Vestibular exercises improve central vestibulospinal compensation after vestibular neuritis

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Article abstract—*Objective and Background:* Animal experiments have shown that central vestibular compensation of unilateral peripheral vestibular lesions can be improved by vestibular exercises. There are, however, no equivalent clinical studies on the efficacy of such specific physiotherapy on acute unilateral peripheral vestibular lesions in humans. *Design and Methods:* To quantify the differential effects of specific vestibular exercises on central compensation in patients with an acute/subacute unilateral vestibular lesion (vestibular neuritis), we determined the time course of recovery of 1) the ocular torsion (OT) for the vestibulo-ocular system, 2) the subjective visual vertical (SVV) for perception, and 3) the total sway path (SP) values for postural control in 19 patients with and 20 patients without vestibular exercises. All patients had a persisting peripheral vestibular deficit for at least 30 days (statistical end point). *Results:* Although normalization of OT and SVV was similar in the control and physiotherapy groups, the total SP values on day 30 after symptom onset differed significantly: 3.2 ± 1.9 m/min in the physiotherapy group and 16.9 ± 6.1 m/min in the control group (ANOVA, $p < 0.001$). *Conclusions:* This prospective clinical study suggests that specific vestibular exercises improve vestibulospinal compensation in patients with acute peripheral vestibular lesions.

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Animal experiments in the monkey and the cat have shown that spontaneous central vestibular compensation of unilateral peripheral vestibular lesions can be improved by vestibular exercises.[1,2] There are, however, no equivalent prospective clinical studies on the efficacy of such physiotherapy in humans who originally had a normal vestibular function before onset of an acute peripheral vestibular deficit.[3] Several authors advocate empirically the early use of exercises to treat patients for active readjustment of vestibular dysfunctions,[4,5] but its efficacy is not yet proved.[6]

In this study we used vestibular neuritis as a model for a unilateral peripheral vestibular loss, which causes a distressing tonal imbalance with rotatory vertigo, spontaneous horizontal nystagmus directed away from the affected ear, and falls toward the affected ear.[7,8] Vestibular neuritis was used because 1) patients with this disease have no peripheral vestibular deficit before symptom onset (in contrast to, for instance, patients with acoustic neuroma or Menière's disease); 2) the onset is acute/subacute within minutes to hours and is, therefore, well defined; 3) it is an isolated peripheral vestibular lesion without additional central dysfunction that would inhibit central compensation; and 4) the possible recovery of peripheral vestibular function can be documented by caloric irrigation. If peripheral vestibular function was recovered, it would be impossible to differentiate between central compensation and peripheral recovery.[9] Because this study focused on the specific influence of vestibular exercises on central compensation, all patients who recovered peripheral vestibular function were excluded to ensure homogeneity of the patient group.

To quantify central compensation after an acute peripheral vestibular lesion we measured the time course of the recovery of three system-specific variables: for the vestibulo-ocular system, the ocular torsion (OT) as part of the ocular tilt reaction after a unilateral peripheral vestibular lesion[10]; for perception, the subjective visual vertical (SVV), which is tilted toward the side of the peripheral lesion[11]; and for postural control and the vestibulospinal reflex, the total sway path (SP) values[7,12] measured with the patient standing on a foam rubber, padded platform (to reduce ankle proprioception and, thereby, somatosensory input) and with the eyes closed (to eliminate visual input). The two conditions—foam rubber platform and eyes closed—were selected to increase the particular sensorial weight of the vestibular input in the multisensory control of postural balance.[13-15]

