EDITORIAL COMMENTARY

Gait disorders

The paper by Stolze *et al*¹ (this issue, pp 289–297) substantially contributes to an improved diagnosis of often occurring gait disorders. The study compares the kinematics of gait in patients with Parkinson's disease before and after treatment with levodopa and those with normal pressure hydrocephalus before and after CSF tapping, as well as with and without external cues.

Clinically, the movement disorder of the two patient groups is usually difficult to diagnose appropriately. Up to now there existed no direct comparative gait analysis between the two diseases, especially in respect to the effects of treatment. From this study¹ it becomes evident that there are characteristic differences between the two gait disorders.

From the patients' point of view the first indication of a central motor system lesion is an impairment of movement, most notably locomotion. However, there is only a weak relation between the physical symptoms and signs found during clinical examination under passive conditions (for example, tendon tap reflexes and muscle tone) and the altered neuronal mechanisms underlying the impairment during active motion (for review see Dietz²). The differential diagnosis of a movement disorder is an important and everyday clinical problem.³ In some classic examples, as in the case of spasticity only recordings of neuronal and biomechanical signals during movement could lead to an adequate concept of pathophysiological and, consequently, of treatment.⁴ By such methodological approaches also objective measures can be obtained about treatment effects.

As shown in the paper of Stolze *et al*¹ a clear differential diagnosis can also be made without extensive EMG and kinematic recordings. Objective measures were obtained about the differential effects of CSF tapping on balance regulation and motor programming in NPH, which indicates that different systems might be involved in the

respective gait disorder. Key features, such as the broad based gait (normal pressure hydrocephalus) or the influence of external cues on locomotion (more pronounced in Parkinson's disease) can lead to an appropriate diagnosis and treatment in the early stage of the disease.

A critical point of the paper might represent the fact that we do not know whether the stage of the disease was comparable between the group of patients with Parkinson's disease and that of patients with normal pressure hydrocephalus, although the authors have tried to select patients with a comparable severity of disease. However, this problem is not so relevant, as most of the key features were independent of the speed of locomotion, and therefore, presumably also from the stage of disease.

This and earlier studies in this field suggest that movement analysis represents a tool that should be included to the usual examinations for the diagnosis of a movement disorder. Such an analysis might exclusively point to the appropriate diagnosis and, consequently, of treatment. Furthermore, such an analysis best reflects the impairment of movement of the patient and the effect of any drug or physical treatment.

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- 2 Dietz V. Neurophysiology of gait disorders: present and future applications [review]. *Electroencephalogr Clin Neurophysiol* 1997;103:333–55.
- 3 Sudarsky L. *Clinical approach to gait disorders of aging: an overview*. Philadelphia: Lipincott-Raven, 1997.
- 4 Dietz V. Human neuronal control of automatic functional movements: interaction between central programs and afferent input. *Physiol Rev* 1992; 72:33–69.