Prism Adaptation During Walking Generalizes to Reaching and Requires the Cerebellum

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Morton, Susanne M. and Amy J. Bastian. Prism adaptation during walking generalizes to reaching and requires the cerebellum. J Neurophysiol 92: 2497–2509, 2004. First published June 9, 2004; 10.1152/jn.00129.2004. Adaptation of arm movements to laterally displacing prism glasses is usually highly specific to body part and movement type and is known to require the cerebellum. Here, we show that prism adaptation of walking trajectory generalizes to reaching (a different behavior involving a different body part) and that this adaptation requires the cerebellum. In experiment 1, healthy control subjects adapted to prisms during either reaching or walking and were tested for generalization to the other movement type. We recorded lateral deviations in finger endpoint position and walking direction to measure negative aftereffects and generalization. Results showed that generalization of prism adaptation is asymmetric: walking generalizes extensively to reaching, but reaching does not generalize to walking. In experiment 2, we compared the performance of cerebellar subjects versus healthy controls during the prism walking adaptation. We measured rates of adaptation, aftereffects, and generalization. Cerebellar subjects had reduced adaptation magnitudes, slowed adaptation rates, decreased negative aftereffects, and poor generalization. Based on these experiments, we propose that prism adaptation during whole body movements through space invokes a more general system for visuomotor remapping, involving recalibration of higher-order, effector-independent brain regions. In contrast, prism adaptation during isolated movements of the limbs is probably recalibrated by effector-specific mechanisms. The cerebellum is an essential component in the network for both types of prism adaptation.

INTRODUCTION

Visuomotor adaptation is the process of recalibrating motor behavior based on changes in visuospatial perception to maintain accuracy of movement. When wearing laterally displacing prism glasses, movements made toward visual targets are initially displaced to the same extent and direction as the visual shift induced by the prisms. With trial-and-error practice, the erroneous displacements are gradually corrected (the adaptation). When the prisms are removed, movements are initially displaced in the direction opposite from the prism visual shift (the negative aftereffect), indicating storage of the adaptation (Shadmehr and Mussa-Ivaldi 1994; Weiner et al. 1983). Prism adaptation can result from visual, proprioceptive, and/or motor recalibrations (Harris 1963; Welch et al. 1974). Visual recalibration involves an adjustment of the felt position of the eye relative to the head; proprioceptive recalibration involves an adjustment of the felt position of the moving limb relative to the head; motor recalibration involves an adjustment of the specific pattern of muscle activations used to generate the movement (Baraduc and Wolpert 2002; Martin et al. 1996b; Welch et al. 1974).

Most studies of prism adaptation have focused on isolated voluntary limb movements. During various types of arm movements, prism adaptation appears to involve a specific recalibration of either arm proprioception (Harris 1963) or arm motor commands (Baraduc and Wolpert 2002; Martin et al. 1996b). Evidence for this comes from the fact that prism adaptation during arm movements is usually highly specific to the exposed arm. Many studies have shown that prism negative aftereffects are very small or absent when reaching (Baily 1972; Baizer et al. 1999) or throwing (Martin et al. 1996b) with the unexposed arm. In some cases the adaptation has even been reported to be specific to the particular type of arm movement exposed: prism adaptation during throwing shows little generalization from an overhand to an underhand throw (Martin et al. 1996b), and a similar visuomotor adaptation during reaching does not show full generalization with changes in arm posture (Baraduc and Wolpert 2002). Likewise, these types of adaptations can also be specific to the exposed movement velocity (Kitazawa et al. 1997).

Prism adaptation during whole body movements such as walking might involve sensorimotor recalibration of a different brain region and/or multiple brain regions. As such, these forms of prism adaptation may or may not have the same degree of specificity as those of single limb movements. Unfortunately, few studies have addressed prism adaptation during more complex movements involving multiple body parts. Healthy subjects are known to adapt their walking trajectory to prism glasses (Harris and Carre 2001; Redding and Wallace 1985a,b, 1987, 1988b; Rushton et al. 1998), but the specificity of this adaptation has not been thoroughly examined. Similarly, it is not clear whether walking prism adaptations utilize visual, proprioceptive, and/or motor recalibrations (cf. Mikaelian 1970; Redding and Wallace 1987, 1988b). Visual recalibrations, unlike proprioceptive and motor recalibrations, would lead to broad generalizability across unexposed movements and body parts, because an alteration in the felt position of eyes-in-head would seemingly influence any subsequent visually guided movement. Another (not mutually exclusive) possibility is that walking prism adaptations recalibrate different brain regions that encode higher-level motor...
parameters, which could also bias the adaptation toward increased generalizability.

Focal cerebellar damage is known to impair prism adaptation of many types of arm movements (Baizer et al. 1999; Deuschl et al. 1996; Martin et al. 1996a; Weiner et al. 1983), suggesting that the cerebellum is necessary for the specific recalibration of arm proprioception or arm motor commands. It is not known whether the cerebellum is involved in the types of adaptations that show broader generalization across movement types. The extent to which the cerebellum is important for locomotor adaptations is also not well understood. The spinal cord is believed to contain sufficient circuitry to produce, and at least to some extent, modify locomotor patterns (Brown 1911; Forssberg and Grillner 1973; Forssberg et al. 1975; Grillner 1975). However, it is likely that more complicated and longer-term adaptations require supraspinal structures such as the brain stem, motor cortex, and/or cerebellum (Armstrong 1986; Grillner and Wallen 1985). In a study of decerebrate cats, Yanigahara and Kondo (1996) found that nitric oxide deprivation in the cerebellum vermis abolished adaptation of the locomotor pattern to a splitbell treadmill. Nitric oxide blockage prevents long-term depression of cerebellar parallel fiber-to-Purkinje cell synapses, which are thought to be responsible for many forms of cerebellar motor learning. In humans, some evidence suggests that patients with cerebellar damage can adapt simpler forms of locomotion, e.g., predictable changes in treadmill speed during walking. However, they still show considerable variability in the timing and duration of muscle activation patterns compared with control subjects (Rand et al. 1998). In another study, it was found that cerebellar patients have a diminished aftereffect following adaptation to walking in place on a rotating disc (Earhart et al. 2002), which alters the proprioceptive gain between the trunk and stance limbs. This indicates a potential role for the cerebellum in storing novel locomotor adaptations based on proprioception.

The purpose of this study was to compare the specificity (generalizability) of reaching versus walking prism adaptations and to determine the role of the cerebellum in the prism adaptation of walking trajectories in humans. In a control study (experiment 1), we tested one group of subjects performing walking after adapting to prisms during reaching; we tested a second group of subjects performing reaching after adapting to prisms during walking. We also tested these subjects for evidence of visual and proprioceptive shifts. In a second study (experiment 2), we compared the ability of control versus cerebellar subjects to adapt to prisms during walking.

