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# Adaptive Motor Behavior of Cerebellar Patients During Exposure to Unfamiliar External Forces

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**ABSTRACT.** The authors investigated adaptation of goal-directed forearm movements to an unknown external viscous force assisting forearm flexion in 6 patients with cerebellar dysfunction and in 6 control participants. Motor performance was generally degraded in cerebellar patients and was markedly reduced under the force condition in both groups. However, patients and controls were able to adapt to the novel force within 8 trials. Only the healthy controls were able to improve motor performance when readapting to a null-force condition. The results indicate that cerebellar patients' motor control system has imprecise estimations of actual limb dynamics at its disposal. Force adaptation may have been preserved because single-joint movements were performed, whereas the negative viscous force alone and no interaction forces had to be compensated.

*Key words:* cerebellum, human, inverse dynamic models, motor control, motor learning

Myriad studies have documented the involvement of the cerebellum in the control and acquisition of voluntary movements. It is well known that cerebellar lesions result in dysmetric and decomposed movements (for a review, see Thach, Goodkin, & Keating, 1992; Timmann & Diener, 1998). Biomechanical analyses have suggested that the underlying reason for the observed ataxia is the inability of cerebellar patients to produce appropriate muscle torques to compensate for interaction joint torques (Bastian, Martin, Keating, & Thach, 1996; Bastian, Zackowsky, & Thach, 2000) or their inability to generate task-adequate levels of muscle force per se (Thach, Perry, Kane, & Goodkin, 1993; Topka, Konczak, & Dichgans, 1998; Topka, Konczak, Schneider, Boose, & Dichgans, 1998). The intact cerebellum is also crucial for motor learning, because it seems to be part of a network that mediates nonassociative (Deuschl, Toro, Zeffiro, Massaquoi, & Hallett, 1996; Lang & Bastian, 1999; Martin, Keating, Goodkin, Bastian, & Thach, 1996; Sanes, Dimitrov, & Hallett, 1990; Sanes, Donoghue, Thangaraj, Edelman, & Warach, 1995) and associative forms of learning (for a review, see Hesslow & Yeo, 1998).

The results of recent systems research suggest that an intact cerebellum is essential for the performance and updating of so-called internal motor models (Wolpert, Miall, & Kawato, 1998). Two types of internal motor models can be distinguished. In forward models, a neural representation of the relationship between the forces causing movements and the resulting movement kinematics is formed. An inverse dynamic model is part of a neural controller that transforms planned kinematic trajectories into appropriate patterns of muscular innervation (Jordan & Rumelhart, 1992; Kalveram, 1992; Wolpert, Ghahramani, & Jordan, 1995a, 1995b).

Concerning internal forward models, it was shown recently that healthy controls benefited from advance information about an incoming mechanical perturbation to the arm by altering their muscular response pattern (earlier triceps onset; Timmann, Richter, Bestmann, Kalveram, & Konczak, 2000). However, a group of cerebellar patients did not benefit from advance information (Timmann et al., 2000). That is, an intact cerebellum seems to be indispensable for the performance and updating of internal forward models so that the inherent time delays in afferent feedback can be overcome (Vercher & Gauthier, 1988).

Accumulating evidence from modeling, experimental, and neurophysiological studies has provided support for the idea that motor systems rely on inverse models when controlling target movements (e.g. Bhushan & Shadmehr, 1999; Kalveram, 1991; Shadmehr & Brashers-Krug, 1997; Shidara,

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Kawano, Gomi, & Kawato, 1993; Wolpert et al., 1998). The main results of those studies have indicated that inverse dynamic models are context specific (Wolpert & Kawato, 1998) and adaptable and are gradually built with practice (Shadmehr & Mussa-Ivaldi, 1994). The models are not global but instead are confined to neighboring regions of the work space that are experienced during the training session (Gandolfo, Mussa-Ivaldi, & Bizzi, 1996). Early stages of learning are driven by a delayed error-feedback signal (Thoroughman & Shadmehr, 1999). Furthermore, two inverse dynamic models can be learned and retained for up to 5 months if the training sessions for each task are separated by an interval of approximately 5 hr so that interferences are avoided (Shadmehr & Brashers-Krug, 1997; Shadmehr & Holcomb, 1999).

