.CC	μV	0.00	0.00	0.00	0.00	NS
TAM.MMA	%	8.79	2.37	2.52	0.89	**
GM.VMA	μV	55.20	11.37	14.40	5.73	**
GM.RC	%	3.39	1.17	2.98	0.29	NS
GM.CC	μV	9.87	3.87	8.33	1.05	NS
GM.MMA	%	16.82	4.57	28.09	7.97	**
<b>SUB B</b>						
FREQ	Hz	1.92	0.06	1.85	0.10	*
ROM	°	29.13	4.03	24.07	4.99	**
PS	°	3268.40	448.96	2617.07	526.37	**
TAM.VMA	μV	142.53	15.40	157.40	22.35	*
ROP	%	92.38	16.63			
TAM.CC	μV	0.00	0.00	0.00	0.00	NS
TAM.MMA	%	3.97	0.68	3.10	1.96	NS
GM.VMA	μV	16.07	1.94	14.73	5.27	NS
GM.RC	%	10.94	1.75	8.94	2.27	*
GM.CC	μV	16.07	1.94	14.73	5.27	NS
GM.MMA	%	17.84	7.36	16.60	8.54	NS

Each subject (SUB) underwent 15 sessions; TAM=tibialis anterior muscle; GM=gastrocnemius muscle; P=P-value paired T-test: \*= $P < 0.05$ ; \*\*= $P < 0.01$ . Explanation of variables: see **Table 1**.

horizontal line was recorded. With a weight of 1 kg, similar to that of an average foot, the effect of inertia was studied. Within the range of 0.09–0.83 Hz and a ROM of 51°, a maximum variation of 0.5 Nm was recorded. Online recordings (with corrections for gravity) revealed a noise of 1.5° and 0.5 Nm caused by the A/D-conversion of the signals in the microcomputer. The influence of the subject's sitting position and the alignment of the ankle axis were also determined: only serious misalignment of the ankle axis influenced the recordings.

Subsequently, experiments were conducted with controls. No difference in TAD was found between the right and left side (**Figure 5** as compared with **Figure 4**). In measurements on 5 different days at an angle of 70° (i.e., 20° dorsiflexion), a maximum difference of 1 Nm was recorded (within-subject variance and error of measurement). No effect of the frequency of movement on the hysteresis curve (range 0.08–0.80 Hz), represent-

ing the viscous stiffness, was found in this range of frequencies (16).

### Measurements in Controls

Our first hypothesis was that parameters expressing abnormal muscle activation would be low in nonimpaired subjects. This was confirmed in the two controls (**Table 3**) for all excess parameters. EMG activity could only be recorded in shortening muscles. No signs of abnormal muscle activation (DSR or TSR) were found (**Figure 3**).

### Measurements in Hemiplegic Subjects

#### *Comparison of the Affected and the Unaffected Sides*

The second hypothesis assumes that differences are expected to be found between the hemiplegic and unaffected side.

Excess symptoms: both the hemiplegic and the unaffected side showed some TSR completely absent in

the controls (Table 4). Minimal muscle activity was elevated, as expected. The level of co-contraction was unexpectedly low, and did not exceed the values of controls ( $15 \pm 5 \mu\text{V}$ ).

Deficit symptoms: as expected, a deficit in performance and voluntary muscle activation during FFRM was found for all parameters on the hemiplegic side ( $p < 0.01$ , see Table 5).

**Table 4.**

Muscle activation in 10 subjects with: hemiplegia excess symptoms.