METHODS

Subjects

A total of 43 subjects participated in the study. In experiment 1, we tested 10 healthy subjects pseudo-randomly assigned to one of two groups. Group 1 consisted of three females and two males [mean age, 36.40 ± 4.34 (SE) yr]; group 2 consisted of two females and three males (mean age, 47.00 ± 5.87 yr). Nine additional healthy subjects (3 females and 6 males; mean age, 38.22 ± 4.21 yr) later participated in one of three variations of experiment 1 to clarify specific effects. In experiment 2, we tested 12 subjects with cerebellar damage (4 females and 8 males; mean age, 41.17 ± 5.60 yr) and an additional 12 healthy age- and gender-matched controls (4 females and 8 males; mean age, 46.08 ± 3.98 yr). All subjects gave informed consent prior to participating, and a Human Studies Committee approved the study.

For subjects in the cerebellar group, cerebellar damage was confirmed by MRI or computed tomography (CT) scan, and localization to a specific zone was determined when possible. Prior to testing, these subjects also underwent a thorough motor neurological examination. Subjects with clinical evidence of involvement of other brain structures (e.g., motor weakness, sensory loss, hyperreflexia, bradykinesia, rigidity) were excluded from the study (see Table 1 for cerebellar subject information). Additional detailed information regarding subject diagnoses is provided below.

Four of the 12 subjects had focal cerebellar damage, caused either by stroke or tumor resection (CBL-4, -7, -10, -11); the rest had diffuse cerebellar damage. MRI reports for these four subjects showed that the damage was restricted to the cerebellum. The two subjects who had tumors also underwent postsurgical chemotherapy and radiation. Seven subjects had diffuse cerebellar degeneration that was either an autosomal dominant spinocerebellar ataxia (CBL-5, -6, -12), or idiopathic in origin (CBL-1, -2, -3, -9). None of these subjects had evidence of involvement of any brain region beyond the cerebellum either clinically or by radiological report, none had any sign of

TABLE 1.  Cerebellar subject information

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age</th>
<th>Gender</th>
<th>Diagnosis</th>
<th>Onset</th>
<th>Post and Gait (34)</th>
<th>Limbs (52)</th>
<th>Speech (8)</th>
<th>Eyes (6)</th>
<th>Total (100)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CBL-1</td>
<td>57</td>
<td>M</td>
<td>Pancerebellar atrophy</td>
<td>4</td>
<td>10</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>40</td>
</tr>
<tr>
<td>CBL-2</td>
<td>68</td>
<td>M</td>
<td>Pancerebellar atrophy</td>
<td>7.5</td>
<td>8</td>
<td>1</td>
<td>0</td>
<td>3</td>
<td>12</td>
</tr>
<tr>
<td>CBL-3</td>
<td>51</td>
<td>F</td>
<td>Pancerebellar atrophy</td>
<td>8.5</td>
<td>9</td>
<td>14</td>
<td>4</td>
<td>4</td>
<td>31</td>
</tr>
<tr>
<td>CBL-4</td>
<td>73</td>
<td>M</td>
<td>R PICA, R SCA, L PICA strokes</td>
<td>5</td>
<td>5</td>
<td>3</td>
<td>4</td>
<td>3</td>
<td>15</td>
</tr>
<tr>
<td>CBL-5</td>
<td>34</td>
<td>F</td>
<td>SCA6</td>
<td>3</td>
<td>9</td>
<td>19</td>
<td>2</td>
<td>5</td>
<td>35</td>
</tr>
<tr>
<td>CBL-6</td>
<td>39</td>
<td>F</td>
<td>SCA2</td>
<td>1.5</td>
<td>2</td>
<td>10</td>
<td>0</td>
<td>1</td>
<td>13</td>
</tr>
<tr>
<td>CBL-7</td>
<td>29</td>
<td>F</td>
<td>Post-fossa medulloblastoma resect</td>
<td>13</td>
<td>7</td>
<td>18</td>
<td>1</td>
<td>4</td>
<td>30</td>
</tr>
<tr>
<td>CBL-8</td>
<td>44</td>
<td>M</td>
<td>Pancerebellar atrophy with dystonia</td>
<td>24</td>
<td>7</td>
<td>24</td>
<td>2</td>
<td>3</td>
<td>35</td>
</tr>
<tr>
<td>CBL-9</td>
<td>44</td>
<td>M</td>
<td>Pancerebellar atrophy</td>
<td>24</td>
<td>12</td>
<td>20</td>
<td>3</td>
<td>2</td>
<td>37</td>
</tr>
<tr>
<td>CBL-10</td>
<td>8</td>
<td>M</td>
<td>L SCA ischemic stroke</td>
<td>3</td>
<td>3</td>
<td>10</td>
<td>0</td>
<td>1</td>
<td>14</td>
</tr>
<tr>
<td>CBL-11</td>
<td>16</td>
<td>M</td>
<td>R lat cerebellar astrocytoma resect</td>
<td>0.5</td>
<td>4</td>
<td>6</td>
<td>0</td>
<td>2</td>
<td>12</td>
</tr>
<tr>
<td>CBL-12</td>
<td>31</td>
<td>M</td>
<td>SCA8</td>
<td>5</td>
<td>10</td>
<td>15</td>
<td>2</td>
<td>3</td>
<td>30</td>
</tr>
</tbody>
</table>

ICARS, score on International cooperative ataxia rating scale; PICA, posterior inferior cerebellar artery; SCA, superior cerebellar artery; SCA2, SCA6, SCA8, spinocerebellar ataxia types 2, 6, or 8. Onset refers to length of time since symptom onset. Age and onset are given in years. ICARS scores are provided for each of the four categories of movement impairment: posture and gait, limb kinematics, speech, and ocuulmotor. Highest possible scores in each category are indicated in parentheses.
autonomic system dysfunction, and none with idiopathic degeneration had a family history of similar symptoms. Subject CBL-8 had idiopathic cerebellar degeneration but also presented with mild hand dystonia that did not interfere with reaching. It was unclear whether the dystonia was related to the cerebellar disease or a separate phenomenon, because other clinical signs were consistent with a relatively pure cerebellar dysfunction. Because the only extracerebellar sign was the focal dystonia, this subject was included in the study.

Part of the motor neurological examination consisted of rating the severity of ataxia using the International Cooperative Ataxia Rating Scale (ICARS). The ICARS is an ordinal scale clinical measure that quantifies ataxia in four categories of movement: posture and gait, limb kinetics, speech, and eye movements (Trouillas et al. 1997). Scores range from 0 (no ataxia) to 100 (most severe ataxia, e.g., unable to stand or walk). Based on this scale, the cerebellar group had relatively mild ataxia on average (see Table 1).

Paradigm

In experiment 1, we compared the extent to which prism reaching adaptations generalized to walking versus the extent to which prism walking adaptations generalized to reaching. Subjects in group 1 adapted to prisms during reaching; subjects in group 2 adapted to prisms during walking. In experiment 2, we compared the performance of control subjects versus patients with cerebellar damage during the prism walking adaptation and subsequent generalization to reaching.