There is also growing evidence that inverse dynamic models are either located in the cerebellum or mediated by cerebellar processes (Kawato, Furawaka, & Suzuki, 1987; Kawato & Gomi, 1992a, 1992b; Schweighofer, Arbib, & Kawato, 1998; Shidara et al., 1993; Wolpert et al., 1998). According to the proposed neural mechanism, afferent information about planned trajectories and the actual limb state reach the cerebellar cortex by way of mossy and parallel fibers, whereas an efference copy of the motor command arrives through climbing fibers, thus providing an error signal for the adaptation of the feedforward motor command. The notion of such "cerebellar feedback error learning" (Wolpert et al., 1998, p. 339; Kawato & Gomi, 1992a; Kawato et al., 1987) has obtained support from neurophysiological studies of the ventral paraflocculus in monkeys during eye-tracking movements (Gomi et al., 1998; Kobayashi et al., 1998) and from brain imaging studies of visually guided arm movements (Imamizu et al., 1997; Imamizu et al., 2000; Kitazawa, Kimura, & Yin, 1998). Furthermore, retention of a newly learned inverse dynamic model seems to involve the cerebellar cortex, as shown in an imaging study conducted by Shadmehr and Holcomb (1997). One can investigate adaptation of inverse dynamic models by asking participants to move their arms in unfamiliar force fields. It can be shown that healthy participants adapt to the changed environment quickly; that is, they learn to move as accurately as they do in a baseline condition without force application (Shadmehr & Mussa-Ivaldi, 1994). Impaired adaptation to unfamiliar force fields or spring-like loads has been demonstrated in patients with Parkinson's disease (Krebs, Hogan, Hening, Adamovich, & Poizner, 2001) and in patients with severe hemiparesis after stroke (Dancause, Ptito, & Levin, 2002). However, the cerebellar involvement in the learning of new inverse dynamic models in patients with cerebellar diseases has not yet been explicitly addressed.

In the present experiment, therefore, we focused on feedforward control mechanisms and investigated whether the performance differences between cerebellar patients and healthy controls are consistent with the notion of cerebellum-based inverse dynamic models. We tested whether the adaptation to a negative viscous external force during forearm flexion movements was impaired in that patient group.

## Method

### *Participants*

Six patients with impaired cerebellar function ($M$ = 53 years, $SD$ = ± 16 years, range = 30–70 years) and 6 healthy age- and gender-matched control participants ($M$ = 48 years, $SD$ = ± 14 years, range = 32–66 years) participated in the study. All but 1 participant were right-handed. All patients had marked to severe cerebellar ataxia, according to their score on the International Cooperative Ataxia Rating Scale of the World Federation of Neurology (WFN scale; Trouillas et al., 1997). Four patients had a degenerative cerebellar disease, either of unknown etiology ($n$ = 3; idiopathic cerebellar ataxia) or spinocerebellar ataxia type 6 ($n$ = 1). The 5th patient presented with alcoholic cerebellar degeneration. The last patient suffered from ischemic infarction in the territory of the right superior cerebellar artery. Basic characteristics of patients with cerebellar lesions are summarized in Table 1. Written informed consent was obtained from all participants. The local research ethics committee approved the experiment.

**TABLE 1. Basic Characteristics of Patients With Cerebellar Lesions**

| P | Age | Gender | Diagnosis | Onset of disease | Total WFN ataxia score |
|---|---|---|---|---|---|
| 1 | 57 | m | IDCA | 1999 | 27/100 |
| 2 | 39 | m | Alcoholic cerebellar degeneration | 1995 | 64/100 |
| 3 | 66 | m | IDCA | 1997 | 26/100 |
| 4 | 30 | m | Right SCA infarction | 1989 | 24/100 |
| 5 | 56 | m | Spinocerebellar atrophy | 1984 | 59/100 |
| 6 | 70 | f | IDCA | 1997 | 22/100 |

*Note.* P = patient; m = male; f = female. WFN = World Federation of Neurology. Maxiumum WFN ataxia score = 100; the higher the score, the worse the clinical ataxia. IDCA = idiopathic cerebellar atrophy; SCA = superior cerebellar artery.

*Procedure*

Participants performed ballistic flexion movements of the right forearm around the elbow joint. Their right forearm was inserted into an orthosis that was attached to the upper lever arm of a manipulandum. The lower lever was connected to a torque motor and was coupled to the upper lever by two flat irons. Viscous forces of the arm–lever system could be generated by a torque motor, which received its input from a PC that used MATLAB (The MathWorks, Natick, MA) and SAS software. We measured angular position with a potentiometer attached to the motor shaft.

Participants viewed the goal position and their starting position on a convex screen located about 1.5 m in front of them. An illuminated arrow on the screen indicated current arm position. The position arrow disappeared after the movement velocity exceeded 2°/s so that feedback-driven visual guidance of the arm would be avoided. Furthermore, a Styrofoam board that covered the manipulandum prevented visual feedback of the arm. The apparatus is shown in Figure 1.

Participants were instructed to hold an elbow position of –20°, that is, 20° right of the midsagittal, and to relax their arm before trial onset. At trial onset, the target arrow moved to a position 10° left of the midsagittal axis. Thus, movement amplitude was 30°. Participants were instructed to match the position of the goal arrow with the arrow representing arm position on the convex screen in front of them. Instructions were given to match the two arrows as fast and accurately as possible. Intertrial intervals were pseudorandomized and ranged between 10 and 14 s.