Methods. *Patients.* The diagnosis of vestibular neuritis was based on

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1. Patient history (acute/subacute onset of severe, prolonged rotational vertigo and nausea)
2. Clinical examination (horizontal/torsional spontaneous nystagmus [fast phase and the upper pole] toward the unaffected ear without evidence of a central vestibular lesion)
3. Postural imbalance with ipsiversive fall in the tandem (sharpened) Romberg test
4. Electronystagmography with caloric irrigation showing hyporesponsiveness or unresponsiveness of the horizontal canal of the affected ear as described elsewhere.[16] The maximum slow-phase velocity (MSPV) during caloric irrigation with 30 °C warm and 44 °C hot water should be less than 2 to 3 °/sec on the affected side for each irrigation. Because there is large intersubject variability we also used the vestibular paresis formula of Jongkees et al.[17]:
   (((R 30 °C + R 44 °C) − (L 30 °C + L 44 °C))/(R 30 °C + R 44 °C + L 30 °C + L 44 °C)) × 100
   where, for instance, R 30 °C is the MSPV during caloric irrigation with 30 °C warm water. Vestibular paresis was defined as more than a 25% asymmetry between the right-side and the left-side responses.[18] Our study included only those patients whose MSPV was less than 2 to 3 °/sec on the affected side *and* whose asymmetry was more than 25%. Caloric irrigation was performed on days 1, 7, 11, and 30. Patients who showed partial or complete recovery of labyrinth function (MSPV during caloric irrigation with 30 °C warm and 44 °C hot water >5 °/sec or a value of the vestibular paresis formula[17] < 25% asymmetry[18]) were excluded from the study.
5. Incomplete ocular tilt reaction (displacement of the SVV[11] and OT [cyclorotation] toward the side of the lesion[19] without skew deviation)
6. High-resolution MRI of the brainstem and inner ear that revealed no pathologic findings[20]

We excluded all patients with a history of vestibular dysfunction before actual symptom onset, a central vestibular disorder, polyneuropathy, marked decreased visual acuity, or other diseases that impair mobilization. If necessary, the patients received antiemetics (dimenhydrinate) for a maximum of 3 days because sedatives are known to retard central compensation.[21]

Initially, 82 patients with vestibular neuritis were included in the study from 1994 to 1997 and were randomized into two groups (control group versus therapy group). The patients in the control group did no specific vestibular exercises. Both groups, however, received identical encouragement to engage in regular daily activities. From the beginning of admission, the patients in both groups were instructed to get up at 7:00 AM; to walk to the bathroom (during the first 2 or 3 days with the help of a nurse, then subsequently alone); to take all meals at the table; to walk around their room, the ward, and, from day 3 onward, in the hospital park; and to watch TV and read newspapers or books.

The patients in the therapy group received, in addition, intensive physiotherapy (approximately 30 minutes three times daily) with components of Cawthorne-Cooksey and Norre exercises (described later[6]). Physical therapists initially treated all patients of this group in the hospital for 5 to 7 days. Subsequently the patients received a written home exercise program and instructions for vestibular exercises on three videos that were to be performed for another 3 weeks. Each video lasted 10 minutes. The patients were to perform the exercise program shown on the video three times daily, and subsequently they were to do the exercises according to the written instructions also for about 10 minutes three times daily. The patients were encouraged to perform especially those movements that provoked or increased their symptoms. The following three systems were trained specifically in the hospital as well as during the home exercise program:

1. The visual, ocular motor, and optokinetic systems, with smooth-pursuit, saccadic, and optokinetic eye movements. To generate smooth-pursuit eye movements, the patient was seated in front of a screen and was asked to follow a laser spot, which moved at random horizontally (±40°), vertically (±20°), and diagonally (±40°) at velocities of 10 to 70 °/sec. To generate optokinetic nystagmus, the patients viewed broad stripes (full-field stimulation) moving horizontally and vertically before them at velocities of 10 to 70 °/sec. To generate saccades, the patients viewed a large spot that jumped randomly on the screen in the horizontal, vertical, and diagonal directions with different amplitudes. Further, the patients had a ball that they had to toss from one hand to the other while following the ball with their eyes. These exercises were done three times daily in random order, and their duration was extended gradually from 1 to 5 minutes.
2. The vestibular system with repetitive head and body rotations and tilts. Patients were instructed to turn their head in the yaw, roll, and pitch planes at different velocities and frequencies while fixating on a large point. Frequency and amplitude of the head movements were indicated by instructions on the screen in combination with a metronome.
3. The somatosensory systems. *Standing:* Patients had to stand with their eyes open or closed and with their feet apart, close together, or in tandem. Under these different conditions they had to move their trunk in anteroposterior and mediolateral directions. Patients then had to stand with their head turned to the right and left, as well as flexed and extended, while performing the same movements anteroposteriorly and mediolaterally. A physical therapist supervised all movements. From day 3 onward the patients did these exercises on a foam exercise mat. Later they had to jump on a trampoline, always under the supervision of a physiotherapist. *Walking and running:* Patients were instructed to walk, with their eyes open or closed, forward, backward, sideways, and along a line. Later the patient had to perform these same exercises on a foam exercise mat and on ramps. From days 4 to 6 onward patients were instructed to walk outside the hospital on uneven surfaces and to start jogging. *Cervico-ocular reflex:* Patients were seated on a rotating chair while fixating a target in front of them. They were instructed to move their trunk while keeping their head position constant.

