Clinical Rehabilitation 2005; **19:** 146–154

Comparison of the impact of a single session of isokinetic or isotonic muscle stretch on gait in patients with spastic hemiparesis

V Maynard Faculty of Health and Social Work, University of Plymouth, **AMO Bakheit** Peninsula Medical School and Plymouth Primary Care Trust and **S Shaw** School of Mathematics and Statistics, University of Plymouth, Plymouth, UK

Received 28th February 2004; returned for revisions 24th August 2004; revised manuscript accepted 5th September 2004.

Objective: To establish if isokinetic and isotonic muscle stretch (with or without weight-bearing) of the ankle plantar flexors improves gait in hemiplegic patients. A further aim was to compare the effectiveness of these treatment methods.
Design: A randomized, parallel group prospective study.
Setting: A stroke rehabilitation unit.
Subjects: Ambulatory hemiparetic stroke patients with mild to moderately severe muscle hypertonia of the lower limb and a group of healthy control subjects.
Intervention: Subjects were randomized to receive a single 20-min session of isokinetic muscle stretch or isotonic muscle stretch with or without weight-bearing.
Outcome measures: Selected kinematic, kinetic and spatio-temporal gait parameters were measured at baseline, immediately after the muscle stretch and 24 h later.
Results: Sixty-six patients and 21 healthy control subjects were recruited and completed the study. There were statistically significant differences between the patient groups and the healthy subjects on most of the gait parameters studied. However, the differences between the patient groups or between the three measurements over time for each type of muscle stretch did not reach statistical significance.
Conclusions: A single session of isokinetic or isotonic muscle stretch (with or without weight-bearing) of the ankle plantar flexors has no clinically observable effect on the gait of hemiplegic stroke patients.

Introduction

Spastic hemiplegia frequently results in significant gait abnormalities due to a combination of muscle weakness and hypertonia. Although the increased muscle tone is usually beneficial, in some cases it can interfere with safe and energy efficient walking.[1,2] For example, hypertonia of the ankle plantar flexors often does not allow the correct foot placement on the ground in the stance phase of the gait cycle and can also prevent adequate clearance of the ground in the swing phase. This may cause the patient to trip over and, consequently increases the risk of falls. In addition, muscle hypertonia increases the physiological cost of ambulation.[3] Therefore, one of the important strategies of gait training in patients with upper motor neurone lesions is to control the reversible component of muscle hypertonia (i.e., spasticity) with physical and pharmacological methods.

Address for correspondence: Professor AMO Bakheit, Beauchamp Centre, Mount Gould Hospital, Plymouth PL4 7QD, UK. e-mail: magid.bakheit@pcs-tr.swest.nhs.uk

10.1191/0269215505cr853oa

Table 1 Definitions of isokinetic and isotonic stretch

Isotonic muscle stretch	The slow, passive movement of the limb to its maximum range of motion and then holding the limb in this position. When the stretched position of the limb is maintained through the patient's body weight support in standing this is described as isotonic stretch with weight-bearing
Isokinetic muscle stretch	The passive movement of the spastic limb with a constant force and angular velocity

Different types of muscle stretch are used in the management of spasticity. Isokinetic muscle stretch has been shown to reduce spasticity by altering the spinal reflex mechanisms and the visco-elastic properties of the hypertonic muscle.[4] Similar results were obtained with isotonic stretch.[5,6] (For the definition of isotonic and isokinetic stretch see Table 1.) Although isokinetic and isotonic muscle stretch reduce muscle hypertonus, their effect on motor function has not been conclusively demonstrated. Furthermore, it is not clear which of these methods results in a better and more lasting functional change.

The main aim of the present study was to establish whether isokinetic and isotonic muscle stretch (with and without weight-bearing) of the ankle plantar flexors improves gait in hemiplegic patients. A further aim was to compare the effectiveness of these methods.

Subjects and methods

Adult patients with stroke of at least six weeks duration and resulting in hemiplegia were asked to take part in the study. Ambulatory patients with spasticity of the ankle plantar flexors were recruited if they had at least a score of 1 on the Modified Ashworth Scale[7] (MAS) and were not receiving antispasticity drugs. Those with fixed muscle contractures at the ankle joint were excluded. A fixed muscle contracture was defined as a score of 5 on the MAS. (In this study the MAS score 1+ was interpreted as 2, thus making the maximal MAS score = 5) Apparently healthy age- and sex-matched subjects were recruited as a control group. None of these subjects had a history of neurological or musculoskeletal disease. The study was approved by the local research ethics committee and all participants gave informed written consent.