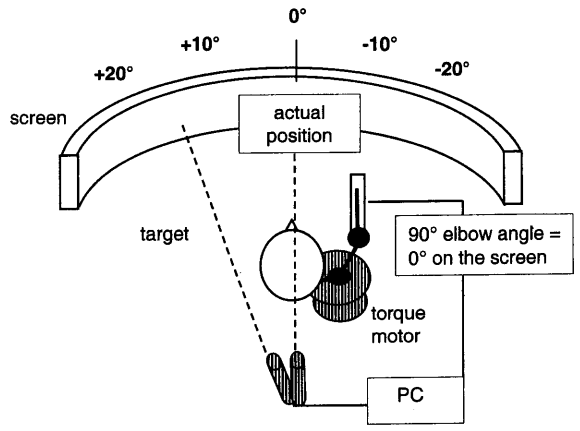


**FIGURE 1.** Experimental setup. Participants sat on a chair, with their right forearm inserted into an orthosis. Participants viewed two illuminated arrows on a screen in front of them, corresponding to the target and to the actual position of the arm, respectively. Ninety-degree elbow flexion corresponded to 0° position of the manipulandum. A torque motor attached to the manipulandum generated a negative viscous force.

*Experimental Design*

Movements were performed under two different force conditions. In the null-force condition, no external force was applied to the manipulandum during movement execution. In the underdamped condition, a viscous force of –2 cNm/(°/s) was applied to the manipulandum at movement onset, which assisted the arm movement proportional to its velocity. Three conditions were performed, with 60 trials in each condition. The amplitude of the movement was always 30° (from –20° to 10°). The order of force application was null force–force–null force. The 180 trials were performed in approximately 45 min.

*Data Analysis*

Angular position was measured for each trial by a potentiometer at the motor shaft. The data were sampled at 520 Hz and were digitized with a 12-bit analog-to-digital converter (Meilinghaus ME300). Digital data were stored on hard disk and then filtered offline with a second-order Butterworth filter at a 10-Hz cutoff frequency. To accomplish comparability between the trajectories, we aligned the curves to movement onset. Movement onset was determined as the time when angular path exceeded –18°. We performed filtering and subsequent statistical analysis by using routines based on MATLAB and SAS-software.

We derived the following measures from the raw kinematics:

*Target error.* This variable was computed as the absolute difference between target position and the first position maximum (see Figure 2 for an illustration). It represents differences in spatial accuracy with respect to the target:

$$\text{Target error} = |\text{target position } (^\circ) - \text{first position maximum } (^\circ)|. \quad (1)$$

When there was no overshoot in the movement, the final position error was used for computing target error.

*Individual trajectory difference score* (*ITDS*). For each participant, the mean absolute difference between the trajectory of each trial and the mean null-force baseline trajectory of that participant was computed (individual null-force baseline trajectory). The individual null-force baseline trajectory was determined as the mean trajectory of Trials 31–60, that is, the second half of the first condition of unperturbed movements. One could reasonably assume that participants had mastered the task during those trials and that initial learning difficulties were not a confounding factor (see Figure 3). We computed the difference score for each timed sample by using the following formula:

$$(\Sigma|\text{participant}_k \text{ baseline position}_i - \text{position}_i \text{ of trial}_j|)/\text{number of samples}, \quad (2)$$

where $k$ is the participant number, $i$ is the $i$th sample, and $j$ indicates the $j$th trial of participant $k$.The region considered

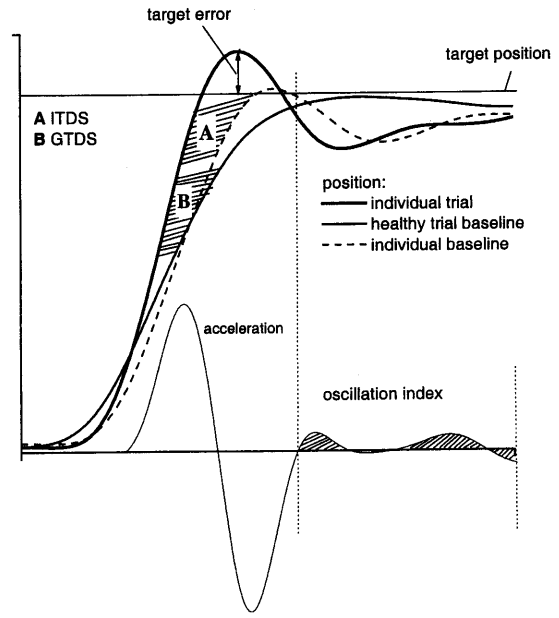


**FIGURE 2.** Dependent variables. Target error = absolute difference between target position and first position maximum. Individual trajectory difference score (ITDS) = mean absolute difference between the actual trajectory and an individual null-force baseline trajectory. Group trajectory difference score (GTDS) = absolute difference between the actual trajectory and the unimpaired individuals' trial-based reference trajectory for each trial. The latter was determined as the mean trajectory of each trial of all healthy control participants. Oscillation index = mean absolute difference between the acceleration curve and the *x*-axis after the second zero crossing of the acceleration.

ranged from movement onset (time when angular path exceeded –18°) to the end of the acceleration phase (second zero crossing of acceleration).

The ITDS allows one to assess within-participant differences with respect to each participant's baseline performance level (during the second portion of the first null-force condition) and, thus, to measure the effect of the negative viscous force on kinematics.