TAD		HS		HS.A		US	
		mean	(SD)	mean	(SD)	mean	(SD)
FK							
GM DSR	$\mu\text{V}$	0.1	(0.6)	0.2	(0.4)	1.1	(1.7)
GM TSR	$\mu\text{V}$	1.9	(0.7)	1.1	(0.6)	1.5	(0.7)
SM DSR	$\mu\text{V}$	0.8	(0.8)	0.3	(0.9)	1.0	(1.8)
SM TSR	$\mu\text{V}$	4.4	(6.5)	1.6	(0.6)	5.6	(5.4)
EK							
GM DSR	$\mu\text{V}$	0.2	(1.2)	0.1	(0.7)	0.7	(1.1)
GM TSR	$\mu\text{V}$	4.7	(7.7)	2.8	(3.7)	3.3	(4.2)
SM DSR	$\mu\text{V}$	0.9	(1.2)	0.1	(0.3)	0.1	(0.7)
SM TSR	$\mu\text{V}$	5.6	(9.7)	2.9	(4.0)	3.8	(2.7)
FFRM							
GM MMA	%	18.7*	(28)	2.9	(8.6)	10.5	(6.3)
GM RC	%	30.0	(83)	6.4	(10.8)	7.1	(5.3)
GM CC	$\mu\text{V}$	2.6*	(2.8)	3.6	(6.3)	11.3	(7.9)
TAM MMA	%	33.8*	(26.3)	35.4	(19.9)	10.6	(7.0)
TAM CC	$\mu\text{V}$	5.1*	(7.4)	3.4	(6.1)	13.3	(9.5)

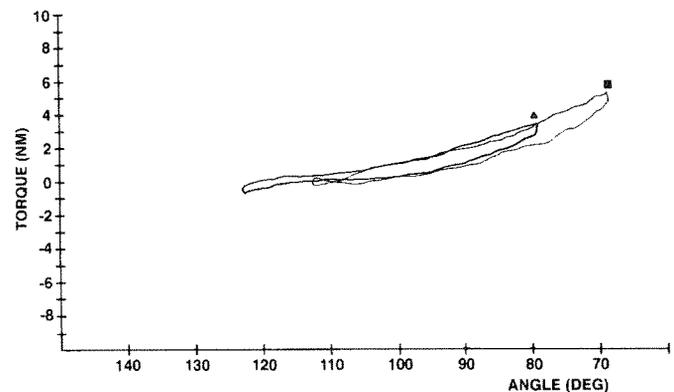
FK=flexed knee; EK=extended knee; HS=hemiplegic side; HS.A=hemiplegic side after anaesthesia; US=unaffected side; TAD=torque angle diagram; FFRM=free frequency repetitive movement. Explanation of the parameters; see Table 1. \*= $P < 0.05$  paired T-Test: HS/US. Limitations in the capacity of the computer made it impossible to make FFRM recordings of the soleus muscle as well.

**Table 5.**

Muscle activation in 10 subjects with: hemiplegia deficit symptoms.

FFRM		HS		HS.A		US	
		mean	(SD)	mean	(SD)	mean	(SD)
PS	$^{\circ}$	1038	(788)	971	(887)	2980	(1029)
FREQ	Hz	0.80	(0.15)	0.78	(0.28)	1.08	(0.32)
ROM	$^{\circ}$	21	(14)	21	(12)	50	(20)
TA VMA	$\mu\text{V}$	53	(45)	58	(61)	166	(26)
ROP	%	25	(15)	28	(33)		

HS=hemiplegic side; HS.A=hemiplegic side after tibial nerve anaesthesia; US=unaffected side; FFRM=free frequency repetitive movements. Parameters: see Table 1.



**Figure 5.**

Torque angle diagrams of the ankle of a nonimpaired subject; no surface EMG activity was recorded.

### Muscle Stiffness

Differences in muscle stiffness (STIFF) between the hemiplegic and unaffected sides were not significant and showed a large between-subject variance (Table 6). However, in subjects 2, 4, 7, 9, and 10, a significant increase ( $>2$  SD) in stiffness on the hemiplegic side was found. Furthermore, stiffness was significantly increased in the group of subjects with an Ashworth spasticity score of 3, compared to those with an Ashworth score of 2.

### Muscle Length

A shortened SM on the hemiplegic side, compared with the unaffected side, was found in nine subjects (mean  $9.8^{\circ}$ ) and both a shortened SM and a shortened GM (mean  $6.8^{\circ}$ ) in eight (Table 7). The presence of a shortened muscle showed no correlation with the parameters of muscle stiffness, excess symptoms, or Ashworth score. The second hypothesis was thus confirmed for all four groups of parameters, except for the parameter of co-contraction.