Following a baseline assessment, patients were randomly allocated to one of three groups and received either isotonic muscle stretch with weight-bearing, isotonic stretch without weight-bearing or isokinetic stretch. For each patient group there was a randomly allocated control group of healthy subjects. Comparison with healthy controls was considered necessary in order to control for the effects of age on gait. The randomization procedure was based on a system of sealed envelopes. Assessment was repeated immediately after the muscle stretch and again 24 h later. The purpose of the assessment 24 h after the intervention was to see if there was a carryover effect of treatment.

Table 2 The demographic and clinical characteristics of the study groups

Variable	Isokinetic stretch		Isotonic stretch		Isotonic stretch + weight-bearing	
	Controls ($n = 7$)	Patients ($n = 22$)	Controls ($n = 7$)	Patients ($n = 22$)	Controls ($n = 7$)	Patients ($n = 22$)
Mean age (range) in years	58.1 (48–69)	60.4 (33–76)	53.4 (43–78)	59.8 (33–79)	52.4 (38–70)	57.9 (35–81)
Male:female	2:5	16:6	6:1	15:7	2:5	16:6
Mean duration since stroke (SD) in months	NA	16.1 (12.7)	NA	12.8 (12.0)	NA	12.4 (12.4)
Mean MAS (SD) at baseline	NA	1.6 (0.7)	NA	1.5 (0.7)	NA	1.5 (0.7)

MAS, Modified Ashworth Scale.

Table 3 The mean (SD) of the kinematic and kinetic gait parameters for the whole group of patients and control subjects at baseline

Parameter	Controls	Patients
Hip angle		
IC	30.8 (6.5)	25.5 (9.0)
MST	7.9 (6.8)	11.1 (10.0)
MSW	27.6 (5.8)	26.0 (8.2)
Knee angle		
IC	8.2 (3.1)	15.4 (5.5)*
MST	12.9 (4.1)	10.5 (10.1)
MSW	50.3 (6.9)	29.4 (11.4)*
Ankle angle		
IC	3.5 (3.5)	−1.5 (9.4)*
MST	12.1 (2.1)	9.3 (6.4)
MSW	7.4 (3.3)	1.7 (9.4)*
Hip power		
IC	−0.04 (0.1)	−0.07 (0.2)
MST	0.01 (0.3)	0.03 (0.2)
MSW	0.02 (0.0)	0.02 (0.0)
Knee power		
IC	0.25 (0.2)	−0.01 (0.1)*
MST	0.02 (0.0)	0.04 (0.0)
MSW	−0.01 (0.01)	−0.08 (0.0)*
Ankle power		
IC	−0.01 (0.0)	−0.01 (0.0)
MST	−0.3 (0.2)	−0.08 (0.1)*
MSW	0.003 (0.0)	0.003 (0.0)*
Hip moment		
IC	0.17 (0.1)	−0.01 (0.1)*
MST	0.02 (0.1)	0.07 (0.1)
MSW	−0.02 (0.1)	−0.03 (0.0)
Knee moment		
IC	−0.11 (0.0)	−0.03 (0.0)*
ST	−0.02 (0.01)	0.05 (0.3)
SW	−0.04 (0.0)	−0.05 (0.1)
Ankle moment		
IC	−0.08 (0.1)	−0.03 (0.0)
MST	0.01 (0.0)	0.42 (0.3)*
MSW	−0.008 (0.0)	0.01 (0.2)

IC, initial contact; MST, mid-stance; MSW, mid-swing phase of the gait cycle.
*$p \leq 0.01$.

The therapeutic interventions

Isotonic stretch of the calf muscles of the hemiplegic leg was delivered with a Biodex dynomometer (Biodex Corporation, Shirley, New York, USA). The subjects were positioned in semi-reclined sitting with the knees and hips fully extended and the ankle resting on the footplate of the dynamometer so that the anatomical axis of the ankle was aligned with the shaft of the dynamometer. The footplate was then rotated until the maximum ankle dorsiflexion was achieved. The stretched muscles were maintained in this position for 20 min.

For isotonic stretch with weight-bearing the subjects were asked to lie flat on a tilt table with their hips and knees fully extended. The footplate was adjusted until the maximum ankle dorsiflexion was obtained. The tilt table was then slowly raised and kept in the vertical position.

Isokinetic stretch was delivered with a speed controlled stretch with the dynamometer. Treatment was given with the subjects in the same semi-reclined position as for isotonic stretch. The dynamometer delivered sinusoidal displacements from maximum ankle plantar flexion to maximum ankle dorsiflexion at a velocity of 30°/s and a torque limit of 30 Nm/kg. The velocity of 30°/s was chosen because it is sufficient to stretch the muscles without triggering a stretch reflex.[8] Subjects received one session of treatment. The duration of stretch was the same for all treatment groups and lasted 20 min.

The outcome measures

The effects of muscle stretch on gait were evaluated with fully computerized laboratory gait analysis immediately after the intervention and 24 h later.