Figure 1 shows the experimental setup. For reaching, subjects stood facing a rectangular panel and made single movements with the index finger toward a target on the panel. The target was a small “X” (3.2 × 3.2 cm) located directly in front of the shoulder, at ~90% of arm’s length. For walking, subjects walked with arms across their chest (to ensure no arm motion) within boundary lines (45 cm apart, 120 cm long) marked on the floor of the open laboratory space. Prior to each trial, subjects were positioned ~100 cm in front of and directly between the right and left boundary lines. In both tasks, subjects were instructed to move at a fast but comfortable pace.

All subjects participated in baseline, adaptation, generalization, and postadaptation phases. In the baseline phase, subjects were asked to look at the target (reaching) or the boundary lines on the walkway (walking), close their eyes, and either reach to the target or walk between the lines with eyes closed. After each trial, subjects were allowed to open their eyes and view their performance (i.e., view their index finger position relative to the target or their body position relative to the boundary lines). Subjects performed three to eight trials of both types of movements. In the adaptation phase, subjects wore 30-dioptr prism (Fresnel Prism and Lens, Eden Prairie, MN) mounted base left onto standard safety goggles, inducing a visual field shift of −17° to the right. Subjects were allowed to view the target (or walkway) and their body during the entire adaptation period. Subjects performed 25 trials of the instructed movement (either reaching or walking but not both), except for a few subjects who, because it was clear they had reached a plateau in performance, completed fewer trials (subject CNT-6, 15 trials; subject CNT-2, 20 trials; and subject CBL-6, 22 trials). In the generalization phase, subjects removed the prism glasses and performed a single trial of the unexposed movement with eyes closed (i.e., subjects who performed reaching during the adaptation phase were tested walking; subjects who performed walking during the adaptation phase were tested reaching). In the postadaptation phase, subjects performed 8–15 trials of the adapted (exposed) movement with eyes closed. Table 2 shows the testing order for subjects in each experiment.

In addition to the reaching and walking movements, subjects in experiment 1 also performed visual and proprioceptive shift tests (Redding and Wallace 1994, 1988a,b). The visual shift test examined the degree to which the prism adaptation caused a shift in the perceived direction of straight ahead or the felt position of the eyes relative to the head. Subjects were seated in total darkness, facing a wall ~5 m in front of them, with the trunk and head aligned and oriented straight ahead. A light shined on the wall from behind and was passed in front of the field of view. Subjects were instructed to indicate when they believed the light to be directly in front of them and were allowed to verbally ask for adjustments (move left or right). If the prisms induced a visual shift, subjects would subsequently identify a light shined to the right of center in total darkness as the perceived visual straight ahead. This would occur because, after wearing rightward shifting prisms, rightward eye (relative to the head) positions become perceived as straight ahead (Redding and Wallace 1994, 1988a,b). A rightward visual shift would induce leftward aftereffects during movement because, when the eyes are fixated on a target directly in front of them (eye-in-head angle, 0°), subjects perceive the target as leftward and therefore direct their movements to the left. The proprioceptive shift test examined the degree to which the prism adaptation caused a shift in the felt position of the arm relative to the head. Subjects were seated with eyes closed, holding their arm out laterally with index finger extended. When instructed, subjects actively moved their extended arm to the front of their body until they felt their index finger to be exactly in front of their nose. The arm used was the same as that used during reaching. In contrast to the visual shift test, if the prisms induced a proprioceptive shift, subjects would subsequently place their finger to the left of their nose when asked to identify the felt position of the head relative to the hand. This would occur because, after wearing rightward shifting prisms, straight ahead arm (relative to the head and/or trunk) positions became felt as rightward (Redding and Wallace 1994, 1988a,b). A leftward proprioceptive shift would induce leftward aftereffects during movement because, when subjects orient the body to a target directly in front of them (head, trunk angles, 0° relative to the target), subjects feel leftward arm positions to be straight ahead and therefore direct their movements to the left. Subjects performed five trials of both visual and proprioceptive shift tests in the baseline phase (prior to the reaching and walking baseline) and a single trial of both tests after the adaptation phase but before the generalization phase (see Table 2).

We conducted further testing in experiment 1 with nine other healthy control subjects. Groups 1b and 1c (3 subjects/group) performed the prism reaching adaptation (similar to subjects in group 1).
with different modifications to control for factors that could have affected the extent of generalization. First, since each walking adaptation trial was longer than each reaching adaptation trial, we tested control subjects (group 1b) adapting to prisms during slow reaching (paced to a metronome). The metronome was set so that each reach would take as long or longer to complete than each walking trial (∼4 s). Second, we tested another set of control subjects (group 1c) adapting to prisms during normal-speed reaching, but for an extended number of trials. These subjects performed additional reaching adaptation trials until the total exposure time to prisms was equal to or greater than the average total exposure time experienced by subjects performing the prism walking adaptation (∼7 min). Subjects in the third group (group 2b) performed the prism walking adaptation in a manner identical to that of subjects in group 2 of experiment 1, but we recorded EMGs from this group and group 1c. This was done to determine the extent of arm muscle activity during walking and leg muscle activity during reaching. Although there were no overt arm movements during (crossed-arm) walking and no overt leg movements during (standing) reaching, we wanted to determine whether significant arm or leg muscle activity could have been related to the extent of generalization. That is, arm muscle activity during walking might assist in the generalization from walking to reaching. Likewise, leg muscle activity during reaching might assist in the generalization from reaching to walking.

Data collection

Two OPTOTRAK (Northern Digital, Waterloo, Ontario, Canada) sensors, one on each side of the body, were used to record position data. We placed 12 infrared emitting diodes on the head (center, right, and left sides), shoulders, index finger, pelvis, ankles, and feet laterally to record body segment positions and rotations in the horizontal dimension (about the long-axis of the body). We defined the coordinates of our laboratory space such that forward-backward movements occurred in the x-direction, vertical movements in the y-direction, and lateral movements in the z-direction (see Fig. 1). Position data were collected at 100 Hz.

For subjects in groups 1c and 2b, EMGs were recorded from surface electrodes using an MA-300 EMG System (Motion Lab Systems, Baton Rouge, LA). Recordings were made from the posterior deltoid, the long head of the biceps, the lateral head of the triceps, the anterior tibialis, and the medial gastrocnemius muscles, bilaterally. We selected the three arm muscles based on previous work that indicated that these particular muscles are modulated with the step cycle during walking while swinging the arms (Dietz 2002; Dietz et al. 2001). EMG data were collected at 1,000 Hz and time-synched with the position data.