### Comparison of Muscle Function before and after Local Anaesthesia of the Tibial Nerve

The third hypothesis assumes a decrease in excess parameters of muscle activation, while muscle stiffness remains unchanged. This hypothesis has been confirmed: after local anaesthesia of the TN, all excess symptoms decreased, as was expected (Table 4). With regard to deficit symptoms, seven subjects deteriorated in performance, and two improved (Table 8).

Muscle stiffness did not decrease significantly after local anaesthesia, confirming that it represents biomechanical properties of the muscle-tendon complex (Table 6). The significant difference in stiffness of the hemiplegic side, compared with the unaffected side, for the group with an Ashworth score of 3, was unaffected by nerve-blocking.

For the determination of differences in muscle length, no EMG activity must be present. For that reason, muscle length can only be assessed after nerve-blocking, so it is impossible to assess any difference before and after that intervention.

### Inter-individual Differences

The fourth hypothesis assumes that, notwithstanding a uniform spastic gait pattern, differences can be detected with regard to all four groups of parameters. This hypothesis was also confirmed.

#### Excess parameters

Elevated MMA of the GM was present in five subjects. None had an increased level of DSR. One had a high level of TSR, in combination with high MMA and reciprocal coefficient of the GM. This caused a substantial decrease in dorsiflexion of the ankle joint, which improved after blocking the TN (Figures 6a and 6b).

Deficit parameters also showed considerable variation. The difference between the ROP and the product score in relation to the unaffected side suggested differences in the efficacy of force generation in relation to muscle activation in subjects (Table 8).

Abnormal stiffness of the SM (with flexed knee) was recorded in three subjects, all with a high Ashworth score. Increased stiffness of the GM (with extended knee) was measured in four individuals; one had a normal Ashworth score. Two showed an increase in muscle stiffness within one session during TAD after local anaesthesia, without any EMG activity (Figure 7).

**Table 6.**

Muscle stiffness in 10 subjects with hemiplegia.

	HS-US		HS-HS.A		HS.A-US	
	mean	(SD)	mean	(SD)	mean	(SD)
Whole group						
FK Nm	0.63	(1.72)	0.38	(2.58)	0.25	(1.77)
EK Nm	1.08	(2.37)	0.22	(2.37)	0.85	(1.47)
Subgroup (N=5): Ashworth 3						
FK Nm	1.75*	(1.66)			2.05*	(2.32)
EK Nm	1.43	(2.00)			0.68	(2.89)
Subgroup (N=5): Ashworth 2						
FK Nm	-0.50	(0.87)			-1.30	(1.60)
EK Nm	0.72	(3.53)			-0.23	(1.94)

FK=flexed knee; EK=extended knee; HS=hemiplegic side; HS.A=hemiplegic side after tibial nerve anaesthesia; US=unaffected side. Ashworth 2=marked increase in tone. Ashworth 3=considerable increase in tone. Parameters: see Table 1. \* $p < 0.05$  unpaired T-test: Ashworth 3 versus Ashworth 2.

**Table 7.**

Muscle length in subjects with hemiplegia.

SUB	EK	FK
1	11	14
2	4	7
3	0	0
4	12	9
5	23	10
6	5	8
7	6	0
8	22	5
9	10	7
10	5	8

Muscle shortening of the hemiplegic side in degrees, compared with the unaffected side. SUB=subject; EK=extended knee; FK=flexed knee.

**Table 8.**

Deficit parameters in 9 subjects with hemiplegia.

SUB	PS%		ROP		ASHW
	HS	HS-A	HS	HS.A	
2	33	41	0	0	3
3	47	45	36	109	2
4	9	9	2	9	2
5	74	23	35	19	3
6	12	2	15	18	2
7	21	12	15	18	3
8	37	31	5	12	2
9	52	29	60	33	3
10	19	25	15	15	3

SUB=subject; PS%=product score; ROP=reduced output paresis; ASHW=Ashworth score; HS=hemiplegic side; HS.A=hemiplegic side after tibial nerve anaesthesia. Data from SUB 1 are missing.

### Muscle Length

The SM was shortened in nine subjects, the GM in eight (Table 7). A shortened GM was always accompanied by a shortened SM. No correlation was found between muscle shortening and excess symptoms.