Laboratory gait analysis

Selected kinematic (joint angles), kinetic (powers and moments) and the spatiotemporal parameters of gait (see below) were measured during level walking using the Cartesian Optoelectronic Dynamic Anthropometer (CODA mpx30) motion analysis system (Charnwood Dynamics, Barrow on Soar, Leicestershire, UK). Two CODA mpx30 systems that were integrated with two AMTI forceplates (Advanced Mechanical Technology Inc, Watertown, MA, USA) were used for data acquisition.

CODA mpx30 is a three-dimensional precalibrated system of three optical sensors mounted on a rigid frame within a scanner unit. The scanner captures infrared light signals pulsed sequentially by markers placed on anatomical landmarks in the lower limbs and on a pelvic frame and thigh and shin wands (as described below). The kinetic data

Table 4 The mean (SD) of the kinematic and kinetic gait parameters before and after isokinetic stretch[a]

Parameter	Baseline		Post stretch		24 h later	
	Controls	Patients	Controls	Patients	Controls	Patients
Hip angle						
IC	28.5 (2.2)	28.6 (9.0)	32.6 (5.2)	28.7 (7.9)	30.0 (4.5)	28.7 (7.2)
MST	5.5 (4.4)	12.5 (10.0)	8.6 (7.2)	12.3 (8.4)	7.6 (4.1)	13.8 (9.4)
MSW	25.9 (3.1)	28.3 (11.8)	31.8 (7.5)	29.0 (7.4)	28.3 (4.5)	29.8 (7.0)
Knee angle						
IC	7.2 (2.3)	17.0 (5.5)	10.6 (5.0)	17.5 (6.7)	9.2 (2.7)	18.5 (6.2)
MST	10.1 (4.1)	10.8 (10.1)	12.8 (6.2)	11.4 (11.3)	12.3 (2.6)	12.3 (10.9)
MSW	47.3 (3.8)	31.1 (13.3)	52.2 (6.5)	33.2 (9.4)	49.8 (1.9)	32.0 (10.4)
Ankle angle						
IC	2.0 (3.3)	0.7 (7.3)	3.2 (1.2)	0.6 (6.8)	2.9 (2.0)	1.0 (6.7)
MST	11.4 (2.1)	8.9 (5.8)	12.6 (2.7)	9.5 (6.1)	12.7 (2.5)	9.6 (4.9)
MSW	8.5 (3.3)	3.4 (9.7)	7.9 (2.4)	3.1 (7.5)	8.6 (2.8)	3.4 (7.4)
Hip power						
IC	−0.04 (0.0)	−0.07 (0.2)	−0.002 (0.0)	−0.008 (0.0)	−0.012 (0.0)	−0.04 (0.1)
MST	0.01 (0.1)	0.05 (0.1)	0.12 (0.26)	0.08 (0.2)	0.11 (0.1)	0.08 (0.1)
MSW	0.02 (0.0)	0.02 (0.0)	0.03 (0.0)	0.02 (0.0)	0.03 (0.0)	0.04 (0.0)
Knee power						
IC	0.19 (0.1)	−0.009 (0.1)	0.24 (0.2)	−0.007 (0.0)	0.18 (0.1)	0.003 (0.0)
MST	−0.07 (0.0)	0.02 (0.0)	−0.29 (0.06)	0.09 (0.1)	−0.01 (0.0)	0.08 (0.2)
MSW	−0.19 (0.0)	−0.09 (0.1)	−0.20 (0.0)	−0.09 (0.0)	−0.18 (0.0)	−0.08 (0.1)
Ankle power						
IC	−0.01 (0.0)	−0.02 (0.0)	−0.004 (0.0)	−0.02 (0.0)	−0.01 (0.0)	−0.03 (0.08)
MST	−0.3 (0.2)	−0.07 (0.1)	−0.26 (0.2)	−0.06 (0.1)	−0.28 (0.2)	−0.08 (0.1)
MSW	0.0009 (0.0)	0.0004 (0.0)	0.007 (0.0)	−0.0004 (0.0)	0.0037 (0.0)	0.001 (0.0)
Hip moment						
IC	0.13 (0.1)	−0.07 (0.1)	0.16 (0.1)	−0.0009 (0.1)	0.11 (0.1)	−0.04 (0.1)
MST	0.04 (0.2)	0.07 (0.1)	0.08 (0.1)	0.10 (0.1)	0.11 (0.1)	0.11 (0.1)
MSW	−0.03 (0.0)	−0.04 (0.0)	−0.03 (0.0)	−0.05 (0.0)	−0.04 (0.0)	−0.06 (0.0)
Knee moment						
IC	−0.09 (0.0)	−0.02 (0.0)	−0.09 (0.0)	0.004 (0.2)	−0.09 (0.0)	−0.02 (0.0)
MST	−0.08 (0.0)	0.009 (0.2)	−0.02 (0.1)	0.09 (0.2)	−0.002 (0.1)	0.09 (0.3)
MSW	−0.04 (0.0)	−0.07 (0.1)	−0.05 (0.0)	−0.05 (0.0)	−0.04 (0.0)	−0.04 (0.0)
Ankle moment						
IC	−0.007 (0.0)	−0.01 (0.0)	−0.008 (0.0)	−0.009 (0.0)	−0.009 (0.0)	−0.008 (0.0)
MST	0.8 (0.1)	0.42 (0.3)	0.81 (0.1)	0.34 (0.2)	0.76 (0.1)	0.41 (0.3)
MSW	−0.008 (0.0)	0.05 (0.2)	−0.009 (0.0)	−0.006 (0.0)	−0.01 (0.0)	−0.007 (0.0)

