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Does calf muscle spasticity contribute to postural imbalance? A study in persons with pure hereditary spastic paraparesis

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ABSTRACT

Objectives: The contribution of spasticity to postural imbalance in patients with upper motor neuron syndrome is still unclear. This study aimed to evaluate the responses to support-surface perturbations in patients with hereditary spastic paraparesis (HSP). These patients typically suffer from bilateral spasticity with relatively preserved muscle strength of the lower limbs. Particularly toes-up rotations were expected to be destabilizing due to insufficient suppression of calf muscle stretch reflexes.
Methods: Participants were seventeen symptomatic community-dwelling patients with autosomal dominant pure HSP and seventeen healthy controls. All patients had increased muscle tone of the triceps surae (TS) but no muscle contractures. Perturbations were applied by rotating or translating a platform with increasing intensity in four sagittal-plane directions. The primary outcome was maximum intensity ('limit of stability') sustained without stepping or grabbing in each type of perturbation. Leg muscle tone and strength were assessed with the Modified Ashworth Scale and Medical Research Council (MRC) scale, respectively.
Results: For toes-up perturbations, limits of stability in patients were substantially lower than in controls, which were related to TS muscle tone but not to tibialis anterior (TA) strength. Toes-down rotations were indiscriminative. For backward perturbations, patients also had lower limits of stability, unrelated to TA strength or TS muscle tone. In forward perturbations, patients with TS strength MRC 4 were less stable than patients with normal TS strength and controls.
Conclusion: Calf muscle spasticity and weakness differently contribute to postural imbalance in patients with HSP. This notion could have implications for the clinical management of spasticity.

1. Introduction

Lower limb spasticity has been suggested to contribute to postural imbalance in various types of patients with upper motor neuron syndrome such as stroke and multiple sclerosis [1–3]. However, many of these patients suffer from other sensorimotor deficits such as muscle weakness, loss of motor selectivity and sensory impairment [4], rendering it difficult to estimate the 'unique' contribution of spasticity to balance during functional activities. Patients with hereditary spastic paraparesis (HSP) constitute a better clinical model to evaluate the influence of lower limb spasticity on functional balance. HSP is a neurodegenerative disorder characterized by slowly progressive lower limb spasticity [5] with a prevalence ranging between 0.5 and 12 per 100,000 inhabitants [6]. The main neuropathological feature of HSP is axonal degeneration that is most prominent in the terminal portions of the longest descending and ascending spinal tracts (i.e. crossed and uncrossed corticospinal tracts to the legs, fasciculus gracilis fibers and, to a lesser extent, spinocerebellar fibres), while the neuronal cell bodies of the degenerating fibers are preserved [5]. Although the disease is heterogeneous, both clinically and genetically, it can phenotypically be classified into pure and complicated forms [5,7]. The phenotype of the complicated form includes, in addition to spasticity, symptoms of e.g. dementia and peripheral neuropathy. In contrast, patients with pure HSP mainly experience lower body spasticity with relatively preserved muscle strength, motor selectivity and proprioception of the lower limbs [5,7,8]. Therefore, patients with pure HSP represent a group with

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minimal confounding effects of other sensorimotor deficits in assessing the influence of lower limb spasticity on balance maintenance.

Clinical experience shows that in patients with a pure form of HSP, spasticity generally develops from distal to proximal and is, therefore, often most pronounced in the calf muscles (i.e., triceps surae (TS) muscles). The potentially destabilizing effect of calf muscle spasticity on balance is likely to be greatest during toes-up perturbations (i.e. rotation of the support surface inducing fast ankle dorsiflexion) causing fast muscle stretch. During these perturbations, stretch-induced destabilizing calf muscle responses [9–11] need to be suppressed in order to prevent a plantar flexion moment at the ankles that would pull the center of mass backward. Indeed, extensive research [12–15,11] has shown that in healthy subjects, stretch-induced calf muscle activity during toes-up perturbations is almost completely suppressed [16], while long-latency (120–220 ms) responses in the antagonist (i.e., tibialis anterior (TA)) muscle stabilize the body. In the case of inadequate suppression of calf muscle activity, plantar flexion moments may be large enough to reach the limits of stability, which would result in backward stepping or grabbing for support in order to recover balance. In patients with spastic calf muscles, several electrophysiological studies [17,18] have shown that stretch-induced responses are inadequately suppressed. Yet none of these studies has actually looked at the influence of spasticity on balance maintenance by assessing postural behavior and determining limits of stability. Only one study [18] applied support-surface rotations in 7 patients with spastic paraparesis (including 4 with HSP), and found increased activity of the calf muscles in response to toes-up perturbations indicating exaggerated stretch reflexes. This study, however, did not assess the functional consequences of the observed disinhibition, i.e. whether subjects actually were destabilized to such an extent that it endangered or disrupted balance, possibly leading to a fall.