## DISCUSSION

We formulated four hypotheses. The first assumes that parameters expressing abnormal muscle activation would be low in nonimpaired subjects. This hypothesis was confirmed. The second hypothesis assumes that differences between the hemiplegic and the unaffected side are expected to be found in all four groups of parameters (i.e., excess symptoms, deficit symptoms, muscle stiffness, and muscle length). This hypothesis was confirmed for most parameters. Unexpectedly, both the hemiplegic and the unaffected sides showed some TSR. Also unexpected was the low level of co-contraction of the GM, probably indicating that the clinical significance of this phenomenon is limited.

The third hypothesis was also confirmed: after nerve-blocking, parameters expressing abnormal muscle activation decreased, while parameters expressing muscle stiffness remained unchanged. Only excess symptoms were influenced by TN local anaesthesia.

The fourth hypothesis assumes that between-subject differences are to be expected. Differences were found with regard to disturbed muscle activation, muscle stiffness, and muscle length. This supports the proposed classification of muscle dysfunction in UMNS. No signs were found of an increased level of DSR. Undoubtedly, this was due to the conditions of the measurements: the maximal angular velocity of 30°/s and the sinusoidal shape of the imposed movements (30).

The results are discussed according to the proposed classification:

### Changes in Muscle Activation

The excess parameters, derived from the measurements, showed specific and coherent changes after local anaesthesia of the TN. The parameters expressing TSR in TAD and MMA in FFRM were elevated on the hemiplegic side, and diminished after nerve-blocking. However, the product score improved slightly in only two subjects. This raises the question of whether the negative influence of stretch reflexes on voluntary muscle function has been overrated.

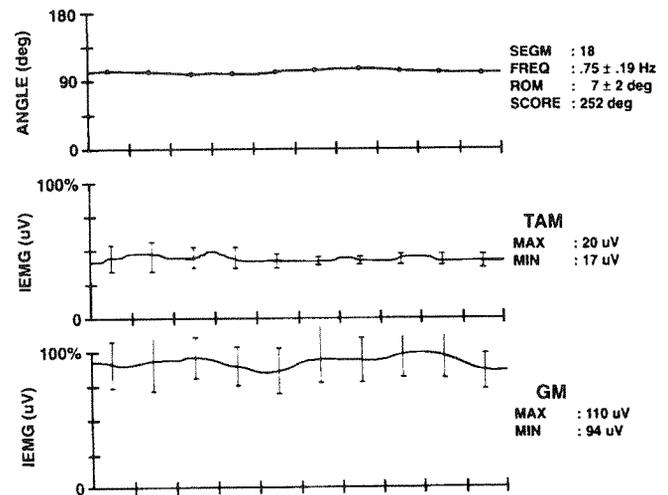
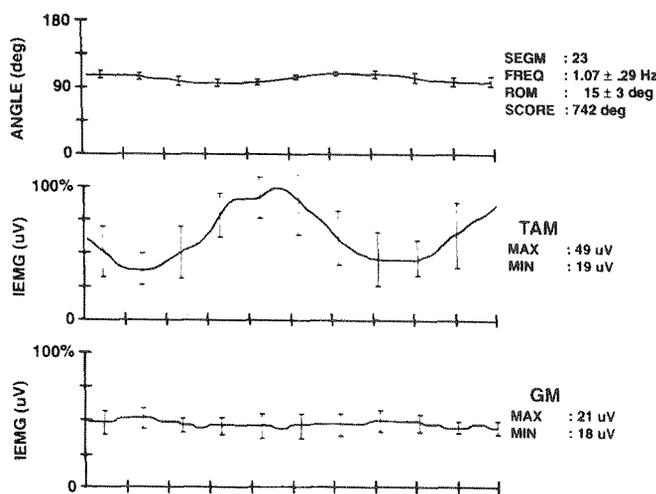


Figure 6a.