*Group trajectory difference score (GTDS).* To examine if trajectories differed between patients and controls, we computed the absolute difference between each actual trajectory and a trial-based "healthy" reference trajectory. The latter was determined as the mean trajectory of each trial of all 6 healthy control participants. The absolute difference between the reference trajectory and an individual trial trajectory was computed for a fixed interval of 900 timed samples from movement onset and was subsequently summed, as follows:

$$\Sigma|\text{control group mean position}_i \text{ of trial}_j - \text{position}_i \text{ of trial}_j|, \quad (3)$$

where $i$ is the $i$th sample and $j$ indicates the mean position on the $j$th trial, respectively, of an individual participant in the healthy control group.

Thus, for the computation of GTDS, there was a unique reference trajectory for each trial (180 trajectories). Each reference trajectory represented the mean position curve for the 6 healthy participants. That reference trajectory represented the "gold standard"; that is, it supposedly captured the prototypic shape of a trajectory generated by a healthy motor system.

We designed the GTDS to express between-group differences. That measure allowed us to compare each individual's performance with that of a hypothetical healthy trajectory. The measure was biased because it was based on the mean performance of the control group. Consequently, differences were expected to be smaller for control participants than for cerebellar patients.

*Oscillation index.* To determine whether trajectories differed after the first movement unit (time after first acceleration and deceleration), we computed an oscillation index by summing up the absolute difference between the acceleration curve and the *x*-axis after the second zero crossing of the acceleration until the end of the movement recording, which had a duration of 2.9 s. We then divided that sum by the number of samples to get an average index. The oscillation index provided information about the degree of the final intention tremor in patients.

$$(\Sigma|\text{acceleration}|/\text{number of samples}). \quad (4)$$

Because the movements of cerebellar patients were generally more variable and less smooth than those of healthy adults, it was important to analyze not only the movement trajectory but also whether the target was reached accurately (target error; Day, Thompson, Harding, & Marsden, 1998). Consequently, we computed ITDS to analyze the individual variability of the trajectory paths in cerebellar patients and in healthy controls. With the GTDS measure, we emphasized between-group differences by comparing trajectory smoothness in cerebellar patients with the smoothness of the average trajectory of healthy controls. Finally, the oscillation index enabled us to measure well-known difficulties of cerebellar patients in the braking process of target movements (Hore, Wild, & Diener, 1991; Topka et al., 1999).

### *Statistical Analysis*

We knew from previous pilot work (Richter, 2001, unpublished data) that healthy adults adapt within the first 20 trials to the negative viscous force. To capture any possible delays in adaptation in the cerebellar patient group and to guarantee that learning was complete, we required participants to perform 60 trials in each experimental condition. Our initial analysis then revealed that the main kinematic changes in both groups took place well within the first 20 trials (see Figure 3). For comparison, in Table 2 are the group means of the target error of the 1st to 4th, 17th to 20th, and the last 4 trials of each experimental condition. As

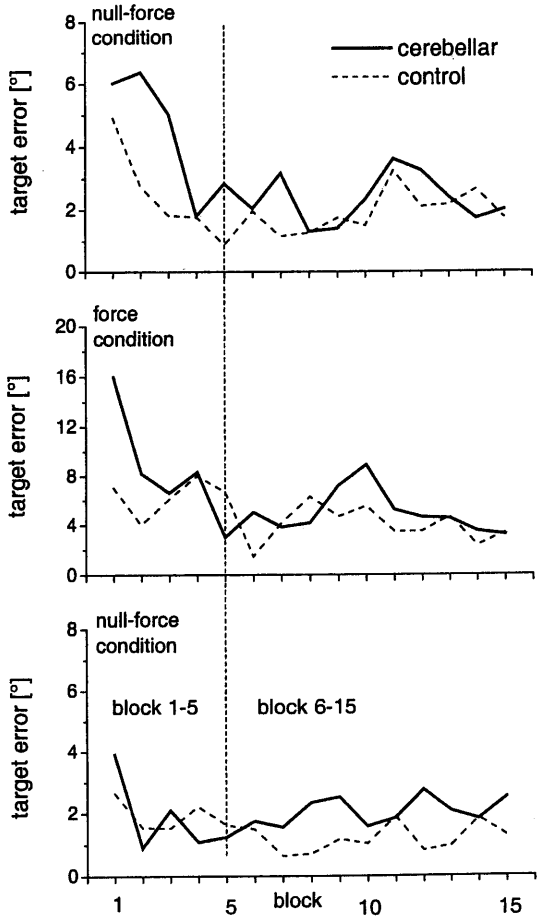


**FIGURE 3.** Mean individual target error (°) over 4 successive trials each in the three conditions (null force, viscous force, and null force) for 1 exemplary participant in each experimental group. Note that main kinematic changes took place well within the first 20 trials, that is, five blocks.

can be seen in the table, the mean of the last 4 trials in each experimental condition was similar to the mean of the 17th to 20th trials. That is, target error had reached its final value by the end of the first 20 trials of each experimental condition. Therefore, we report here results of only the first 20 trials of each experimental condition.