IC, initial contact; MST, mid-stance; MSW, mid-swing phase of the gait cycle.
[a]Mixed between–within ANOVA after Bonferroni adjustment, p = NS for all values.

were computed from foot–ground forceplate measurements using a method of link-segmental analysis and inverse dynamics.[9] This mathematical model predicts the joint's centre from data recorded in two co-ordinates and anthropometric measurements, namely the patient's weight, height, width of the ankle and knee joints and the width and depth of the pelvis. Powers were defined as the product of the joint moment of force and the joint angular velocity.

The patient's height was measured with a metal ruler mounted on a wall scale and the body weight was measured using a digital scale. The width of the ankle and knee joints was measured with dial calipers. A metal ruler was used to measure front pelvic frame width, and side pelvic frame depth. An alignment frame was used to define the axis of rotation of the ankle joint.

An anatomical co-ordinate was defined with a set of infrared light-emitting diode markers. Three

Table 5 The mean (SD) of the kinematic and kinetic gait parameters before and after isotonic stretch[a]

Parameter	Baseline		Post stretch		24 h later	
	Controls	Patients	Controls	Patients	Controls	Patients
Hip angle						
IC	30.0 (6.0)	23.7 (4.7)	32.0 (6.1)	24.9 (6.7)	31.2 (5.1)	22.4 (9.1)
MST	6.5 (5.1)	9.6 (7.0)	7.6 (3.2)	10.9 (6.8)	7.2 (4.5)	15.7 (18.7)
MSW	26.2 (4.9)	23.9 (8.2)	27.4 (3.8)	25.4 (8.1)	27.7 (3.1)	24.8 (11.8)
Knee angle						
IC	7.0 (3.1)	15.8 (5.3)	7.8 (4.4)	14.5 (4.4)	8.0 (3.9)	14.4 (9.1)
MST	12.6 (3.6)	10.4 (9.8)	13.4 (2.8)	9.7 (8.1)	12.8 (3.6)	13.0 (9.7)
MSW	51.9 (4.9)	26.5 (11.2)	53.0 (2.8)	27.1 (9.6)	53.1 (4.2)	29.8 (13.5)
Ankle angle						
IC	3.9 (3.5)	−2.5 (8.4)	3.3 (3.6)	−2.6 (8.5)	4.9 (2.6)	−3.2 (8.0)
MST	11.9 (1.6)	9.9 (6.4)	9.1 (8.6)	8.7 (5.2)	11.6 (1.2)	9.7 (5.4)
MSW	6.2 (3.0)	0.9 (9.4)	4.9 (4.2)	0.9 (9.2)	5.9 (2.1)	−0.28 (8.9)
Hip power						
IC	−0.03 (0.1)	0.009 (0.1)	−0.02 (0.1)	−0.003 (0.0)	−0.01 (0.1)	0.03 (0.1)
MST	−0.06 (0.2)	0.04 (0.0)	−0.06 (0.2)	0.04 (0.0)	−0.03 (0.4)	0.04 (0.1)
MSW	0.02 (0.0)	0.04 (0.0)	0.03 (0.0)	0.02 (0.0)	0.02 (0.0)	0.06 (0.1)
Knee power						
IC	0.4 (0.2)	−0.02 (0.07)	0.36 (0.2)	−0.01 (0.03)	0.33 (0.1)	−0.006 (0.1)
MST	−0.01 (0.1)	−0.06 (0.1)	0.02 (0.1)	−0.01 (0.1)	−0.04 (0.1)	0.01 (0.1)
MSW	−0.17 (0.0)	−0.07 (0.0)	−0.14 (0.0)	−0.06 (0.0)	−0.20 (0.0)	−0.06 (0.0)
Ankle power						
IC	−0.01 (0.008)	−0.009 (0.0)	−0.008 (0.0)	−0.01 (0.0)	−0.01 (0.0)	−0.01 (0.02)
MST	−0.34 (0.2)	−0.05 (0.1)	−0.38 (0.2)	−0.07 (0.1)	−0.34 (0.1)	−0.07 (0.1)
MSW	0.003 (0.0)	−0.005 (0.0)	0.003 (0.0)	−0.002 (0.0)	0.005 (0.0)	−0.0008 (0.0)
Hip moment						
IC	0.24 (0.1)	0.01 (0.1)	0.17 (0.1)	−0.01 (0.1)	0.15 (0.1)	0.004 (0.1)
MST	−0.02 (0.1)	0.10 (0.2)	−0.01 (0.1)	0.09 (0.2)	0.01 (0.2)	−0.0001 (0.1)
MSW	0.02 (0.01)	−0.03 (0.0)	−0.02 (0.0)	−0.03 (0.0)	−0.01 (0.0)	−0.06 (0.1)
Knee moment						
IC	−0.13 (0.0)	−0.03 (0.0)	−0.11 (0.0)	−0.03 (0.0)	−0.10 (0.0)	−0.02 (0.0)
MST	−0.02 (0.1)	0.03 (0.3)	0.01 (0.1)	0.02 (0.2)	−0.03 (0.1)	0.10 (0.2)
MSW	−0.04 (0.0)	−0.04 (0.0)	−0.03 (0.0)	−0.04 (0.0)	−0.04 (0.0)	−0.03 (0.0)
Ankle moment						
IC	−0.009 (0.0)	0.001 (0.0)	−0.01 (0.0)	−0.0002 (0.01)	−0.01 (0.0)	0.003 (0.0)
MST	0.83 (0.1)	0.33 (0.3)	0.53 (0.6)	0.32 (0.3)	0.81 (0.1)	0.33 (0.3)
MSW	−0.01 (0.0)	−0.006 (0.0)	−0.01 (0.0)	−0.007 (0.0)	−0.01 (0.0)	−0.006 (0.0)