Data analysis

Performance was measured by calculating the lateral (left-right) errors in index finger endpoint location (reaching) and the maximal lateral deviations of the walking path, measured at the level of the pelvis (walking), for all trials in each testing phase. Lateral deviations were converted to degrees using the following method. For reaching, we calculated the distance between target location and index finger endpoint location in the medial-lateral dimension (z-dimension in laboratory coordinates) and the distance between target location and each subject’s standing location in the anterior-posterior dimension (x-dimension). The angle of shift was calculated as the inverse tangent of the ratio of the z- to x-distances. For walking, we first generated the walking path for each subject by calculating the average location of the right and left pelvis markers for every sample in time. We calculated the distance between the maximal lateral deviation of the walking path and the exact midpoint of the walkway in the medial-lateral dimension (z-dimension). We also calculated the distance between the maximal lateral deviation of the walking path and each subject’s standing location in the anterior-posterior dimension (x-dimension). As with reaching, the angle of shift was calculated as the inverse tangent of the ratio of the z- to x-distances. In both cases, positive and negative angles were assigned to indicate rightward and leftward deviations, respectively. We compared performance of the exposed movement at several time periods: 1) the baseline phase (average of all trials), 2) the early adaptation phase (1st trial), 3) the late adaptation phase (average of last 5 trials), and 4) the early postadaptation phase (1st trial). We also compared performance of the unexposed movement at two time periods: 1) the baseline phase (average of all trials) and 2) the generalization phase (single trial).

We calculated rotations in the horizontal plane (the xz-plane, or as seen in an overhead view, see Fig. 1) for several segments. Head-on-trunk angles were calculated using the positions of the right and left head markers relative to the right and left shoulder markers; trunk-on-legs angles were calculated using the positions of the shoulder markers relative to the pelvis markers; legs-on-feet angles were calculated using the positions of the pelvis markers relative to the ankle markers. The feet-on-floor angles were calculated using the orientations of the foot and ankle markers relative to the target location (reaching trials) or the exact midpoint of the walkway (walking trials), which were designated to be at 0° rotation. Positive and negative values indicated rightward and leftrightward rotations, respectively. These measures allowed us to determine whether there was a systematic rotation at any segment(s) that could explain negative aftereffects or generalization. We recorded rotations at the start of each movement.

In experiment 1 only, we also compared the magnitudes of visual and proprioceptive shifts before and after prism exposure. For the visual shifts, subjects were positioned sitting in a chair so that the head, shoulders, and pelvis were in alignment with each other and with the chair, and the chair was positioned so that its back was parallel to the wall (i.e., head-on-trunk, trunk-on-legs, and legs-on-shoulders angles were all 0°). An examiner marked the locations on the wall that subjects indicated as “straight ahead.” We calculated the distance between the center head marker and the identified straight ahead location on the wall in the medial-lateral dimension (z-dimension).

<table>
<thead>
<tr>
<th>Testing paradigm</th>
<th>Baseline</th>
<th>Adaptation</th>
<th>Generalization</th>
<th>Postadaptation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>dark</td>
<td>ec</td>
<td>ec</td>
<td>ec</td>
</tr>
<tr>
<td>E1, G1:</td>
<td>1. vis</td>
<td>2. prop</td>
<td>3. walk</td>
<td>4. reach</td>
</tr>
<tr>
<td>E1, G2:</td>
<td>1. vis</td>
<td>2. prop</td>
<td>3. reach</td>
<td>4. walk</td>
</tr>
<tr>
<td>E2:</td>
<td>—</td>
<td>—</td>
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<td>2. walk</td>
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<tr>
<td>E2:</td>
<td>—</td>
<td>—</td>
<td>1. reach</td>
<td>2. walk</td>
</tr>
</tbody>
</table>
| Testing order (numbered in order) and vision condition for each group of subjects. E1, G1, subjects in experiment 1, group 1; E1, G2, subjects in experiment 1, group 2; E2, subjects in experiment 2; dark, in total darkness; ec, eyes closed; eo, eyes open; vis, visual perceptual shift test; prop, proprioceptive perceptual shift test.
sion) and the distance between the chair and the wall in the anterior-posterior dimension (x-dimension). For the proprioceptive shifts, we calculated the distance between the center head marker and the index finger location in the medial-lateral dimension (z-dimension) when subjects tried to align the hand with the nose. We also calculated the distance between head location and index finger location in the anterior-posterior dimension (x-dimension). For both tests, angles of shift were calculated as the inverse tangent of the ratio of the z- to x-distances. Positive and negative angles were assigned to indicate rightward and leftward deviations, respectively. We compared performance on the shift tests at 1 the baseline phase (average of all trials) and 2) just after the adaptation phase (single trial).

EMG signals were preamplified at the electrode site with a gain of x20. Signals were digitized, amplified, and band-pass filtered (20 Hz high-pass and 500 Hz low-pass) from a remote amplifier. Off-line, EMG signals were notch filtered to remove any noise in the 60-Hz range. Data were rectified and low-pass filtered with a fourth-order Butterworth at 50 Hz. To quantify amplitudes of muscle activity, we normalized each muscle’s EMG signal during reaching and walking movements to its peak EMG amplitude during an isometric maximal voluntary contraction (MVC). We then expressed muscle activity as a percentage of MVC.

In experiment 1 only, we also measured rates of adaptation. We fit exponential functions to plots of the maximal lateral walking deviation versus trial number during the adaptation phase for each subject (see Fig. 5 for examples). The exponential decay constants were used as approximations of the adaptation rate (Deuschl et al. 1996a; Martin et al. 1996a).

All data were analyzed using custom MATLAB (Mathworks, Natick, MA) software. Statistical comparisons were completed using CoStat (CoHort Software, Minneapolis, MN) and Statistica (StatSoft, Tulsa, OK) software. Performance (lateral deviations during reaching and walking) and body segment rotations were compared using 2 × 2 (group × testing phase) repeated measures ANOVA. When the ANOVA yielded significant results, post hoc analyses were done using Tukey’s honest significant different test. Adaptation rates were compared using Student’s t-test for independent samples. The level of statistical significance for all measures was set at P < 0.05.

RESULTS

Experiment 1

In experiment 1, we tested whether a prism adaptation in healthy subjects would generalize from reaching (performed while standing) to walking or from walking (performed without any arm motion) to reaching. Subjects in group 1 practiced reaching while wearing laterally displacing prisms; subjects in group 2 practiced walking.

Subjects performed equally well during the baseline phase (eyes closed) reaching and walking movements and were accurate to within a few degrees in the lateral dimension. In the adaptation phase (prisms on), all subjects showed an initial rightward deviation in their reaching endpoint (group 1) or walking direction (group 2), which gradually improved with practice to near-baseline levels. When the prisms were removed in the postadaptation phase (eyes closed), all subjects also showed a clear negative aftereffect with the trained movement, a leftward deviation in reaching endpoint (group 1) or walking direction (group 2), indicating that both groups stored their respective adaptations. However, only the group that adapted walking showed generalization to reaching; the opposite did not occur. Figure 2, A and C, shows reaching and walking trajectories from selected trials from two typical subjects, one each from groups 1 and 2. The subject in group 1 showed no generalization to walking after prism exposure during reaching. However, the subject from group 2 showed clear generalization, as indicated by the substantial leftward deviation in reaching endpoint after adapting to prisms during walking.

