Cerebellar Stroke With Speed-Dependent Gait Ataxia

To the Editor:

We report on a 63-year-old woman with ischemic stroke of the right cerebellar hemisphere. Her symptoms, consisting of acute gait ataxia and left hemiataxia, were resolved in the following weeks, but we detected a speed-dependent improvement in her gait by moving fast. We therefore believe that the hypothesis of an autonomic spinal gait program becomes more evident and could be used in rehabilitation.

It was recently shown that gait training under maximum speed improves gait deficit in stroke patients.¹ Despite the frequency of cerebral ischemic stroke, it is often difficult to answer the question concerning the persistence of an acute neurological deficit. Usually, a good clinical outcome is anticipated if the initial deficit is moderate and/or other parts of the brain seem to be able to compensate functional failure. In cases of gait disturbance, some authors postulate a spinal automatic gait program that could take over control from higher central nervous system regions if thy are destroyed or disturbed.^{2–4} Physical therapy should take this into account, and all available facilities that rely on an improvement in these brain independent programs could improve outcome.

We describe here for the first time a patient with cerebellar strokeinduced gait disturbance who profits from walking fast compared with moving slowly. This 63-year-old woman was admitted to our hospital with acute onset of gait ataxia and a mild right-sided sensory loss for temperature. Neurological examination showed saccadic eye movements, mild hemiataxia in the left arm and left leg, and moderate unsteadiness while standing or walking.

The initial gait analysis, done with the patient supported by a nurse, showed an irregular gait with great variance in heel-to-toe and side-to-side movements as a hint for gait ataxia. The analysis was done with the patient wearing special shoes with 16 contact sensors that measured the time and pressure on 8 different points of both feet while walking for 20 seconds, as published elsewhere.⁵ MRI displayed a cerebellar stroke of the left hemisphere caused by a paradoxical embolism from a patent foramen ovale but without any lesions in the vestibular-associated brain stem (controlled in 2 MRIs with diffusion-weighted imaging on days 1 and 7 after ischemia). Electronystagmography showed hypometric saccadic eye movement without any spontaneous or gaze-related nystagmus consistent with cerebellar

Moving slow

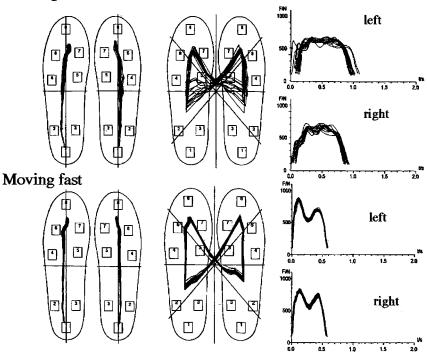
damage, sparing the brain stem and without hints of concomitant vestibular lesions.

After treatment with physiotherapy, symptoms improved during the following weeks, but clinical examination and further gait analysis on day 20 revealed the surprising feature that the patient was much better off walking fast (3-fold increase in speed and 2-fold increase in stride length) than walking slowly (the Figure). A variability of single support lines of 6.07 ± 0.85 moving normally to 1.54 ± 0.21 moving fast (control population, 4.24 ± 1.07^5) was observed.

We suggest an automatic spinal program that takes over control when the patient walks fast, thereby suppressing the misleading false cerebellar inflow. This program interacts with the somatosensory input from the feet and the vestibular-cerebellar-thalamic circuits. The same program could enable paraplegic patients to walk on a moving treadmill while their body weight is suspended. Patients with ischemic lesions of a cerebellar hemisphere also could profit by training fast movements first, as described in the report of Pohl et al.¹

Marc Fatar, MD Hansjoerg Baezner, MD Martin Griebe, MD Mark Stroick, MD Michael Hennerici, MD From the Department of Neurology Klinikum Mannheim University of Heidelberg Heidelberg, Germany

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Computer-based gait analysis showing the difference in slow and fast movements done alternately on the same day.





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