IC, initial contact; MST, mid-stance; MSW, mid-swing phase of the gait cycle.
[a]Mixed between–within ANOVA after Bonferroni adjustment, $p = \text{NS}$ for all values.

markers were placed on the pelvic frame in order to track pelvic movement. The pelvic frame was strapped over the anterior and posterior superior iliac spine. Further markers were positioned over the lateral epicondyle of the femur, the lateral malleolus, and the end of the heel and the fifth metatarsal bone. The second co-ordinate was used to establish the orientation of the femur and tibia. With the subject sitting and both feet together the thigh and shank wands were fastened with Velcro straps above and below the knee, respectively. The thigh wand was parallel to a T-bar placed between the knees. Alignment of the shank wand was made with the subject sitting. The wand was adjusted parallel to the axis of the ankle joint (i.e., perpendicular to the line joining the medial and lateral malleolus). Markers were placed near each of the anterior and posterior ends of the thigh and shank wands.

The following gait parameters were studied: walking speed, duration of stance, the angle of the hip, knee and ankle joint in mid-stance and

Table 6 The mean (SD) of the kinematic and kinetic gait parameters before and after isotonic stretch and weight-bearing[a]

Parameter	Baseline		Post stretch		24 h later	
	Controls	Patients	Controls	Patients	Controls	Patients
Hip angle						
IC	34.0 (6.5)	24.4 (6.8)	31.5 (5.4)	28.7 (7.1)	32.9 (4.9)	29.7 (13.7)
MST	11.7 (6.8)	11.3 (9.8)	8.6 (5.1)	12.4 (9.3)	10.8 (5.5)	12.8 (9.1)
MSW	30.8 (5.8)	26.0 (7.8)	29.1 (4.9)	28.5 (9.5)	30.9 (3.5)	28.0 (9.8)
Knee angle						
IC	10.7 (2.1)	13.6 (4.9)	11.3 (3.1)	13.8 (5.6)	10.2 (3.1)	14.3 (5.4)
MST	16.0 (2.8)	10.7 (9.5)	15.4 (3.2)	9.4 (10.4)	15.0 (2.9)	10.1 (9.9)
MSW	51.7 (6.9)	30.8 (11.4)	53.6 (7.2)	35.0 (13.2)	53.4 (3.0)	33.2 (14.5)
Ankle angle						
IC	4.8 (2.7)	−3.2 (9.4)	5.9 (3.5)	−4.7 (10.5)	4.6 (2.3)	−3.0 (8.2)
MST	13.0 (2.0)	9.1 (5.1)	13.1 (1.3)	8.2 (5.2)	12.8 (2.1)	8.5 (5.6)
MSW	7.6 (2.9)	1.0 (9.0)	8.0 (3.0)	−0.7 (9.5)	7.0 (3.1)	−0.02 (8.6)
Hip power						
IC	0.04 (0.0)	−0.007 (0.1)	−0.02 (0.0)	0.02 (0.1)	0.04 (0.0)	0.03 (0.1)
MST	0.06 (0.3)	0.002 (0.1)	0.06 (0.2)	0.04 (0.1)	0.02 (0.2)	0.12 (0.5)
MSW	0.01 (0.0)	0.01 (0.0)	0.04 (0.0)	0.04 (0.0)	0.05 (0.0)	0.01 (0.0)
Knee power						