The group data showed similar results: subjects who adapted during reaching had no generalization to walking, but subjects who adapted to prisms during walking showed robust generalization to reaching. Figure 3, A and B, shows average reaching and walking deviations from groups 1 and 2 for key time periods during the adaptation and generalization. During the generalization phase, there were clear differences between the two groups. Average walking deviation after the reaching adaptation was 1.32 ± 1.02° left, not significantly different from walking deviations prior to the reaching adaptation (post hoc P = 0.514). In contrast, average reaching deviation after the walking adaptation was 5.48 ± 1.24° left, significantly different from reaching deviations prior to the walking adaptation (post hoc P = 0.001). Thus only subjects who adapted walking (group 2) showed generalization. This finding is replicated in the larger sample of control subjects in experiment 2 (see Figs. 6, A and B, and 7).

The analysis of body segments failed to show any consistent rotations that could explain the generalization. Figure 4 shows the measured feet-on-floor (FoF), legs-on-feet (LoF), trunk-on-legs (ToL), and head-on-trunk (HoT) rotations for each of the subjects (each represented by a different symbol) in group 2 (walking adaptation). The resulting head-in-space (HiS) rotation, or the sum of FoF, LoF, ToL, and HoT angles, is also depicted. Figure 4 shows changes in each of the segmental rotations from the reaching baseline phase to the generalization phase for each subject. No subject showed a systematic leftward rotation across body segments that could explain the generalization from walking to reaching. Likewise, as a group, there were no differences in rotations between baseline and generalization phases for any of the body segments that could account for the leftward reaching endpoint errors (HiS, P = 0.092; HoT, P = 0.866; ToL, P = 0.341; LoF, P = 0.341; FoF, P = 0.798). The large inter- (shown) and intrasubject (not shown) variability in segmental rotations suggests that the generalization was achieved not through a systematic, fixed change in standing postural alignment (e.g., standing rotated relative to the target or with leftward trunk or head rotation, etc.), but rather through a fundamental coordinate remapping of the sensorimotor system.

Neither group showed significant differences in the proprioceptive or visual shift tests. After the prism reaching adaptation, group 1 showed an average proprioceptive shift of 1.03 ± 1.46° left and an average visual shift of 1.45 ± 1.46° left. After the prism walking adaptation, group 2 showed an average proprioceptive shift of 0.79 ± 1.66° left and an average visual shift of 0.78 ± 0.34° right. None of these shifts was significantly different from baseline measures (P > 0.10 for all). However, while none of the subjects in group 1 (reaching adaptation) had a rightward visual shift, four of the five
subjects in group 2 (walking adaptation) had a rightward visual shift.

Results from the additional groups of subjects in experiment 1 (groups 1b, 1c, and 2b) are shown in Fig. 3, C–E. Findings were similar to those from the main analysis. That is, neither group that adapted to prisms during reaching (groups 1b and 1c) had any appreciable generalization to walking. All three groups adapted and stored the practiced movements, but only subjects in group 2b (those who adapted during walking) showed any generalization to the other movement type. Just as the other five subjects (group 2) in the main portion of experiment 1, group 2b showed robust generalization from walking to reaching (difference between group 2b and groups 1b and 1c, post hoc $P < 0.05$). Neither increasing the movement duration (i.e., trial duration, group 1b) nor increasing the total time of exposure to prisms (i.e., adaptation phase duration, group 1c) appeared to increase the likelihood for generalization when the movement being adapted was reaching.

We also examined EMG from the arms and legs from subjects adapting during reaching (group 1c) and walking (group 2b). Figure 5 shows EMG amplitudes from leg and arm muscles during a typical walking adaptation trial from a subject in group 2b. During the walking adaptation (crossed-arm posture), these subjects showed some arm muscle activity that was minimal in magnitude and often tonic, as opposed to the phasic bursting from the leg muscles that was well-modulated according to the timing of the stride cycles. Arm muscle activity during walking was substantially reduced compared with that which occurred during reaching. Ratios of (walking-to-reaching) EMG activity averaged over the entire movement period were 0.52, 0.82, and 0.42 for the reaching arm posterior deltoid, biceps, and triceps muscles, respectively, where values of 1.0 indicate equal activity during the two movements and values lower than 1.0 indicate reduced arm muscle activity during walking compared with reaching. During reaching, leg and arm muscles were activated approximately equally (average arm muscle activity of 4.51/2.12% MVC vs. average leg muscle activity of 4.63/1.80%), whereas during walking, leg muscles were activated to a much greater extent than arm muscles (average arm muscle activity of 2.40/1.02% vs. average leg muscle activity of 8.69/1.34%).

**Experiment 2**

In experiment 2, we tested whether cerebellar damage impairs the prism adaptation during walking. The healthy subjects from this experiment served as comparison data for the subjects with cerebellar damage, as well as confirmation (with an independent and larger sample) of the findings from subjects in group 2 of experiment 1. Both control and cerebellar subjects performed similarly in the baseline phase, completing reaching and walking movements (eyes closed) to within a few degrees of error, laterally. In the adaptation phase, all subjects also showed an initial rightward deviation in the walking path. However, latter portions of the adaptation phase, as well as the generalization and postadaptation phases were strikingly dif-

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**FIG. 2.** A and B: results from a typical control subject from experiment 1, group 1 (generalization from reaching to walking). C and D: results from a typical control subject from experiment 1, group 2 (generalization from walking to reaching). A and C: overhead view of the reaching and walking paths for selected trials. Dashed vertical black lines indicate a perfectly straight trajectory. Target location (reaching) is indicated by the square box; boundary lines (walking) are indicated by the shaded rectangle. Note the scale changes between finger and walking paths. B and D: reaching and walking lateral deviations plotted vs. trial number for all trials from the same 2 subjects in A and C. Positive and negative values represent rightward and leftward angular deviations, respectively.

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different between the two groups. Figure 6, A and C, depicts reaching and walking trajectories from selected trials from a typical control and a cerebellar subject who has an idiopathic cerebellar degenerative disorder. Although the cerebellar subject improved his walking trajectory somewhat during adaptation, it can be seen that he did not return to baseline levels. Similarly, his leftward negative aftereffects during the postadaptation phase were reduced, and the generalization from walking to reaching was minimal. Figure 6, B and D, shows reaching and walking lateral deviations for all trials from the same two subjects. Compared with the control, the subject with cerebellar damage did not improve his walking trajectories to the same extent (i.e., walking trajectories did not return to near-baseline levels like the control). The cerebellar subject also took longer to show improvement in his walking trajectories. His adaptation rate, calculated by fitting an exponential function to the data, was slower than that of the control.