We performed a 2 (group: healthy controls and cerebellar patients) × 5 (block) × 3 (force: null force, force, null force) analysis of variance, including planned quantitative comparisons for the block variable. The first 20 trials analyzed in each condition were averaged in five groups of 4 consecutive trials. That averaging resulted in smoothed learning curves whose time course was masked by considerable intraindividual variation (see Figure 3). We attempted exponential curve fitting, as demonstrated by Deuschl et al. (1996) and by Lang and Bastian (1999). Those procedures did not provide a good fit for some of the data, however, so we resorted to alternative methods.

## Results

### Effects on Motor Performance

Cerebellar patients had basic difficulties in motor performance that were evidenced by less accurate movements than those of control participants. Target error was larger in patients than in controls: main effect of group for target error ($M_{patient}$ = 5.3°, $SD$ = 3.2°; $M_{control}$ = 3.8°, $SD$ = 2.6°), $F(1, 150) = 14.76$, $p < .0002$. In addition, the shape of the patients' trajectories differed from that of control participants. Patients' trajectories showed a greater deviation from the healthy reference trajectory than did the controls' trajectories: main effect of group for GTDS ($M_{patient}$ = 1,589°, $SD$ = 1,063°; $M_{control}$ = 1,047°, $SD$ = 669°), $F(1, 150) = 17.84$, $p < .0001$. Finally, patients had more difficulties terminating the movement on the target. They showed more oscillations after the first movement phase than did controls: main effect of group for oscillation index ($M_{patient}$ = 52.1°/s$^2$, $SD$ = 57.6°/s$^2$; $M_{control}$ = 16.1°/s$^2$, $SD$ = 12.7°/s$^2$), $F(1, 150) = 47.69$, $p < .0001$.

Individual (three pre- and four posttransitional) trajectories of 1 healthy control participant and 1 cerebellar patient are shown in Figure 4. It is evident that both participants were disturbed by the change in force condition, which they expressed by overshooting (force condition) or undershooting (second null-force condition) the target. It is important to note that the patient was disturbed to a greater extent than the control participant by a change in force condition, mainly in the viscous-force condition.

Patients had more difficulties in terminating the movement on the target after the introduction of the external force than healthy controls did. Accordingly, we found a significant Group × Force interaction for oscillation index, $F(2, 150) = 16.98$, $p < .0001$. In addition, healthy participants improved performance in comparison with their individual null-force baseline trajectory in the last null-force condition, whereas cerebellar patients returned to the initial performance level. Although the healthy controls' trajectories were more similar to the individual null-force baseline trajectory in the last null-force condition than in the first null-force condition, the cerebellar patients' trajectories were not; Group × Force interaction for ITDS, $F(2, 150) = 3.77$, $p < .0254$ (see Figure 5).

### Effects of Learning

Repeated measures analysis showed that both cerebellar patients and healthy controls were able to increase their performance over blocks. Block effects were found but not an interaction between block and group. All participants' trajectories became increasingly similar to the healthy reference trajectory over blocks; main effect of block for GTDS, $F(4, 150) = 2.86$, $p < .0253$. In addition, all participants' trajectories became increasingly similar to their individual null-force baseline trajectory over blocks; main effect of block for ITDS, $F(4, 150) = 11.62$, $p < .0001$. Last, patients and controls reached the target more accurately over blocks;

**TABLE 2. Mean, Standard Deviation (*SD*), and Range of Target Error (deg) in the 1st, 5th, and Last Block of Each Experimental Condition in Cerebellar Patients and in Healthy Controls**

| | Cerebellar patients | | | Healthy controls | | |
|---|---|---|---|---|---|---|
| Block (Trials) | *M* | *SD* | Range | *M* | *SD* | Range |
| | | | *Null-force condition* | | | |
| Block 1 (Trials 1–4) | 5.1 | 1.5 | 3.3–7.2 | 4.1 | 1.3 | 2.5– 6.0 |
| Block 5 (Trials 17–20) | 3.3 | 1.9 | 0.9–6.5 | 3.4 | 1.9 | 1.4–5.8 |
| Block 15 (Trials 57–60) | 4.0 | 3.2 | 1.4– 8.2 | 3.6 | 1.2 | 2.0–5.5 |
| | | | *Force condition* | | | |
| Block 1 (Trials 61–64) | 8.3 | 3.0 | 4.1–12.6 | 7.8 | 4.3 | 4.3–16.0 |
| Block 5 (Trials 77–80) | 5.9 | 2.7 | 1.2–8.3 | 4.1 | 1.9 | 1.5–7.1 |
| Block 15 (Trials 117–120) | 4.1 | 2.2 | 0.8–7.4 | 4.6 | 1.4 | 3.2–7.2 |
| | | | *Null-force condition* | | | |
| Block 1 (Trials 121–124) | 6.0 | 2.4 | 2.7–8.6 | 3.2 | 1.2 | 1.7–5.1 |
| Block 5 (Trials 137–140) | 1.8 | 0.4 | 1.5–2.3 | 2.7 | 2.4 | 0.9–7.2 |
| Block 15 (Trials 177–180) | 2.2 | 1.1 | 1.3–4.0 | 2.5 | 0.7 | 1.9–3.5 |