IC	0.17 (0.0)	0.01 (0.0)	0.18 (0.3)	0.04 (0.1)	0.22 (0.1)	0.03 (0.0)
MST	0.06 (0.1)	0.03 (0.1)	0.06 (0.1)	0.03 (0.1)	0.06 (0.1)	0.04 (0.1)
MSW	−0.23 (0.0)	−0.08 (0.0)	−0.14 (0.0)	−0.09 (0.0)	−0.15 (0.0)	−0.09 (0.0)
Ankle power						
IC	−0.02 (0.01)	−0.01 (0.0)	−0.0004 (0.0)	−0.01 (0.0)	−0.01 (0.0)	−0.02 (0.0)
MST	−0.21 (0.1)	−0.13 (0.1)	−0.29 (0.3)	−0.14 (0.1)	−0.42 (0.3)	−0.18 (0.3)
MSW	0.004 (0.0)	−0.001 (0.0)	0.004 (0.0)	0.001 (0.0)	0.006 (0.0)	0.001 (0.0)
Hip moment						
IC	0.16 (0.0)	0.04 (0.1)	0.10 (0.2)	0.06 (0.1)	0.12 (0.0)	0.04 (0.1)
MST	0.05 (0.1)	0.04 (0.1)	0.02 (0.1)	0.77 (0.1)	−0.01 (0.1)	0.10 (0.2)
MSW	−0.008 (0.0)	−0.03 (0.0)	−0.02 (0.0)	−0.05 (0.1)	−0.04 (0.0)	−0.04 (0.0)
Knee moment						
IC	−0.11 (0.0)	−0.04 (0.0)	−0.06 (0.1)	−0.05 (0.0)	−0.09 (0.0)	−0.03 (0.0)
MST	0.04 (0.1)	0.12 (0.2)	0.06 (0.1)	0.27 (0.2)	0.07 (0.1)	0.07 (0.2)
MSW	−0.05 (0.02)	−0.04 (0.02)	−0.03 (0.0)	−0.04 (0.0)	−0.03 (0.0)	−0.04 (0.0)
Ankle moment						
IC	−0.01 (0.0)	0.001 (0.0)	−0.009 (0.0)	−0.0007 (0.0)	−0.01 (0.0)	0.0008 (0.0)
MST	0.67 (0.3)	0.40 (0.3)	0.75 (0.1)	0.38 (0.3)	0.74 (0.1)	0.49 (0.3)
MSW	−0.01 (0.0)	−0.007 (0.0)	−0.008 (0.0)	−0.006 (0.0)	−0.009 (0.0)	−0.006 (0.0)

IC, initial contact; MST, mid-stance; MSW, mid-swing phase of the gait cycle.
[a]Mixed between–within ANOVA after Bonferroni adjustment, p = NS for all values.

mid-swing and the moments and powers generated at these joints. These gait parameters were studied because of their importance for safe and energy-efficient walking.[10]

The phases of the gait cycle were determined from inspection of the animated 'stick diagram' and confirmed by the position of the ground force vector relative to the foot. Although mid-stance and mid-swing are not discrete points in time, they were defined in this study (for simplicity) as 50% of the respective phase of the gait cycle. Only the data in the sagittal plane were analysed. The kinetic and kinematic data were acquired and digitized with a sampling frequency of 200 Hz. Data synchronization and normalization of moments and powers to body weight and height were made automatically by the CODA mpx30 software.

The gait laboratory was 15 m long and 4 m wide and the forceplates were embedded in the middle of the walkway. Subjects were asked to walk,

Table 7 The mean (SD) of the spatio-temporal gait parameters for patients and healthy control subjects[a]