**FIG. 3.** Averaged data from subjects in experiment 1. A: data from group 1, generalization from reaching to walking. B: data from group 2, generalization from walking to reaching. C: data from group 1b, generalization from reaching to walking, with adaptation trial durations matched to that of groups 2 and 2b. D: data from group 2b, generalization from walking to reaching, with EMG recorded from arms and legs. D: data from group 1c, generalization from reaching to walking with reaching adaptation phase duration matched to that of groups 2 and 2b and EMG recorded from arms and legs. Mean lateral deviations are shown during baseline, adaptation, generalization, and postadaptation phases. B, average error over the entire baseline phase; A1, average error from the 1st trial of the adaptation phase; A, average error from the last 5 trials of the adaptation phase; P1, average error from the 1st trial of the (exposed movement) postadaptation phase; G1, average performance from the generalization trial (unexposed movement). Data represent group means ± SE. *Significant differences from baseline performance (P < 0.05) from the post hoc analysis.

**FIG. 4.** Segmental rotations from subjects in group 2 of experiment 1 (generalization from walking to reaching). Each graph depicts the relative horizontal rotation at a particular segment: (A) feet-on-floor; (B) legs-on-feet; (C) trunk-on-legs; (D) head-on-trunk; (E) head-in-space. For each relative rotation (each graph), there are 5 pairs of data points, with each pair (connected by a line) representing a single subject. On the left of each graph are individuals’ average rotations during the reaching baseline phase; on the right are individuals’ rotations during the reaching generalization trial. Positive and negative values represent rightward and leftward angular rotations, respectively.
A decay function to the data, was nearly three times that of the control subject. The cerebellar subject also showed a reduced walking negative aftereffect and essentially no generalization from walking to reaching, unlike the control subject.

Figure 7 shows group results from experiment 2. As a whole, cerebellar subjects had multiple indicators of impaired prism walking adaptation. Control and cerebellar groups performed similarly during the walking and reaching baseline phases, as well as during the initial trial of the adaptation phase. However, by the end of the adaptation phase, the cerebellar group showed less improvement in their walking deviations compared with controls (maximal walking deviation: control = $2.40 \pm 0.28^\circ$ right vs. cerebellar = $4.76 \pm 0.82^\circ$ right; post hoc $P = 0.017$). Similarly, the cerebellar group showed a reduced negative aftereffect during the initial trial of the postadaptation phase, suggesting diminished storage of the prism walking adaptation (walking negative aftereffect: control = $6.48 \pm 0.62^\circ$ left vs. cerebellar = $3.99 \pm 1.03^\circ$ left; post hoc $P = 0.012$). Cerebellar subjects also adapted more slowly than controls. Figure 8 shows rates of adaptation, derived from the exponential fits, for control and cerebellar groups. On average, cerebellar subjects took almost twice as long to adapt their walking direction to the prisms as controls (rates of adaptation: control = 4.57 $\pm$ 0.77 trials vs. cerebellar = 8.76 $\pm$ 1.83 trials; $P = 0.047$). As in experiment 1, the control group showed robust generalization from walking to reaching (see Fig. 7). The mean reaching endpoint error from the generalization trial was $8.94 \pm 3.51^\circ$ left (difference from baseline, post hoc $P =$...
Significant differences (f.i. generalization trial (unexposed movement). Data represent group means from the post hoc analysis.

- Adaptation phase
- 5 trials of the adaptation phase
- Error from the 1st trial of the adaptation phase
- G1, average performance from the generalization trial (unexposed movement). Data represent group means ± SE.

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To further investigate the issue of impaired generalization in the cerebellar group, we examined the relationship between walking negative aftereffects and the generalization to reaching. It was not surprising that we saw reduced generalization in the cerebellar group, given that they had reduced storage of the adaptation (i.e., decreased walking negative aftereffects). Still, the cerebellar group showed an unusually poor relationship between generalization and adaptation that we had not specifically predicted. Figure 9 shows the correlation between walking negative aftereffects and reaching generalization for control and cerebellar groups. For the control group, the strength of adaptation storage (i.e., the magnitude of the walking negative aftereffect) predicted the extent of generalization (significant positive correlation; r = 0.58, P = 0.047). The cerebellar group showed a small, nonsignificant negative correlation between these two variables (r = -0.35, P = 0.262). Therefore, for subjects with cerebellar damage, the strength of adaptation storage had no influence over the extent of generalization. For example, the best two adapters (who, notably, showed negative aftereffects well within the normal range) and the worst two adapters from the cerebellar group showed comparable (and reduced) amounts of generalization (see Fig. 9).

We also correlated cerebellar subjects’ walking adaptation performance to their ICARS scores. The ICARS is a 100-point ordinal scale clinical test that rates the degree of ataxia in four categories of movement (the 4 subscores): posture and gait, limb movements, speech, and eye movements. We calculated specific correlations to determine whether the walking adaptation was best predicted by the overall degree of ataxia and/or by deficits of specific movement types. We examined the correlations between each ICARS score (posture and gait subscore, limb movement subscore, speech subscore, oculomotor subscore, and total score) and performance during the adaptation, measured by the extent of adaptation (the mean maximal lateral deviation from the last 5 trials of the adaptation phase) and the rate of adaptation (derived from the exponential fits). The ICARS posture and gait subscore was significantly correlated with the extent of adaptation (r = 0.655, P = 0.021). The positive correlation indicated that with worsening posture and gait (increased scores), the walking path remained laterally deviated, even after extensive practice. Neither the total ICARS score nor any of the other ICARS subscores was related to the extent of adaptation. No ICARS scores were correlated with the rate of adaptation.

**DISCUSSION**

In the first experiment, we showed that visuomotor adaptations using prisms can be highly general or highly specific, depending on the type of movement practiced. We report here a clear asymmetry between reaching and walking prism adaptations: the adaptation generalizes broadly from walking to reaching but not from reaching to walking. In the second experiment, we showed that cerebellar damage impairs the walking prism adaptation, resulting in reduced adaptation magnitudes, slowed adaptation rates, and reduced negative aftereffects and generalization. We also replicate the findings from experiment 1 in a larger sample of 12 different healthy control subjects. This is the first report of asymmetric generalization following prism walking adaptations and the first demonstration of cerebellar involvement in adapting the walking trajectory to prism glasses.

**Possible mechanisms for prism walking generalization**

Little evidence suggests that prism adaptations can generalize across very different movement types. Weak intermanual transfer of reaching adaptations can occur if the hand is viewed only at movement termination (Redding and Wallace 1988a);
transfer from arm to leg (Elliot and Roy 1981) or leg to arm (Elliot and Roy 1981; Mikaelian 1970) may also occur. These types of generalization are thought to be due to either visual recalibrations, such as altered perception of eye position and/or the perception of straight-ahead (Elliot and Roy 1981; Redding and Wallace 1988a), or proprioceptive recalibrations involving an alteration in the felt position of the head (Held and Freedman 1963; Mikaelian 1970). Rossetti et al. (1998) found that stroke patients with hemispatial neglect show some generalization from reaching to drawing tasks or to line bisection tests. In addition, some evidence indicates that both controls and patients with neglect can produce very slight shifts in their standing postural center of pressure after prism reaching adaptations (Michel et al. 2003; Tilikete et al. 2001). In most cases, the extent of generalization across movements was quite small compared with the strong generalization from walking to reaching that we observed here.