Report of an FFRM test of a subject on the hemiplegic side before local anaesthesia of the TN. TAM=tibialis anterior muscle; GM=gastrocnemius muscle; SEGM=number of segments of movement; FREQ=frequency; ROM=range of motion; Score=product score; X-axes: One and a half cycles of movement (dorsiflexion-plantarflexion-dorsiflexion). Y-axes: top, ankle movement in degrees. Mean values of 18 cycles calculated, 1 SD plotted: only 7 degrees of movement present; middle, the integrated (smoothed rectified) EMG of the TAM is represented: 100 percent mean IEMG activity is set to 40  $\mu$ V; almost continuous activation of the TAM is present; bottom, the IEMG activity of the medial head of the GM is shown: 100 percent is the mean maximal IEMG activity; a high level of continuous activation of the GM is present.

Deficit symptoms, expressed in ROP and the product score, were responsible for low performance during FFRM. Although all subjects were scored at stage 3 of the Fugl-Meyer Motor Assessment Scale, there was a remarkable difference between the performance of subjects during FFRM. The FFRM test therefore seems to be a sensitive method of measuring voluntary muscle performance. Differences between the ROP and the product score, both expressed as a percentage of the value of the unaffected side (Table 8), support the assumption that the EMG-force relationship in some subjects is disturbed (5,31,32).

After local anaesthesia of the TN, the mean product score of the hemiplegic side during FFRM showed a slight decrease. However, the individual scores showed either an increase or a decrease in product score performance, which was related in one person with an elevated reciprocal coefficient of the GM. This parameter might be able to predict the effect



**Figure 6b.**

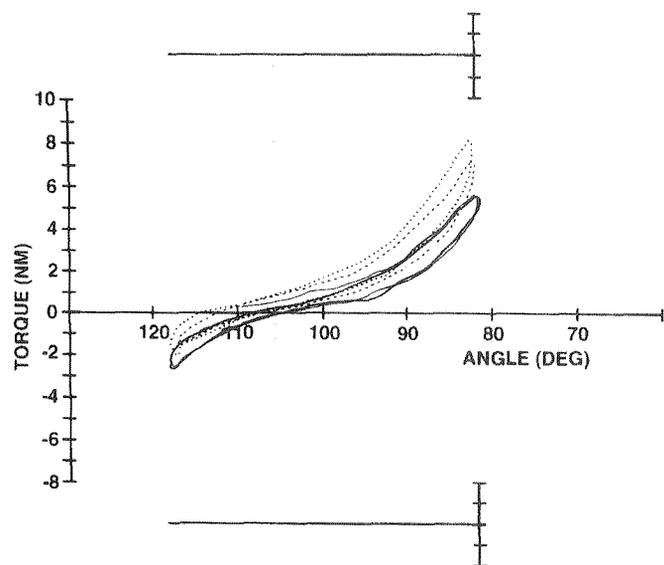
Report of an FFRM test of a subject on the hemiplegic side after local anaesthesia of the TN. TAM=tibialis anterior muscle; GM=gastrocnemius muscle; SEGM=number of segments of movement; FREQ=frequency; ROM=range of motion; Score=product score; X-axes: one and a half cycles of movement (dorsiflexion-plantarflexion-dorsiflexion); Y-axes: top, ankle movement in degrees; mean values of 23 cycles calculated: 15 degrees of movement registered; middle, the integrated (smoothed rectified) EMG of the TAM: 100 percent mean IEMG activity is set to the mean maximal activity (49  $\mu$ V); a decrease in continuous activation of the TAM is present, and now phasic activity is present; bottom, the IEMG activity of the medial head of the GM is shown in the same way (100 percent is set to 40  $\mu$ V); also, a decreased continuous activation of the GM is present.

of nerve-blocking in the treatment of spasticity of the GM and the SM.

#### Muscle Stiffness

Muscle stiffness, measured by comparing the position of the hysteresis curve on the torque axis of the affected side with the unaffected side, showed a good relationship with the clinical Ashworth score. As muscle stiffness remained unchanged after blocking the TN, the pathophysiological origins of spasticity and elevated muscle stiffness are likely to be different.