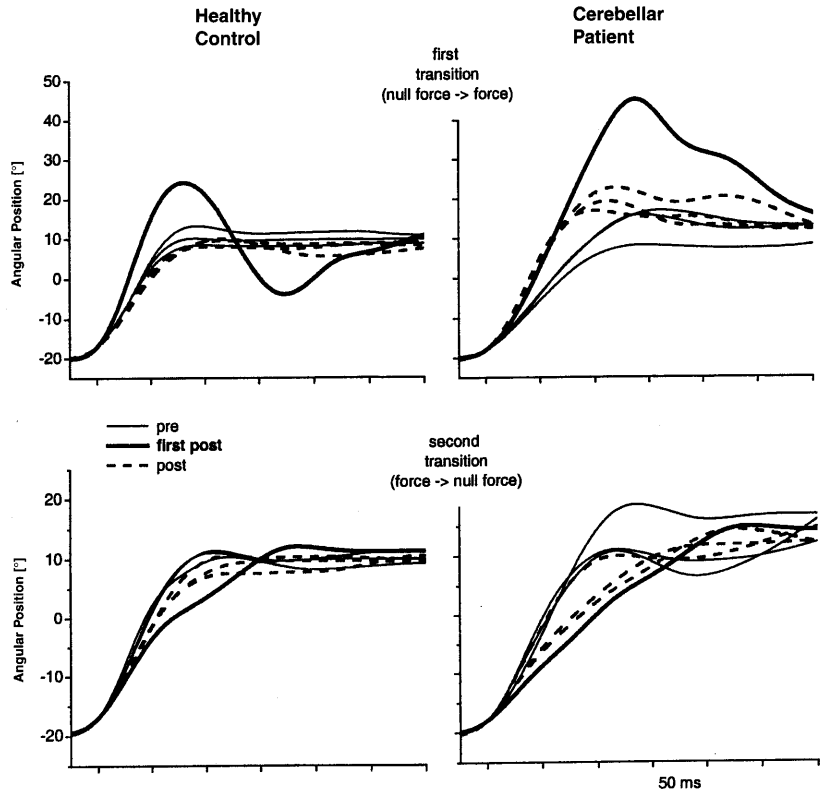


**FIGURE 4.** Individual trajectories (angular position in degrees) of 1 healthy participant and 1 cerebellar patient. Shown are the trajectories of three pre- and four posttransitional trials. In the first transition, from the null-force to the force condition, participants showed overshooting; in the second transition, from the force condition to the second null-force condition, they revealed undershooting.

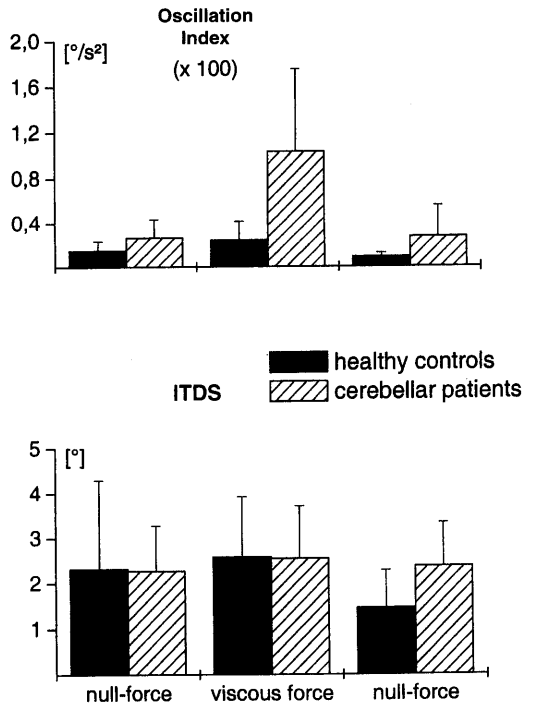


**FIGURE 5.** Mean oscillation index and individual trajectory difference score (ITDS) for healthy controls (shaded area) and cerebellar patients (lined area) in the three experimental conditions. Shown are the means and standard deviations for each condition.