Therefore, the aim of this study was to assess the contribution of calf muscle spasticity to postural imbalance by comparing the responses to sagittal-plane support-surface perturbations in patients with HSP to those in healthy control subjects. It was hypothesized that particularly the responses to toes-up perturbations would be poor compared to controls and that balance performance would be negatively associated with calf muscle tone. In contrast, no differences were expected in balance performance between patients and controls in response to forward perturbation (i.e. backward translation of the support surface). Even though such perturbations also induce fast muscle stretch, calf muscle activation is usually stabilizing because a concomitant plantar flexion moment pulls the center of mass backward.

2. Methods

2.1. Participants

All patients with a symptomatic pure form of HSP who visited the outpatient clinic of the departments of Neurology and Rehabilitation Medicine at our university hospital during a period of 1 year (September 2009 until July 2010) were eligible. In addition, active recruitment took place through advertisements and oral presentations for the national patient organization (VSN). Inclusion criteria were: (1) having clinical symptoms related to TS spasticity (e.g. muscle cramps, stiffness, pain, clonus), (2) having autosomal dominant pure HSP (either genetically proven or based on family history), and (3) being a community ambulator (Functional Ambulation Categories (FAC) score 5) [19]. Exclusion criteria were (1) TS muscle tone greater than 2 on the Modified Ashworth Scale (MAS) [20], (2) TS and TA muscle strength lower than 4 on the Medical Research Council (MRC) scale [21], (3) passive ankle range of motion (pROM) less than 10° dorsiflexion with an extended knee, (4) motor selectivity lower than stage 5 of the Brunnstrom stages [22], all on either side of the body. In addition, age and gender matched healthy controls were recruited. All subjects gave their written informed consent prior to participation. The study was approved by the regional medical ethics committee and conducted according to the rules laid down in the Declaration of Helsinki [23].

2.2. Clinical assessment

All clinical assessments were performed by the same physiatrist (ACHG). Muscle tone of the TS and TA was assessed using the MAS [20]. The TS was tested with the knee both flexed and extended. In addition, the Achilles tendon reflex was tested to identify reflex hyperexcitability (present/absent). Muscle strength of the TS and TA was assessed with the Medical Research Council (MRC) scale. An MRC grade 5 indicated normal muscle strength and grade 4 indicated that muscle strength was reduced but still sufficient to move the ankle joint against gravity (i.e., to be able to stand on the heels when testing the TA and to stand on the toes when testing the TS) [21].

2.3. Balance assessment

Subjects stood barefoot on a moveable platform with their eyes open, their knees extended, and their feet at shoulder width. To prevent possible falls, they wore a safety harness suspended to the ceiling. The platform imposed four different types of perturbations (Fig. 1). Platform rotations resulted in either dorsiflexion (toes-up perturbation) or plantar flexion (toes-down perturbation) at the ankle joint. Platform translations resulted either in forward perturbation (backward translation) or in backward perturbation (forward translation). Due to the large dimensions of the platform (1.2 m × 1.8 m), it was possible to make a compensatory step without the risk of stepping off the platform (Fig. 2).

As the primary outcome, each subject's limit of stability was determined for the four types of perturbation. The 'limit of stability' was defined as the maximum perturbation intensity that the subject could sustain at least one out of three times without taking a step, grabbing for support or bending the knees. The latter criterion was used because flexion of the knees enhances postural stability by lowering the center of body mass. To determine the maximum intensity, the perturbation intensity was gradually increased using fixed steps. In the rotational perturbations, the platform rotated at a maximum angular velocity of 51 $^{\circ}s^{-1}$ from a horizontal position to an angle ranging from 1° to a maximum of 9° with increments of 0.5°. In the translational perturbations, the platform accelerated for 300 ms from 0.125 m s^{-2} to a maximum of 2.5 m s^{-2} with increments of 0.125 m s^{-2}. Because platform acceleration causes destabilizing torques acting on the body and subsequent deceleration would have a stabilizing influence [24], the acceleration period was followed by a 500 ms period of constant velocity before a period of deceleration was imposed (300 ms).

During the balance assessment, subjects were first exposed to a try-out of each of the four perturbations at the lowest intensity to familiarize them with the experimental situation. After this familiarization, the four types of perturbation were imposed on each subject in a random order. Subjects were instructed to maintain balance, without stepping or grabbing for support and without bending their knees. Furthermore, participants were forewarned by the researcher of the upcoming perturbation at a random time interval of 2–10 s prior to perturbation onset, but its direction and precise timing could not be anticipated. Subjects were allowed a maximum of three consecutive attempts at each