Increased stiffness without EMG activity after local nerve-blocking, and the absence of dorsiflexion of the foot in gait, in spite of EMG activity of the TAM and without co-activation of the SM and GM, have also been reported in other studies (3,6). The initial decrease in muscle stiffness immediately after nerve-blocking (Figure 7) suggests, nevertheless, an influence of neural activity. However, muscle stiffness seems to be inde-



**Figure 7.**

Torque angle diagram of the ankle in the hemiplegic side 30 min after local anaesthesia of the TN. GM=gastrocnemius muscle; SM=soleus muscle; TAM=tibialis anterior muscle; solid lines: before nerve-blocking; dotted lines: after nerve-blocking; Increase in torque level is found after local anaesthesia without surface EMG activity.

pendent of muscle activation, recorded with surface EMG. The nature of this phenomenon is not clear.

#### Muscle Shortening

As was expected, the measurements objectified the presence of a shortened SM and/or GM in most, but not all subjects. This finding is in accordance with clinical experience. The method of measurement, comparing the hysteresis curve of the affected side with that of the unaffected side, allows only an estimation of the degree of shortening, as the level of applied external force influences the measurement of muscle shortening when there is a combination of elevated stiffness and shortening of a muscle.

In accordance with the literature, we found a considerable between-subject variance of TAD in the ankle, associated with age and sex (33). No relationship was found between excess parameters, muscle stiffness, and muscle shortening. As all subjects had a stable hemiplegia for at least 6 years, spasticity could have occurred in the first years after any stroke, causing the muscle shortening. It remains unclear why muscle shortening occurs in some subjects and not in others.

Measuring differences in muscle dysfunction in hemiplegic persons may offer an indication for the

selection of therapy. Spasmolytic drugs to enhance presynaptic inhibition in the spinal cord, such as baclofen, are effective in those with excess symptoms. Elevated muscle stiffness could be influenced by dantrolene sodium. When muscle shortening is already present, high-heeled walking or surgical lengthening is possible. The results of this study show that it is possible to measure different types of muscle dysfunction, which offers a potential for the objective evaluation of therapy.

However, no definite conclusions can be drawn from this study. Only a small number of persons was studied, and although their clinical characteristics were similar, their spastic hemiplegia had different causes. Larger groups of subjects with a homogeneous medical background need to be studied to confirm the assumed heterogeneity in muscle impairments.