It seems logical and behaviorally advantageous for prism adaptation of a movement like walking to generalize to reaching given the linkage between the two actions: humans walk through space to bring themselves close enough to interact with (reach for) objects. Prism adaptation of reaching might not generalize to walking because there is no reverse dependence (i.e., humans walk to reach, but don’t reach to walk). Still, the precise neural mechanism(s) by which the generalization from walking to reaching occurs is not clear.

There were several differences between the walking and reaching adaptation paradigms that could explain our observation of asymmetric generalization. First, prism adaptation during walking could generalize to reaching if overt arm movements occurred during walking. This was not the case because our subjects held the arms crossed against the chest during the entire prism exposure period. There is also broad activation of trunk and limb muscles during walking. Thus it is possible that muscle activity in the arms (in the absence of movement) during prism exposure caused generalization to reaching. However, the EMG analysis (groups 1c and 2b) indicated that arm muscle activity during crossed-arm walking was rather minimal and did not show modulation according to the phases of the stride cycle. In addition, there was clear postural muscle activity in the legs during reaching, yet the reaching adaptation did not generalize to walking. In fact, leg muscle activity during reaching never exceeded arm muscle activity during walking. This would argue that nonspecific muscle activation of a body part during prism exposure is insufficient to induce generalization.

We addressed two other important distinctions between the walking and reaching adaptations. In the main analysis (groups 1 and 2), movement and adaptation durations varied between the two tasks. The length of time to perform each movement and the length of time required to complete the entire adaptation were always more for the walking task than the reaching task. Increased exposure to the prisms, either by lengthening each trial or by increasing the total amount of time wearing prisms, could potentially lead to better adaptation and perhaps better generalization. However, when we matched reaching to walking in terms of movement duration (compare groups 1b and 2b) and adaptation duration (compare groups 1c and 2b), we found no change in the generalization. Generalization from walking to reaching remained robust, while generalization from reaching to walking remained negligible. Therefore neither movement duration nor total adaptation duration appeared to contribute to the asymmetric generalization.

The type and length of visual feedback can also affect prism adaptation (Kitazawa et al. 1995) and generalization (Norris et al. 2001; Redding and Wallace 1988a). For instance, delayed visual feedback prolongs adaptation rates (Kitazawa et al. 1995). Terminal visual feedback (as opposed to continuous vision) during prism reaching adaptations increases the likelihood of weak intermanual transfer (Redding and Wallace 1988a). Similarly, viewing the hand itself (as opposed to watching a real-time computer representation of the hand) during prism reaching adaptations increases negative aftereffects when tested for when the hand is visible (Norris et al. 2001). Nevertheless, in our paradigm, subjects were always allowed full and continuous vision of their hand and arm (reaching) and legs and feet (walking) throughout the adaptation phase. Therefore we do not think differences in visual feedback could have produced the asymmetric generalization.

Walking with prisms could also recalibrate vestibular information, such that rightward head or trunk rotation (with prisms on) might become associated with forward linear acceleration of the body. Vestibular recalibration via galvanic stimulation has been shown to produce lateral deviations in walking trajectory (Bent et al. 2000; Fitzpatrick et al. 1999; Kennedy et al. 2003) and may even cause reaching endpoint errors (Bresciani et al. 2002). However, reaching deviations produced by galvanic stimulation are much smaller (<1° on average) than the strong generalization aftereffects we reported here. Thus it does not appear that a vestibular recalibration alone could explain our findings. An aftereffect in head or trunk rotation could also have altered reaching direction. However, the analysis of body segment rotations indicated no systematic trunk or head twisting aftereffects, so the generalization from walking to reaching was not a by-product of a postural change. Walking with prisms could also alter optic flow processing, but walking direction during prism exposure is believed to rely much more on perceived target direction than optic flow (Harris and Bonas 2002; Rushton et al. 1998).
Thus asymmetric generalization likely depends on other factors. We mentioned that there could be recalibration of visual perception, limb proprioception, and/or motor commands. Adaptations involving a visual shift are expected to generalize to other movements because a change in visual perception of straight ahead would affect all subsequent visually guided movements. Adaptations involving a limb proprioceptive shift are not expected to generalize because changes in the felt position of a limb would be specific to that limb (Baraduc and Wolpert 2002; Martin et al. 1996b). However, a proprioceptive shift occurring at a higher level, such as the head-on-trunk segment, would be expected to cause generalization, because a change of the felt position of the head on the body would alter all successive limb movements (just as would the eyes-in-head visual shift). Alternatively (or additionally), a more distributed proprioceptive shift might occur. A widespread shift in proprioception occurring at multiple segments including the eyes, head, trunk, and limbs could also produce generalization. In experiment 1, we used accepted methods (Redding and Wallace 1994, 1988a,b) to directly test for proprioceptive and visual perceptual shifts after reaching and walking prism adaptations. We found, in both cases, small and nonsignificant visual and proprioceptive perceptual shifts, although they were comparable in magnitude to those previously reported (Redding and Wallace 1994). After adapting to prisms during walking (group 2), four of the five subjects had appropriately compensatory (rightward) visual shifts (mean for 4 rightward shifters: $1.07 \pm 0.23^\circ$), indicating that the walking adaptation may have produced some shift in the perception of the direction of straight ahead. In contrast, none of the subjects who adapted to prisms during reaching (group 1) showed appropriate rightward visual shifts. Still, the extent of visual shift was too small, even in these four subjects, to entirely (or even mostly) explain the magnitude of generalization to reaching. Because the visual and proprioceptive perceptual shift tests showed no clear evidence for a recalibration of the felt positions of either the eyes-in-head, head-on-trunk, or trunk-on-limbs, our results (asymmetric generalization) cannot be explained solely on the basis of a visual and/or proprioceptive shift. Even a proprioceptive shift occurring in a more distributed manner should have been observable through one or both of the perceptual tests.

We therefore think that the recalibration affects some aspect of the motor command. Generation of the motor command can be broadly defined as the process of translating visual information to action (Baraduc and Wolpert 2002). Recalibration of commands in this processing stream could alter motor output without necessarily changing sensory perception. It is possible that some forms of visuomotor adaptation alter movement commands that are specific to an effector and therefore would not generalize to other movements. For example, an adaptation may alter activity of brain regions used to produce a certain arm reach movement. Alterations in the motor command could theoretically be more general if the adaptation affected motor representations that are not effector-specific. For example, an adaptation may alter brain regions that control higher-order motor parameters that can be applied to many different effectors and/or movement types. It may be that prism adaptation during walking, because it involves whole body movements through space and/or because it involves coordination across many muscles, recalibrates a more general, less effector-specific system of visuomotor representation. On the other hand, prism adaptation during isolated movements of the arms, which have more independence and range with respect to the torso, are probably calibrated by a more effector-specific mechanism (Baraduc and Wolpert 2002; Martin et al. 1996b). Brain structures that could be involved in this process are discussed below.