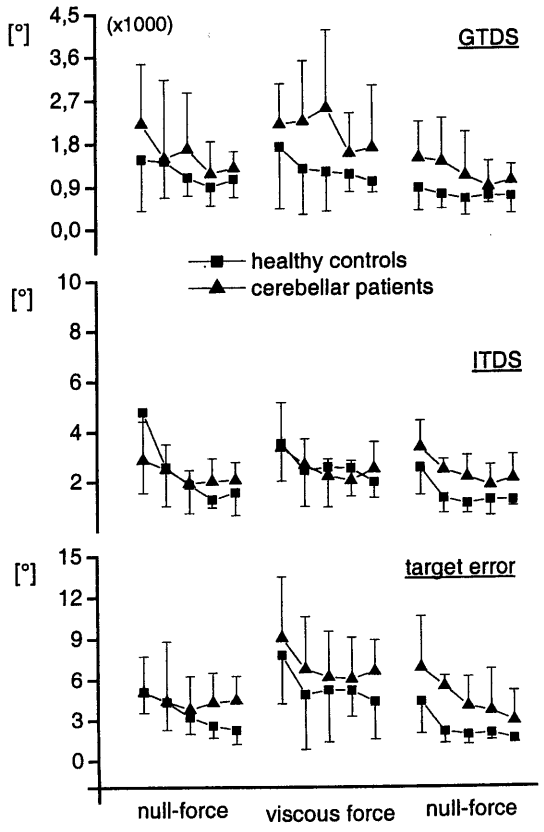


**FIGURE 6.** Learning. Group trajectory difference score (GTDS), individual trajectory difference score (ITDS), and target error in healthy controls (squares) and in cerebellar patients (triangles) in the three force conditions. Shown are the means and standard deviations for each block (= average of four trials). The figure represents the significant block effect, but we separated each group and force condition to illustrate the finding that learning did not differ between groups and force conditions.

main effect of block for target error, $F(4, 150) = 5.90$, $p < .0002$. Planned quantitative comparison revealed that for all variables, the first block (Trials 1–4) differed significantly from the following four blocks (Trials 5–20), which did not differ from one another. Thus, most learning took place in the first two blocks (Trials 1–8). There were additional significant differences between the first to third and the fourth to fifth block only for the ITDS. In other words, there were improvements in performance in later stages of the task (around Trial 12). Figure 6 shows the learning curves for both groups separately for each experimental condition.

### Analysis of Movement Velocity

Movements assisted by a negative viscous force were faster than were movements without external force; significant effects of force for maximum angular velocity, $F(2, 150) = 4.10$, $p < .0184$. Maximum angular velocity did not differ significantly between patients and controls ($M_{patient} = 126.2°/s$, $SD = 66.6°/s$; $M_{control} = 118.3°/s$, $SD = 24.7°/s$), $F(1, 150) = 1.04$, $p < .3103$. No interaction reached significance.

## Discussion

In summary, the main findings were that cerebellar patients learned to move in novel dynamic environments and that their performances were less precise than were those of controls, especially in the force condition.

### Learning to Compensate Novel Dynamics Is Not Abolished in Cerebellar Patients

We found that both cerebellar patients and healthy controls were able to adapt their motor performance within the first two blocks (Trials 1–8) after being exposed to a new negative viscous force; their ability to adapt was evidenced by decreases in individual and group trajectory difference scores (ITDS and GTDS) and in target errors. Although the cerebellar patients in our study adapted to novel arm dynamics, their kinematic performance was generally poorer than that of the healthy control group. That finding is in line with the results of the abundant body of research documenting motor deficits in cerebellar patients (e.g., for reviews, see Thach et al., 1992; Timmann & Diener, 1998).

We observed a sample of cerebellar patients who were able to increase several measures of motor performance over blocks (see Effects of Learning section). Those results are in agreement with earlier findings of improvement in the performance of cerebellar patients in a visuomotor task (Timmann et al., 1996). In contrast with the present results, impaired motor learning in cerebellar patients has been reported in the majority of experiments (Deuschl et al., 1996; Hesslow & Yeo, 1998; Lang & Bastian, 1999; Martin et al., 1996; Sanes et al., 1995).

Although the present findings may indicate that the cerebellum is not critically involved in motor adaptation, there are other possible explanations. First, all of the present patients had long-standing disease and may have acquired compensatory strategies. For example, if the cerebellar patients had performed slower arm movements, then they would have been exposed to lower novel viscous (i.e., velocity dependent) forces. Although compensatory mechanisms cannot be excluded, the absence of significant group differences in arm movement velocities argues against that possibility. Another explanation is that the group of cerebellar patients was too small and heterogeneous to show significant group differences. In some patients, critical cerebellar areas for that type of motor adaptation may have been preserved, or the degree of impairment was not severe enough to evoke significant deficits. Although the present results should be confirmed in a larger group of more severely affected cerebellar patients, the diffuse nature of the disease in all but one patient and the presence of upper limb ataxia in all patients argues against patient population as the main explanation for the preserved adaptation.

A probable explanation for the preserved adaptive ability is that the tested single-joint movements posed little challenge to the motor control system. Thus, learning could take place because the remaining cerebellar networks or networks outside the cerebellum were sufficient to deal with the changes in the force environment. Given that one underlying reason for the observed ataxia in cerebellar patients may be an inability to produce appropriate muscle torques to compensate for interaction joint torques in multijoint movements (Bastian, Zackowsky, & Thach, 2000; Topka, Konczak, & Dichgans, 1998) and given that such interaction torques do not arise in single-joint movements, learning may have been possible for the patients because the viscous force alone, and no additional interaction forces, had to be compensated.

In addition, although no learning deficits based on block effects were found, the analysis of ITDS indicated less extensive adaptation in patients. Cerebellar patients reached the level of the first null-force condition again in the second null-force condition, whereas control participants still improved their performance. The fact that ITDS represented the trajectory difference as compared with the individual baseline means that control participants' trajectories became increasingly similar to the baseline, whereas that was not true for the cerebellar patients. That finding is an expression of the high intraindividual variability found in cerebellar patients.