	Baseline		Post test 1		Post test 2	
	Controls ($n = 7$) Mean (SD)	Patients ($n = 22$) Mean (SD)	Controls ($n = 7$) Mean (SD)	Patients ($n = 22$) Mean (SD)	Controls ($n = 7$) Mean (SD)	Patients ($n = 22$) Mean (SD)
Duration stance						
Isokinetic	0.75 (1.22)	1.18 (0.81)	0.74 (0.14)	1.21 (1.10)	0.73 (0.10)	1.08 (0.50)
IS-NWB	0.64 (0.04)	1.20 (0.49)	0.63 (0.08)	1.14 (0.41)	0.64 (0.03)	1.18 (0.46)
IS-WB	0.67 (0.07)	0.99 (0.30)	0.66 (0.08)	0.94 (0.34)	0.64 (0.04)	0.93 (0.24)
Duration of swing						
Isokinetic	0.45 (0.07)	0.55 (0.16)	0.44 (0.08)	0.55 (0.09)	0.44 (0.06)	0.57 (0.18)
IS-NWB	0.41 (0.03)	0.51 (0.14)	0.41 (0.03)	0.51 (0.09)	0.41 (0.10)	0.48 (0.09)
IS-WB	0.39 (0.02)	0.50 (0.09)	0.40 (0.05)	0.50 (0.08)	0.39 (0.03)	0.50 (0.11)
Walking speed						
Isokinetic	1.01 (0.15)	0.52 (0.26)	1.05 (0.19)	0.54 (0.24)	1.04 (0.11)	0.52 (0.26)
IS-NWB	1.27 (0.11)	0.41 (0.23	1.31 (0.10)	0.42 (0.21)	1.29 (0.10)	0.44 (0.24)
IS-WB	1.11 (0.22)	0.52 (0.30)	1.16 (0.19)	0.59 (0.30)	1.17 (0.12)	0.59 (0.30)

IS-NWB, isotonic stretch without weight-bearing; IS-WB, isotonic stretch with weight-bearing.
[a]Mixed between–within ANOVA after Bonferroni adjustment, p = NS for all values.

without shoes, the length of the room at their usual speed. The gait of each subject was recorded for three gait cycles and the most representative cycle was saved for future analysis. The use of a walking aid was not permitted.

Data analysis

The data were analysed using the SPSS version 9.0 for windows. Comparability of the groups at baseline and after the interventions was made using a one-way between-groups analysis of variance (ANOVA) and mixed between–within ANOVA, respectively. A p-value of 0.05 was accepted as the level of statistical significance. Bonferroni adjustments were applied to any significant results in order to reduce the risk of type 1 error due to analysis of multiple dependent variables. The adjustments were made by dividing the alpha level (0.05) by the number of comparisons made, as previously recommended.[11]

Results

A total of 66 consecutive stroke patients who fulfilled the study entry criteria and 21 age-matched healthy control subjects were recruited. All completed the study. As shown in Table 2, the demographic and clinical characteristics of patients and control subjects were comparable, except that patients who received isokinetic stretch had a slightly longer duration since stroke and a higher mean MAS score. The mean (SD) of the kinematic, kinetic and spatio-temporal gait parameter at baseline, immediately after muscle stretch and 24 h later are given in Tables 3–7.

After Bonferroni adjustment (alpha level = 0.01) there were statistically significant differences between patients and control subjects in the studied kinematic and kinetic variables of the ankle, knee and hip at initial contact (IC) and mid-swing (MSW). Ankle dorsiflexion was significantly less at IC and MSW in patients compared with the control groups. In MSW ankle dorsiflexion was also reduced in patients but knee flexion was greater than in healthy subjects. Knee and hip moments and knee power at IC and ankle moments and power in mid-stance (MST) and walking speed were lower in patients than in controls. The differences between the patient and control groups were statistically significant. There were no statistically significant differences between the patient groups in any of the studied kinematic, kinetic and spatio-temporal gait parameters, neither immediately after muscle stretch or 24 h later. Similarly, there were no significant differ-

ences between the three measurements over time for each type of muscle stretch.

Discussion

The findings of the present study suggest that a single session of isokinetic or isotonic muscle stretch (with or without weight-bearing) of the ankle plantar flexors has no significant impact on the studied kinematic, kinetic and spatio-temporal gait parameters in ambulatory patients with residual hemiplegia. These observations are in agreement with those of Bressel and McNair[12] who reported that a 30-min stretch of the ankle plantar flexors reduced the stiffness at the ankle joint but had no effect on gait.

The lack of a demonstrable change in the subjects' gait after muscle stretch was unlikely to be due to poor sensitivity of the outcome measures used in this study. Ankle joint displacement and knee joint kinematics during walking and angular velocity are reliable indicators of walking performance in hemiplegic stroke patients with mild residual motor impairment.[13] A poor correlation between the reduction of muscle hypertonus and improvement in motor function has been demonstrated after treatment of muscle spasticity with pharmacological agents. For example, treatment of ambulatory patients with spastic paraparesis with baclofen resulted in a significant inhibition of the spinal stretch reflex but had no effect on the kinematic gait parameters or walking speed.[6] The same may be true after muscle stretch. A likely explanation of our findings is that a single session of muscle stretch was not sufficient to cause lasting functional change.