Cerebellar control of locomotor adaptations

Previous work has shown that the cerebellum is necessary for specific arm-related visuomotor prism adaptations (Baizer et al. 1999; Deuschl et al. 1996; Martin et al. 1996a,b; Weiner et al. 1983). In general, very few studies have examined cerebellar contributions to locomotor adaptations. In one case, cerebellar subjects were shown to adapt to predictable changes in treadmill speed, but with a strategy that was different from controls (Rand et al. 1998). In another study, it was shown that cerebellar subjects have impaired storage of the podokinetic locomotor adaptation produced by repeated walking in place on a rotating disc (Earhart et al. 2002).

This is the first demonstration of cerebellar involvement in a visuomotor walking adaptation. Here, we show that the cerebellum is required for a prism-induced adaptation of walking trajectory that is broadly generalizable across body parts. Subjects with cerebellar damage were slow to adapt their walking trajectory to prisms, did not adapt to the same extent as controls, and showed a diminished negative aftereffect, indicating reduced storage of the adaptation. They also showed poor generalization from walking to reaching. Nevertheless, most cerebellar subjects did show some improvement of their walking trajectories over the course of the adaptation phase, and we were able to fit exponential curves to each subject’s data, which provided us with predictions of the rate of adaptation and the absolute adaptation magnitude (Martin et al. 1996a). It is worth noting that our sample of cerebellar subjects was somewhat biased toward the more mildly affected. As evidence, average ICARS scores for the group were 25.2, indicating relatively mild ataxia. To participate, cerebellar subjects had to be capable of walking relatively long distances (~30–40 trials walking) without support, and sometimes with eyes closed (baseline and post-adaptation phases). Moderate to severely ataxic patients would undoubtedly be unable to complete the paradigm. Perhaps cerebellar subjects with more extensive damage would have performed even more poorly. It is also possible that the two younger subjects (8-yr-old CBL-10 and 16-yr-old CBL-11) in the cerebellar group differed from adults in terms of the manner and extent to which they recovered from cerebellar damage. However, the data show that neither of the subjects performed outside the range of our representative cerebellar sample and that the cerebellar group data would not have changed substantially had their data been removed.

Our sample of relatively mildly ataxic subjects provided us with an opportunity to test the effect of cerebellar damage on the generalization of prism adaptations. If none of the subjects was able to store the adaptation, we would be unable to make any conclusions regarding their ability to generalize (given that the adaptation must be stored for it to generalize to different contexts). However, since a few subjects were able to adapt, we were able to assess this issue preliminarily. We found that
the cerebellar subjects who appeared to adapt well often did not show appropriate generalization. The reduced generalization might indicate 1) a fundamental deficit in generalization (in addition to the fundamental deficit in adaptation) or 2) that, although the adaptation was obtained, it was acquired and/or stored through different mechanisms than those used by healthy individuals. Although very intriguing, our speculation must be taken with considerable caution, because no clear interpretation regarding generalization can be made from a sample of subjects who showed minimal adaptation in the first place.

In addition to the cerebellum, other brain regions have also been implicated in prism adaptation of arm movements. In a PET study, humans adapting to prisms during reaching showed selective activation of the posterior parietal cortex (PPC) contralateral to the reaching arm (Clower et al. 1996). The authors concluded that the cerebellum may be responsible for the specific error-correction component of prism adaptation, while the PPC encodes the remapping of proprioceptive and visual representations (Clower et al. 1996). This seems plausible since the comparable brain region in macaque is known to have cells with both somatosensory and visual receptive fields (Colby et al. 1993; Duhamel et al. 1998). Ventral premotor cortex (PMv) has also been implicated in prism adaptation: when the PMv is inactivated via muscimol injection, monkeys lose their ability to adapt to prisms during reaching (Kurata and Hoshi 1999).

The adaptive process likely depends on the integrity of all three of these brain regions: cerebellum, PPC, and PMv. The interconnectivity between the cerebellum and the PPC and PMv is well established. Large portions of the cerebellar output via dentate nucleus project to PPC (Clower et al. 2001; Dum and Strick 2003) and PMv (Dum and Strick 2003; Middleton and Strick 1997) via the thalamus. Similarly, efferents from both PPC (Brodal and Bjaalie 1997) and PMv (Glickstein et al. 1985) provide a substantial share of the afferent projections to the lateral cerebellar cortex via the corticopontocerebellar pathway. It is proposed that one function of the cerebellum is to alter motor output over time using error-feedback (Albus 1971; Gilbert and Thach 1977; Ito 1984; Kawato and Gomi 1992; Kitazawa et al. 1998). If so, then lesioning either PPC or PMv might cause similar impairments of motor adaptation as seen with cerebellar lesions. Damage to PPC or PMv might prevent appropriate error signals from reaching the cerebellum (i.e., disrupted connections from PPC/PMv to cerebellum). Alternatively, damage to either of these regions might prevent the correct, adapted movement (from cerebellum) from reaching downstream motor targets that more directly generate the movement (i.e., disrupted connections from PPC/PMv to primary motor cortex or spinal cord) (Kurata and Hoshi 1999).

Thus far, PPC and PMv have been shown to be involved in limb specific prism adaptations, but each might also be involved in the broader, generalizable adaptation to prisms during walking as well as its subsequent generalization. Regions of both PPC and PMv are active during certain types of movements regardless of whether they are made with the arm or leg, suggesting that they may encode higher-order movement parameters that are not effector-specific (Ehrsson et al. 2000; Rijnjts et al. 1999). These areas, with their direct cerebellar connections (Dum and Strick 2003), may help support cerebellar error-based recalibration of higher-order movement mechanisms (Greger et al. 2004) that generalize across body parts.

In summary, we report here a prism adaptation that generalizes broadly across movement types: prism adaptation of walking trajectory generalizes to reaching—a different behavior involving a different body part. We found that the generalization is asymmetric: adaptation of reaching does not generalize to walking. We propose that the walking adaptation involves a recalibration of motor representation(s) that are independent from the exposed effectors and control motor parameters that can be used for many different behaviors and body parts. We also show that this broadly generalizing walking adaptation is impaired in people with cerebellar damage. Thus, in addition to its role in limb-specific adaptations, the cerebellum is also integral for motor adaptations that invoke more general systems of visuomotor recalibration. We are currently designing studies to determine the specific mechanism(s) for the generalizability of prism walking adaptations. In addition, the exact role the cerebellum plays in the adaptive process, in conjunction with other brain regions such as PPC and PMv, must be further investigated.

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