There are several learning mechanisms that could lead to successful force adaptation in humans. First, humans could adapt to the external negative viscous force by cocontraction of antagonistic muscles, thus enhancing limb stiffness (Latash, 1992; Latash & Gottlieb, 1991). Alternatively, learning could take place by rote memorization (Conditt, Gandolfo, & Mussa-Ivaldi, 1997). Finally, the relationship between the viscous load and the contraction of the muscles can be learned and generalized; thus, an inverse dynamic model is formed (Shadmehr & Mussa-Ivaldi, 1994). It is difficult to decide which mechanism accounted for the learning in the cerebellar and the healthy groups because they may differ in their learning mechanisms (Dancause et al., 2002). We did not record electromyographical activity to analyze cocontraction of antagonistic muscles; nor did we test for generalization of learning.

### Neural Estimation of Limb Dynamics Is Impaired in Cerebellar Patients

In fast goal-directed activities, the first movement unit (first acceleration and deceleration phase) is considered to be under feedforward control, that is, it is the behavioral expression of some internally specified kinematic plan. After the first movement unit, the kinematics can be jointly influenced by feedforward as well as feedback processes. We measured the amount of path oscillations after the first movement unit and found that trajectory oscillations were disproportionately increased in patients when the negative viscous force was present.[1] The observation of increased path oscillations in cerebellar patients is in agreement with the classical notion of an intention tremor, which is seen clinically as one outstanding symptom of cerebellar damage (Hore et al., 1991; Topka et al., 1999). There are electrophysiological findings showing that cerebellar tremor is caused by an absence of preprogrammed or predictive antagonistic muscle activity that stabilizes the limb at the end position (Flament & Hore, 1988). The antagonistic response usually emerges before the onset of muscle stretch. Thus, it is not driven by stretch reflexes. The same mechanism accounts for active position holding: A perturbation to limb position produces a reflex response in the stretched muscle that is followed by later bursts of activity in the antagonist muscle and so compensates for the overshoot. A succession of stretch reflexes cannot explain the agonist–antagonist response, but it is very likely preprogrammed and mediated by the cerebellothalamocortical circuits (Flament & Hore, 1986; Hore & Flament, 1986; Timmann et al., 2000). It is thought that when that predictive activity is absent, compensatory activities become driven by spinal and transcortical reflex activity. To complicate matters, not only do cerebellar patients seem to be impaired in predictive activity, their stretch reflexes are also often abnormal, which could further impair limb stabilization in those patients (Vilis, Hore, Meyer-Lohmann, & Brooks, 1976).

Therefore, the increase in terminal oscillations in cerebellar patients can result from deficient feedforward control or impaired feedback processes, alone or in combination. The following question remains, however: Why did the oscillations increase disproportionately when the viscous force was present? According to the systems view, the application of the unknown viscous force requires an update of the inverse dynamic model. Such an update was unnecessary in the null-force condition, yet terminal oscillations were also seen when no bias force was present. In that scenario, inherent time delays in feedback loops and impaired stretch reflexes may account for cerebellar patients' difficulties in controlling the braking process. In addition, an ill-parameterized inverse dynamic model would lead to a greater spatial error in the early, purely feedforward-controlled phase of the movement. We found that such error increased in the patient population once the negative viscous bias force was applied, which clearly indicates that the feedforward process was also deficient.

Visuomotor processing was an additional contributor to the performance deficits in the patient group. The task required a sensorimotor transformation from the initial and intended final positions of the arm presented on the screen in extrinsic space to the motion of the arm in joint coordinates. In several experiments, cerebellar patients have been found to be impaired in tasks requiring visuomotor control, such as tracking movements (Beppu, Suda, & Tanaka, 1984; Miall, Weir, & Stein, 1987) or the coordination of eye and arm (Brown, Kessler, 1998; Day et al., 1998; Glickstein & Buchbinder, 1998) or leg movement (Marple-Horvart & Stein, 1987). Those impairments may be attributable to an imprecise internal forward model (cf. Timmann et al., 2000; Vercher & Gauthier, 1988), and one cannot exclude the possibility that deteriorated visuomotor transformation was part of the difficulties in performing movements accurately seen in our cerebellar patients (Glickstein, 1998; Stein & Glickstein, 1992).

## Conclusion

Cerebellar patients were more challenged than healthy adults when performing goal-directed forearm movements under the influence of an external negative viscous force. Their already poorer endpoint accuracy degraded even further, and the degree of terminal oscillations increased once they were exposed to negative viscous force. Those findings suggest that motor performance in cerebellar patients is impaired in two ways: first, through a neural controller consisting of an inverse dynamic model that only coarsely reflects the actual limb dynamics, and second, by an impaired feedback-system. However, both cerebellar patients and healthy controls were able to adapt to a novel force over the course of not more than eight trials.

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## NOTE

1. The oscillation index may have turned out to be smaller if we had computed movement termination by using a velocity criterion. Yet, the Group × Force interaction effect was so strong that we believe the result would likely have remained the same.

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