The experiments were performed in accordance with the II Helsinki Declaration, the study protocol was approved by the local ethics committee of our medical faculty, and all patients gave their informed written consent.

Techniques to measure central compensation. To quantify the differential effects of vestibular exercises versus no

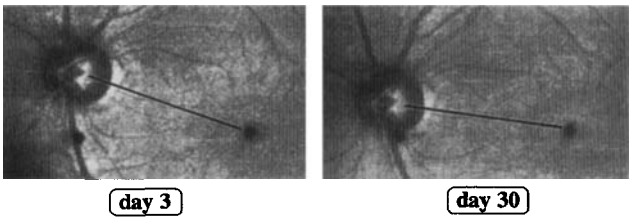

Figure 1. Fundus photograph of the left eye in a patient with vestibular neuritis on the left side shows 20° cyclorotation (ocular torsion) toward the left (excyclotropia, torsion of the papilla-fovea line clockwise from the viewpoint of the observer) on day 3 after symptom onset. On day 30, the ocular torsion was within the normal range ($x \pm 2$ SDs, -1 to 11.5°[23]). The earth horizontal is parallel to the horizontal of the upper and lower edges of the photograph.

particular therapy on central compensation following vestibular neuritis, we determined the time course of recovery for the following three system-specific variables:

1. OT for the vestibulo-ocular system.[19] OT (cyclorotation) of the eye on the affected side was measured in degrees with a scanning laser ophthalmoscope as described elsewhere by Dieterich and Brandt.[2,22] OT is defined as the mean of four to six fundus photographs taken by scanning laser ophthalmoscope with the patient sitting and the head upright. Photographs were taken for both eyes separately during fixation of a central target. An example is shown in figure 1. The position of the eye in the roll plane was measured as the angle formed by a straight line through the papilla and fovea (papilla-fovea meridian) and a horizontal line. According to this method, healthy control subjects had a slightly excyclotropic position of both eyes in the roll plane (i.e., counterclockwise rotation of the right eye, clockwise rotation of the left eye, from the viewpoint of the examiner). The normal range of OT (mean ± 2 SDs) was -1 to 11.5°.[23] Values beyond this range were considered pathologic.
2. SVV (adjustment measured in degrees) for perception[11] as described previously by Dieterich and Brandt,[23,24] and illustrated and explained in detail in figure 2.
3. SP values for postural control[12] measured on the Kistler platform. SP is the length of the path described by the center of force during a given time (20 sec). It is generated by the inherent instability of a subject standing on a recording platform and is approximated by the sum of the distances between two consecutive sampling points in the anteroposterior (sagittal = x) plane (i.e., sagittal sway, calculated as $\Sigma|\Delta x|$), the mediolateral (frontal = y) plane (i.e., frontal sway, calculated as $\Sigma|\Delta y|$), or in two dimensions as the total SP (calculated as $(\Sigma\sqrt{(|\Delta x^2| + \Sigma|\Delta y^2|)}$).

