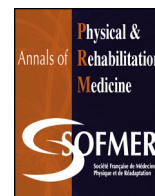




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Original article

Superficial warming and cooling of the leg affects walking speed and neuromuscular impairments in people with spastic paraparesis

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ABSTRACT

Background: People with hereditary and spontaneous spastic paraparesis (HSSP) report that their legs are stiffer and walking is slower when their legs are cold.

Objectives: This study explored the effects of prolonged superficial cooling and warming of the lower leg on walking speed and local measures of neuromuscular impairments.

Methods: This was a randomised pre- and post-intervention study of 22 HSSP participants and 19 matched healthy controls. On 2 separate occasions, one lower leg was cooled or warmed. Measurements included walking speed and measures of lower limb impairment: ankle movement, passive muscle stiffness, spasticity (stretch reflex size), amplitude and rate of force generation in dorsi- and plantarflexors and central and peripheral nerve conduction time/velocity.

Results: For both participants and controls, cooling decreased walking speed, especially for HSSP participants. For both groups, cooling decreased the dorsiflexor rate and amplitude of force generation and peripheral nerve conduction velocity and increased spasticity. Warming increased dorsiflexor rate of force generation and nerve conduction velocity and decreased spasticity.

Conclusions: Superficial cooling significantly reduced walking speed for people with HSSP. Temperature changes were associated with changes in neuromuscular impairments for both people with spastic paraparesis and controls. This study does not support the use of localised cooling in rehabilitation for people with spastic paraparesis as reported in other neurological conditions. Rehabilitation interventions that help prevent heat loss (insulation) or improve limb temperature via passive or active means, particularly when the legs and/or environment are cool, may benefit people with spastic paraparesis.

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1. Introduction

Hereditary and spontaneous spastic paraparesis (HSSP) is a progressive condition resulting in impaired balance and walking [1]. In type I or uncomplicated HSSP, people present lower limb paresis and spasticity because of axonal degeneration of central descending and ascending tracts including the corticospinal tract, spinocerebellar tracts and dorsal columns. In type II or complicated HSSP, additional signs include peripheral neuropathy, cerebellar ataxia or dementia [1]. Focus groups with people with HSSP in the United Kingdom ($n = 36$) highlighted the perception that their walking is often slower when their legs are cold, such as in cold

weather; this is associated with an increase in perceived lower limb stiffness. Warming the lower legs by increasing layers of clothes or being in warmer environments is perceived to help with walking faster and relieve increased leg stiffness.

People with stroke or acquired brain injury show decreased spasticity, measured clinically and electrophysiologically, with periods of superficial cooling [2–4]. Clinically localised cooling or cryotherapy to reduce spasticity is proposed for a range of neurological conditions [4,5]. Conversely, reduced spasticity has been reported with localised and global warming [6–8]. Despite the reduced spasticity, improvements in voluntary movements and function have not been clearly demonstrated [2,3], which may reflect the associated impact of temperature changes on nerve conduction velocity, passive stiffness [9] and muscle strength.

The subjective report of improved function with warming in people with HSSP contrasts with those with multiple sclerosis, which can also be associated with upper motor-neuron syndrome. People with multiple sclerosis often report a worsening of

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symptoms with warming and improvement with whole body or localised cooling. This situation is mainly considered to be mediated by inducing a central nerve conduction block with warming (Uthoffs phenomenon) secondary to demyelination [10,11]. Therefore, central conduction time should be assessed in HSSP.

We investigated whether:

- people with HSSP experience changes in walking speed and measures of neuromuscular impairments (movement, stiffness, strength and nerve conduction velocity) with prolonged superficial cooling and warming;
- whether these changes are comparable to that seen in healthy people. Ultimately, we aimed to determine whether rehabilitation strategies should consider the functional impact of temperature changes in people with HSSP.

2. Materials and methods

2.1. Participants

We included 22 HSSP participants and 19 healthy controls matched on age, gender and body mass index (BMI) (Table 1). HSSP participants were recruited by advertisements in the UK SP support group newsletter and controls by local advertisements. HSSP participants were included if they had a diagnosis of spastic paraparesis with or without a family history. Those with other differential diagnoses were excluded by appropriate imaging, clinical and laboratory tests. Participants had to be able to walk at least 20 m with or without a walking aid and have bilateral spasticity in the ankle plantarflexors (at least grade 1 Ashworth score [12]). We excluded HSSP participants if they had additional orthopaedic or neurological impairments. Exclusion factors for both groups included contraindications to transcranial magnetic stimulation (TMS), poor skin integrity, Raynaud's disease or a fixed ankle inversion contracture. Ethical approval for the study was provided by South West Cornwall and Plymouth Ethics Committee (HS13/14-105). Informed consent was provided by all participants.