## REFERENCES

- Lance JW. Symposium synopsis. In: Feldman RG, Young RR, Koella WP, editors. Miami: Symposia Specialists; 1980. p. 485-500.
- Ashworth B. Preliminary trial of carisoprodol in multiple sclerosis. *Practitioner* 1964;192:540-2.
- Sloan RL, Sinclair E, Thompson J, Taylor S, Pentland B. Inter-rater reliability of the modified Ashworth Scale for spasticity in hemiplegic patients. *Int J Rehabil Res* 1992;15:158-61.
- Bohannon RW, Smith MB. Interrater reliability of a modified Ashworth scale of muscle spasticity. *Phys Ther* 1987; 67:206-7.
- Fellows SJ, Kaus C, Ross HF, Thilmann AF. Agonist and antagonist EMG activation during isometric torque development at the elbow in spastic hemiparesis. *Electroencephalogr Clin Neurophysiol* 1994;93:106-12.
- Thilmann AF, Fellows SJ. The time-course of bilateral changes in the reflex excitability of relaxed triceps surae muscle in human hemiparetic spasticity. *J Neurol* 1991; 238:293-8.
- Thilmann A, Fellows SJ. The mechanism of spastic muscle hypertonus. *Brain* 1991;114:233-44.
- Sahrmann SA, Norton BJ. The relationship of voluntary movement to spasticity in the upper motor neuron syndrome. *Ann Neurol* 1977;2:460-5.
- McLellan DL. Co-contraction and stretch reflexes in spasticity during treatment with baclofen. *J Neurol Neurosurg Psychiatr* 1977;40:1-30.
- Knutsson E, Martensson A. Dynamic motor capacity in spastic paresis and its relation to prime motor dysfunction, spastic reflexes and antagonist co-activation. *Scand J Rehabil Med* 1980;12:93-106.
- Knutsson E, Richards C. Different types of disturbed motor control in gait of hemiparetic patients. *Brain* 1979;102:405-30.
- Levin MF, Hui-Chan C. Ankle spasticity is inversely correlated with antagonist voluntary contraction in hemiparetic subjects. *Electromyogr Clin Neurophysiol* 1994;34:415-25.
- Bohannon RW, Smith MB. Assessment of strength deficits in eight paretic upper extremity muscle groups of stroke patients with hemiplegia. *Phys Ther* 1987;67:522-5.
- Katz RT, Rymer WZ. Spastic hypertonia: mechanisms and measurement. *Arch Phys Med Rehabil* 1989;70:144-55.
- Thilmann AF, Fellows SJ, Ross HF. Biomechanical changes at the ankle joint after stroke. *J Neurol Neurosurg Psychiatr* 1991;54:134-9.
- Lehmann JF, Price R, deLateur BJ, Hinderer S, Traynor C. Spasticity: quantitative measurements as a basis for assessing effectiveness of therapeutic intervention. *Arch Phys Med Rehabil* 1989;70:6-15.
- Sinkjaer T, Magnussen I. Passive, intrinsic and reflex-mediated stiffness in the ankle extensors of hemiparetic patients. *Brain* 1994;117:355-63.
- Halar EM, Stolov WC. Gastrocnemius muscle belly and tendon length in stroke patients and able-bodied persons. *Arch Phys Med Rehabil* 1978;59:476-84.
- Hillstrom HJ, Perlberg G, Siegler S, Sanner WH. Objective identification of ankle equinus deformity and resulting contracture. *J Am Podiatric Med Assoc* 1991;81:519-24.
- Tardieu G, Tardieu C, Colbeau-Justin P, Lespargot A. Muscle Hypoextensibility in children with cerebral palsy: II. therapeutic implications. *Arch Phys Med Rehabil* 1982;63:103-7.
- Shiavi R, Green N. Ensemble averaging of locomotor electromyographic patterns using interpolation. *Med Biol Eng Comput* 1983;21:573-8.
- Inman VT. The joints of the ankle. Baltimore: Williams & Wilkins; 1976.
- Pronk C, Niesing R. Apparatus for measuring the functional capacity of the knee extensors and flexors. *Med Biol Eng Comp* 1983;21:764-7.
- Gottlieb GL. Hyper-reflexia and disordered voluntary movement. In: Thilmann AF, Burke DJ, Rymer WZ, editors. Spasticity: mechanisms and management. Berlin: Springer-Verlag; 1993. p. 155-66.
- Ibrahim IK, Berger W, Trippel M, Dietz V. Stretch-induced electromyographic activity and torque in spastic elbow muscles; differential modulation of reflex activity in passive and active motor tasks. *Brain* 1993;116:971-89.
- Gottlieb G, Agerwal GC. Dynamic relationship between isometric muscle tension and the electromyogram. *J Appl Physiol* 1971;30:345-51.
- Gottlieb GL, Myklebust BM, Penn RD, Agarwal GC. Reciprocal excitation of muscle antagonists by the primary afferent pathway. *Exp Brain Res* 1982;46:454-6.
- Dewald JP, Pope PS, Given JD, Buchanan TS, Rymer WZ. Abnormal muscle coactivation patterns during isometric torque generation at the elbow and shoulder in hemiparetic subjects. *Brain* 1995;118:495-510.
- Fugl-Meyer AR, Jaasko LLI. The post-stroke hemiplegic patient: I. a method for evaluation of physical performance. *Scand J Rehabil Med* 1975;7:13-31.

30. Rack P, Ross HFTF. Ankle stretch reflexes in normal and spastic subjects. *Brain* 1984;107:637-54.
31. Berger W, Horstmann GA, Dietz V. Spastic paresis: impaired spinal reflexes and intact motor programs. *J Neurol Neurosurg Psychiatr* 1988;51:568-71.
32. Dietz V, Trippel MBW. Reflex activity and muscle tone during elbow movements in patients with spastic paresis. *Ann Neurol* 1991;30:767-79.
33. Sepic S, Murray P, Mollinger LA. Strength and range of motion in the ankle in two age groups of men and women. *Am J Phys Med* 1986;65:75-84.

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