The total SP was used in this study to monitor postural control. The patients stood on a compliant, foam rubber, padded posturography platform that fulfilled the requirements suggested by Brandt et al.[25] (i.e., a slab 10 cm high, weighing 40 g/dm). Patients put their feet on symmetric marks drawn at an angle of approximately 30° so that the heels were approximately 2.5 cm apart. Parameters were measured on days 1, 3, 7, 11, 15, and 30 after symptom onset.

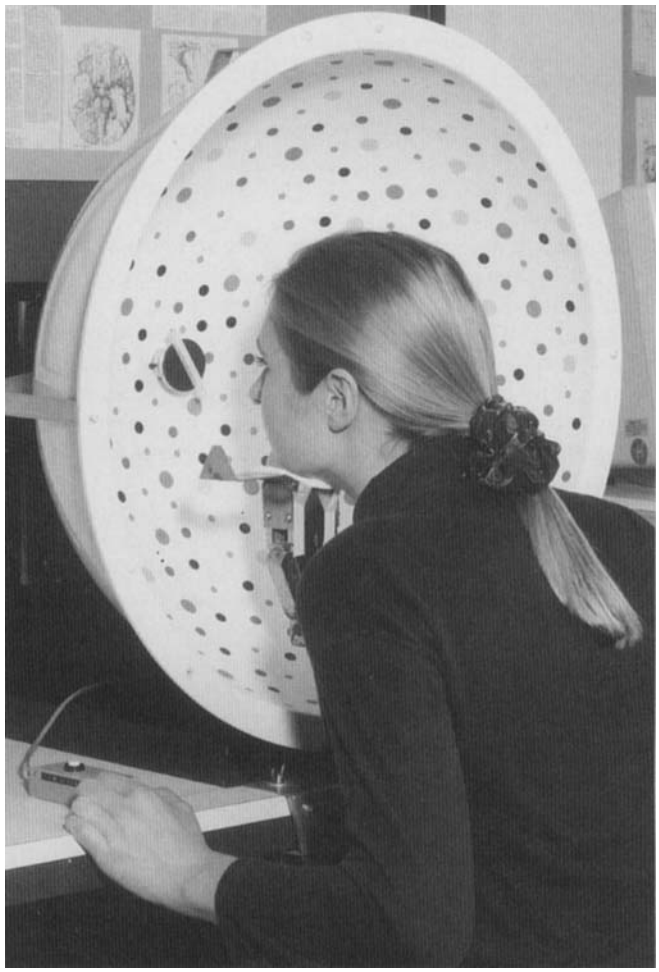

Figure 2. Measurement of the subjective visual vertical (SVV). The SVV was measured as each patient sat in an upright position and looked into a hemispheric dome that was 60 cm in diameter. The surface of the dome extended to the limits of the observer's visual field and was covered with a random pattern of colored dots, containing no clues about gravitational orientation. The center of the dome was fixed to the shaft of a direct current (DC) torque motor. Thirty centimeters in front of the observer was a circular target with a 14-deg visual angle with a straight line through the center; the target was mounted on a coaxial shaft connected to the DC servo motor. The patient had to adjust the central test edge to the vertical using a potentiometer. The output of the potentiometer was recorded in degrees on a personal computer. SVV was determined by the mean of 10 adjustments of the target disk from a random offset position to the subjective vertical. Under these conditions, the normal range ($x \pm 2$ SDs) of SVV is ± 2.5°.[23]

Criteria for dropouts and monitoring of compliance in the control and therapy groups. Our study protocol defined criteria for dropouts as follows: the patient does not want to continue to participate in the study or the patient was not compliant. In other words, for both the control and therapy groups, the patient does not come to the regular examinations (misses two or more of the examinations) and does not follow the instructions of the doctors and nurses for regular daily activities (as described earlier). For example, the patient spends more than 2 hours daily

in bed from day 3 onward during the daytime. In addition, for the therapy group, the patient does not continue the physiotherapy or misses more than 30% of the therapy.