Table 1
Clinical characteristics of participants with hereditary and spontaneous spastic paraparesis (SSP).

Participant	Age/ gender	Family history (genetic diagnosis)	Symptom duration (year)	Barthel Index	Abbr Mental Test Score	Peripheral neuropathy	Ashworth Ankle Plantar flexors (most affected side)	Anti-spasticity medication	Additional signs and symptoms	Pure or complicated
1	64F	No	24	85	10	No	1	No	No	Pure
2	19F	Yes (SPG4)	17	95	8	No	1	Oral baclofen	No	Pure
3	81M	Yes	41	25	9	Yes	3	Diazepam	Cerebellar ataxia, blurred vision	Complicated
4	35F	No (SPG4)		95	9	No	1	No	No	Pure
5	68M	No	37	85	9	Yes	1	No	No	Complicated
6	67M	Yes	62	90	10	No	2	No	No	Pure
7	55F	No	9	95		No	1	Oral baclofen	No	Pure
8	55M	No	11	50	9	Yes	1	Oral baclofen	No	Complicated
9	55F	Yes	5	95	10	No	1	No	No	Pure
10	54M	Yes	10	65	7	Yes	2	Tizanadine	No	Pure
11	69M	Yes	6	100	10	No	1	No	No	Pure
12	63F	Yes (SPG10)	12	90	10	No	1	No	No	Pure
13	64M	Yes	14	95	9	No	2	Botulinum toxin to plantarflexors	No	Pure
14	65M	Yes	15	90	9	No	3	No	No	Pure
15	48F	Yes (SPG4)	11	60	6	No	3	No	No	Pure
16	34M	Yes (SPG4)	2	100	10	No	1	No	No	Pure
17	54F	Yes (SPG3a)	51	95	6	No	1	No	No	Pure
18	48M	Yes (SPG4)	12	95	9	No	2	No	Retinopathy	Complicated
19	50F	Yes	19	95	10	No	2	No	No	Pure
20	60M	Yes	18	90	10	No	1	No	Retinopathy	Complicated
21	46F	No	11	100	9	No	2	Oral baclofen	No	Pure
22	56F	Yes (SPG4)	10	95	10	No	1	No	No	Pure
Control	48.2 ± 10.4	NA	NA	100 (0)	10 (0)	NA	0 (0)	NA	NA	NA

NA: not available; Data are mean ± SD for controls.

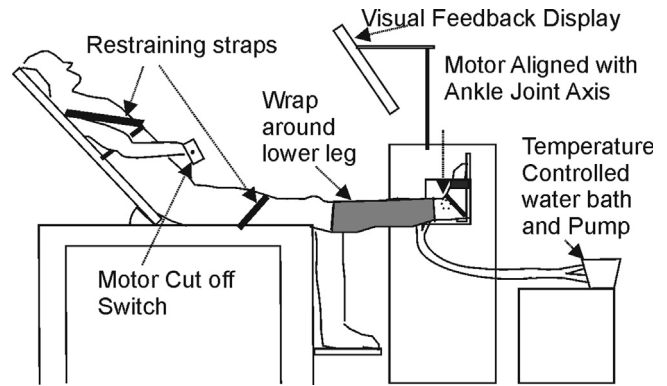


Fig. 1. Experimental set-up.

Participants' baseline characteristics (height, weight, age, sex, family history, genetic diagnosis, length of symptoms and presence of anti-spasticity medication) were recorded. The Abbreviated Mental Test Score was used to screen for dementia and the self-reported Barthel Index was used for functional ability. Skin-fold thickness overlying the ankle plantar flexors was measured by using a Harpenden calliper at the level of the mid-shank with the participant in a seated position and BMI was calculated from height and weight. The Ashworth scale was used to evaluate spasticity in the lower leg. HSSP was classified as pure or complicated by genetic diagnosis and the presence or absence of additional signs and symptoms, including peripheral neuropathy [13,14].

2.2. Intervention

For HSSP participants, the self-reported most affected side was studied, and for healthy controls a similar proportion of dominant and non-dominant legs was assessed. Participants were assessed in a semi-reclined standardised position (Fig. 1). One lower leg was cooled or warmed for 30 min by using a wrap attached to a

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