It is thought that muscle stretch reduces hypertonia by two mechanisms. It inhibits the stretch reflex and also reduces thixotropy and the muscle stiffness due to changes in the visco-elastic properties of the spastic muscle.[14] The effect of stretch on the visco-elastic properties of muscle would require repeated sessions of muscle stretch over an extended period. Consequently, a single treatment session may not improve motor function despite its immediate effect on muscle tone.

Clinical messages

- A single 20-min session of isokinetic or isotonic muscle stretch (with or without weight-bearing) has no demonstrable effect on the gait of ambulatory stroke patients with spastic hemiparesis.
- A course of muscle stretch consisting of repeated treatment sessions over several days may be required to effect improvement in a patient's spastic gait.

Plastic changes in the central nervous system (CNS) develop after repeated training.[15] It is, therefore, plausible that a single session of muscle stretch has little or no effect on the functional plasticity of the neural circuits of the spinal cord. Functional plasticity is required to enable adaptation to the demands of any motor task. For instance, spinal segmental reflexes are modulated during gait and in healthy subjects facilitation of the soleus H-reflex was shown to occur at the end of stance. The same reflex is inhibited in the swing phase of the gait cycle.[16] The ability to modulate the stretch reflex is probably more important for motor function than changes in the resting muscle tone. This may be another explanation for the findings of the present study.

In conclusion, the present study showed that a single session of isokinetic or isotonic muscle stretch (with or without weight-bearing) did not result in a significant improvement in motor function. The main limitation of the present study is that it examined the effect of a single session of muscle stretch, rather than a longer course of treatment. It would appear that repeated treatments are required to effect change in motor function and this issue should be addressed in future research.

References

1 Richards CL, Malouin F, Dumas F. Effects of a single session of prolonged plantar flexor stretch on muscle activation during gait in spastic cerebral palsy. *Scand J Rehabil Med* 1991; **23**: 103–11.

2 Lamontagne A, Malouin F, Richards CL. Contribution of passive stiffness to ankle plantarflexor moment during gait after stroke. *Arch Phys Med Rehabil* 2000; **81**: 351–58.
3 Massin M, Allington N. Role of exercise testing in the functional assessment of cerebral palsy children after botulinum A toxin injection. *J Pediatr Orthop* 1999; **19**: 362–65.
4 Lamontagne A, Malouin F, Richards CL, Dumas F. Evaluation of H-reflex and non-reflex-induced muscle resistance to stretch in adults with spinal cord injury using hand-held and isokinetic dynamometry. *Phys Ther* 1998; **78**: 964–78.
5 Pinniger GL, Nordlund M, Steele JR, Cresswell AG. H-reflex modulation during passive lengthening and shortening of the human triceps surae. *J Physiol* 2001; **534**: 913–23.
6 Nuyens GE, De Weerdt WJ, Spaepen AJ Jr, Kiekens C, Feys HM. Reduction of spastic hypertonia during repeated passive knee movements in stroke patients. *Arch Phys Med Rehabil* 2002; **83**: 930–35.
7 Bohannon RW, Smith MB. Interrater reliability of modified Ashworth scale of muscle spasticity. *Phys Ther* 1987; **67**: 206–207.
8 Nielsen J, Petersen N, Ballegaard M, Biering-Sorensen F, Kiehn O. H-reflexes are less depressed following muscle stretch in spastic spinal cord injured patients than in healthy subjects. *Exp Brain Res* 1993; **97**: 173–76.
9 Winter DA. *The biomechanics and motor control of human movement,* second edition. New York: Wiley, 1990.
10 Perry J. *Gait analysis: normal and pathological gait*. Thorofare, NJ: Slack Inc, 1992.
11 Everitt BS. *The Cambridge dictionary of statistics*. Cambridge: Cambridge University Press, 1998.
12 Bressel E, McNair PJ. The effect of prolonged static and cyclic stretching on ankle joint stiffness, torque relaxation, and gait in people with stroke. *Phys Ther* 2002; **82**: 880–87.
13 Richards JD, Pramanik A, Sykes L, Pomeroy VW. A comparison of knee kinematic characteristics of stroke patients and age-matched healthy volunteers. *Clin Rehabil* 2003; **17**: 565–71.
14 Carey JR. Manual stretch: effect on finger movement control and force control in stroke subjects with spastic extrinsic finger flexor muscles. *Arch Phys Med Rehabil* 1990; **71**: 888–994.
15 Trimble MH, Koceja DM. Modulation of the triceps surae H-reflex with training. *J Neurosci* 1994; **76**: 293–303.
16 Yang JF, Stein RB, James KB. Contribution of peripheral afferents to the activation of the soleus muscle during walking in humans. *Exp Brain Res* 1991; **87**: 679–87.