During the training period in the hospital, compliance was monitored by the doctors, nurses, and, in the therapy group, also by the physiotherapists. The design of the study required that all patients be examined on days 1, 3, 7, 11, 15, and 30 after symptom onset. Furthermore, patients were observed daily during their hospitalization in our Department of Neurology (i.e., from day 1 to 7 by two neurologists [V.A. and M.S.]). During these regular examinations we asked the patients of both groups about their compliance, talked to them to determine whether they had problems with different parts of the exercise program (none of the 39 patients with persisting peripheral vestibular deficit had problems, as described later), and motivated them to continue doing the exercises.

Statistics. Statistical analysis was performed with ANOVA, and all data are expressed as mean ± SD. A level of $p < 0.05$ was defined as significant. Because we defined three major criteria to measure central compensation (SVV, OT, and SP), α was divided by n = 3 due to multiple statistical testing (i.e., α was 0.016; see figures 1 and 2).

Results. Eighty-two patients were initially included in the study and randomized to either the control group or the physiotherapy group. The two groups did not differ in mean age (control group, 52.4 ± 9.9 years; physiotherapy group, 51.7 ± 11.1 years [mean ± SD]) or sex ratio. Forty-three of 82 patients, however, showed a partial or complete recovery of labyrinth function on follow-up studies with caloric irrigation (slow-phase velocity during caloric irrigation with 30 °C warm and 44 °C hot water more than 5 °/sec or a value of the vestibular paresis formula[17] >25% asymmetry[18]) on day 30 after symptom onset and were, therefore, excluded from the study. Finally, the data of 20 patients in the control group and 19 in the physiotherapy group were analyzed. The combination of our instructions, the daily ward procedures and schedule, and the intensive personal care of V.A. and M.S., who observed daily every patient during their hospitalization in our Department of Neurology, ensured that none of these 39 patients with persisting deficits had to be excluded due to insufficient compliance (i.e., from day 3 onward none of the patients with a persisting deficit in the control or therapy group spent more than 2 hours in bed during the daytime).

The control and the physiotherapy groups did not differ in the mean slow-phase velocity of eye movements during caloric irrigation or in the values of the vestibular paresis formula, especially at day 30.

Vestibular neuritis caused an OT of the eye on the side of the lesion toward the affected ear (excyclotropia, see figures 2 and 3) that gradually declined over time, beginning with more than 20° at symptom onset. The control and physiotherapy groups did not differ in degree or time course for normalization of OT (see figure 3). At the statistical end point (day 30) the mean value (± SD) of the cyclorotation of the eye on the affected side toward the affected ear was 8.1 ± 3.3° for the control group (x ± SD, n = 20) and 9.0 ± 3.1° for the therapy group (n = 19). The mean values in both groups on day 30 were within the normal range (−1 to 11.5°[23]).

Vestibular neuritis also caused a displacement of the SVV toward the side of the lesion. Shortly after symptom onset the mean displacement of the SVV was about 11°. Afterward it declined gradually. The two groups also did not differ in normalization of the SVV (figure 4) for the perceptional function. On day 30 the mean value was 2.7 ± 1.8° for the control group and 3.5 ± 2.0° for the physiotherapy group. The normal range of the SVV is ± 2.5°.[23]

Vestibular neuritis caused a marked increase in the SP values. During the first days after symptom onset most of the patients could not stand long enough (>20 sec) on the foam rubber posturography platform with eyes closed to permit quantitative measurement of the SP values. Although in both the control and physiotherapy groups the SP values gradually declined over time, they differed significantly between the two groups at the statistical end

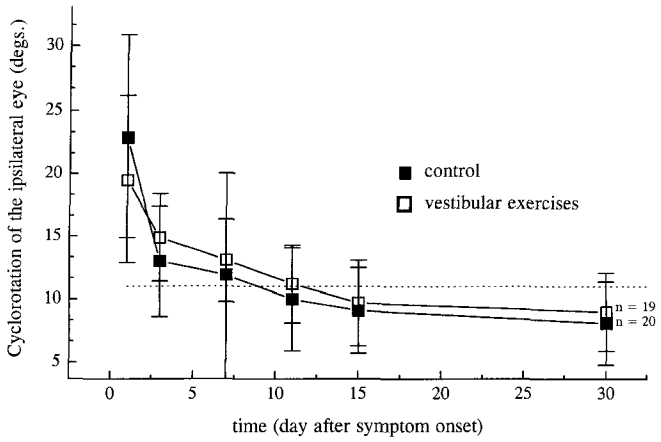

Figure 3. Time course of the changes of ocular torsion (cyclorotation) of the eye ipsilateral to the peripheral vestibular lesion in the control and physiotherapy groups. There is no difference between the two groups (i.e., vestibular exercises have no effect on vestibulo-ocular functions). There was always an ocular torsion ipsiversive to the peripheral vestibular lesion (part of the incomplete ocular tilt reaction), which normalized over time. The dotted line indicates the normal range (x ± 2 SDs, −1 to 11.5°[23]).

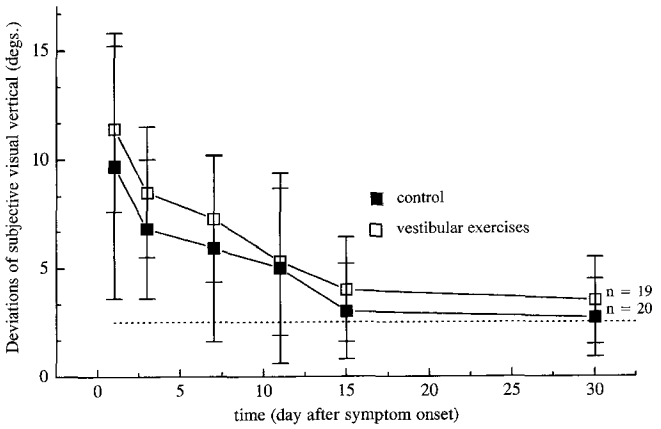

Figure 4. Time course of the deviations of SVV in the control and physiotherapy groups. There is no difference between the two groups (i.e., vestibular exercises have no influence on perceptional functions). Patients with vestibular neuritis always exhibited a deviation toward the side of the lesion, which normalized over time. The dotted line indicates the normal range (x ± 2 SDs, ± 2.5°[23]).

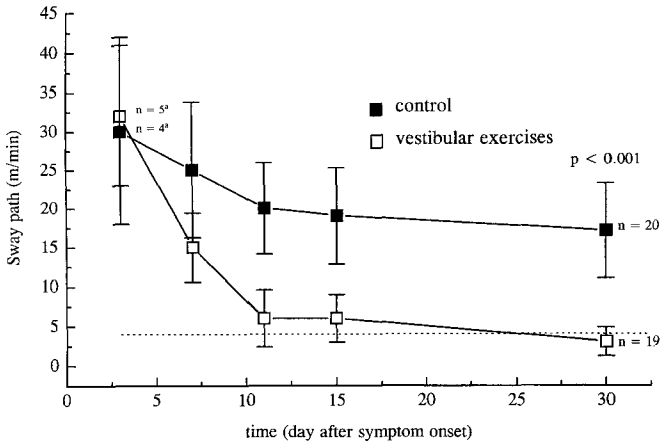

Figure 5. Time course of the changes in total sway path (SP) values for the control and physiotherapy groups. Vestibular exercises improved central vestibulospinal compensation. For postural control we measured the total SP values (meters per minute, mean ± SD) of patients with eyes closed while standing on a compliant, foam-padded posturography platform. There was a significant difference (ANOVA, p < 0.001) between the two groups at the statistical end point (day 30 after symptom onset). The dotted line indicates the normal range. [a]During the first days after symptom onset, not all of the patients could stand long enough (>20 sec) on the platform to permit accurate quantitative measurement of the SP values.

point on day 30 after symptom onset (figure 5). The total SP value in the physiotherapy group was 3.2 ± 1.9 m/min, and in the control group it was 16.9 ± 6.1 m/min (ANOVA, $p < 0.001$; see figures 3 through 5).

Discussion. *Vestibular system and central compensation.* The vestibular system provides an excellent model for investigations of neural and behavioral plasticity because 1) the peripheral lesion can be precisely located and is restricted (without disturbing central parts of the vestibular system, which are important for plasticity) and 2) the recovery of function as well as its time course can be quantified at different levels (perception, vestibulo-ocular, and vestibulospinal reflexes).[26]

Vestibular exercises and central compensation. The use of vestibular exercises in the treatment of patients with vestibular lesions has become increasingly popular, but its efficacy is not yet proved.[6] Such exercises are supposed to accelerate and improve central compensation via the mechanisms of habituation training, which enhance adaptation of the vestibulo-ocular reflex and vestibulospinal reflexes as well as increase substitution. Previous studies (for example, by Shepard, Herdman, and others[27–31]) demonstrated that vestibular exercises can improve vestibular symptoms in patients with *chronic* unilateral and bilateral vestibular failure. There are no prospective studies on the efficacy of vestibular exercises in humans with a normal vestibular function before symptom onset.[3] Only one previous study[31] analyzed the effects of an exercise program during the *acute* stage after vestibular loss. Evaluation of the effects of vestibular adaptation in 21 patients after acoustic neuroma resection showed that vestibular exercises can improve postural stability. There are, however, substantial methodologic differences between this earlier study[31] and ours. For example, the size of the acoustic neuromas reported in study by Herdman et al.[31] varied from 1 to 5 cm. This suggests that those patients had peripheral (and/or central) vestibular lesions before surgery (i.e., had already undergone considerable vestibular compensation during the growth of the tumor). Furthermore, even before surgery the postural stability of those patients differed significantly from that of age-matched normal subjects,[31] either due to a peripheral or a central vestibular dysfunction. In contrast, we could assume that our patients with vestibular neuritis had intact peripheral and central vestibular functions before symptom onset because 1) recurrent vestibular neuritis is very rare[32]; 2) none of our patients had vertigo before the actual symptom onset, which would have indicated a previous acute vestibular deficit; and 3) none of the patients had signs of central vestibular dysfunction. Our prospective clinical study suggests that specific physiotherapy in the acute stage of the disease improves vestibulospinal compensation in patients with acute peripheral vestibular lesions.

Improvement of postural stability in stance and vestibulospinal compensation. In our study we attempted to test and to quantify each system (perception, ocular motor, and vestibulospinal) separately. (All variables were measured in degrees or meters per minute.) This provided information on complementary compensation by different sensory inputs. The total SP values were tested in patients with eyes closed to eliminate the visual contribution and while standing on a foam rubber platform to reduce the contribution of foot-ankle proprioception. In this way, vestibulospinal reflexes could be tested more specifically.[13,33] However, we are aware that even when the SP values are measured under these restricted conditions they do not indicate only dysfunctions of vestibulospinal (head-body loop) reflexes, but also (due to tonal imbalance) indicate disturbances of the VOR, especially during the acute phase of the disease.

The question arose as to why only the SP values were affected by vestibular exercises (i.e., why were effects seen in the vestibulospinal reflexes and not at the level of the vestibulo-ocular reflex and perception?). Incongruencies in the time course and the magnitude of the changes in behavior and neural activity during central compensation have also been described by others.[34] For example, after hemilabyrinthectomy in the frog, 50% of postural recovery is accomplished within the first 2 weeks.[34] At that time the commissural vestibular changes, however, have reached only 5% of their maximum, which is achieved about 60 days after hemilabyrinthectomy for postural recovery as well as commissural chang-

es.[34,35] These studies indicate that multiple processes of compensation occur in distributed neural networks in different locations, at different times, and to different extents.[23,34] Furthermore, it has also been shown in the squirrel monkey and the cat that vestibular exercises have a pronounced effect on locomotor equilibrium compensation.[6,36,37] Our data agree with the findings of these animal experiments. Another explanation for the previously mentioned discrepancy is that the vestibulo-ocular and perceptual systems appear to be trained already when you simply open your eyes, look around, and experience vision. Therefore, studies on the subcomponents of the treatment program and the duration of therapy (see figure 5, which shows major improvements of balance during the first 10 days) are necessary because the therapy battery used in this study was quite extensive.

Due to the faster recovery of balance function by the patients in the therapy group, however, these patients naturally performed more normal daily activities after a few days and resumed more physical activities (e.g., playing tennis or returning to work) earlier. This was a desired secondary therapeutic effect of the vestibular exercises.

Clinical relevance. From a clinical point of view, our findings strongly support empirical prescription of vestibular exercises to improve balance function, which is important for the rehabilitation of patients with a unilateral peripheral vestibular lesion. Based on our results, we would recommend vestibular exercises for patients with a peripheral vestibular deficit. Moreover, from our data (see figure 5) as well as from animal experiments in the monkey, cat, and guinea pig, which provide evidence of a critical period for adaptation and compensation,[2,37-40] we conclude that exercise regimens should begin early after symptom onset, although this has not yet been proven in humans.

Acknowledgment

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CADASIL in a North American family

Clinical, pathologic, and radiologic findings

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Article abstract—*Objective:* To expand the reported phenotypic range of cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL). *Background:* Despite numerous patient reports, our knowledge of the phenotypic range of CADASIL remains incomplete. *Method:* We performed clinical, pathologic, and radiologic examinations on members of a family with CADASIL. *Results:* The proband is a 61-year-old man with a history of migraine and depression who has experienced multiple subcortical infarctions resulting in a stepwise decline. Neuropsychological testing documented a dementia syndrome with frontal lobe features and neurologic examination noted a left hemiparesis and a right-sided palmomental reflex. Brain biopsy with light microscopy revealed a nonatherosclerotic small-vessel angiopathy with periodic acid-Schiff positive granular changes in the media and white matter gliosis, with unremarkable cortex. Genetic testing confirmed a *Notch3* mutation. The proband's first cousin has a history of depression, one seizure possibly resulting from an acute stroke, and a learning disorder. Neuropsychological testing demonstrated deficits in executive function and neurologic examination noted persistent extraneous adventitial movements, poor coordination, and primitive reflexes. Skin biopsy with electron microscopy demonstrated granular osmiophilic material within the basement membrane of vascular smooth muscle cells, which is considered to be pathognomonic of CADASIL. The proband's older son and younger son have histories of migraine and depression, respectively, and both also had learning disorders. MRI revealed diffuse white matter disease extending into the temporal lobes, and lacunar infarctions in these four nonhypertensive patients. Other family members have experienced migraine, recurrent stroke, dementia, and depression. *Conclusions:* CADASIL is a genetic basis for vascular dementia that may be manifest earlier in life than previously reported.

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Numerous studies have investigated cerebrovascular disease as a basis for dementia. Their findings have suggested that many factors work in combination to elevate risk, including specific infarct characteristics such as volume, multiplicity, and location; white matter disease; vascular risk factors such as diabetes mellitus; and comorbid illnesses, particularly those that might produce cerebral ischemia or hypoxia.[1] Although it is understood that certain host characteristics, such as older age and fewer years of education, may also increase the risk of dementia, studies focusing on the role of genetic factors have been sporadic, consisting primarily of reports by Van Bogaert[2] and Sourander and Wålinder[3] of a hereditary form of multi-infarct dementia. Recently, renewed interest has led to a better understanding of the phenotypic manifestations and genetic basis for this disorder, which has been termed cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL).[4] Affected patients typically experience multiple recurrent subcortical ischemic events, leading to a stepwise decline and a dementia syndrome with frontal lobe features. Migraine and depression are also common. Brain imag-

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Vestibular exercises improve central vestibulospinal compensation after vestibular neuritis

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