

Cerebellar Subjects Show Impaired Adaptation of Anticipatory EMG During Catching

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Lang, Catherine E. and Amy J. Bastian. Cerebellar subjects show impaired adaptation of anticipatory EMG during catching. *J. Neurophysiol.* 82: 2108–2119, 1999. We evaluated the role of the cerebellum in adapting anticipatory muscle activity during a multijointed catching task. Individuals with and without cerebellar damage caught a series of balls of different weights dropped from above. In *Experiment 1* (light-heavy-light), each subject was required to catch light balls (*baseline phase*), heavy balls (*adaptation phase*), and then light balls again (*postadaptation phase*). Subjects were not told when the balls would be switched, and they were required to keep their hand within a vertical spatial “window” during the catch. During the series of trials, we measured three-dimensional (3-D) position and electromyogram (EMG) from the catching arm. We modeled the adaptation process using an exponential decay function; this model allowed us to dissociate adaptation from performance variability. Results from the position data show that cerebellar subjects did not adapt or adapted very slowly to the changed ball weight when compared with the control subjects. The cerebellar group required an average of 30.9 ± 8.7 trials (mean \pm SE) to progress approximately two-thirds of the way through the adaptation compared with 1.7 ± 0.2 trials for the control group. Only control subjects showed a negative aftereffect indicating storage of the adaptation. No difference in performance variability existed between the two groups. EMG data show that control subjects increased their anticipatory muscle activity in the flexor muscles of the arm to control the momentum of the ball at impact. Cerebellar subjects were unable to differentially increase the anticipatory muscle activity across three joints to perform the task successfully. In *Experiment 2* (heavy-light-heavy), we tested to see whether the rate of adaptation changed when adapting to a light ball versus a heavy ball. Subjects caught the heavy balls (*baseline phase*), the light balls (*adaptation phase*), and then heavy balls again (*postadaptation phase*). Comparison of rates of adaptation between *Experiment 1* and *Experiment 2* showed that the rate of adaptation was unchanged whether adapting to a light ball or a heavy ball. Given these findings, we conclude that the cerebellum is important in generating the appropriate anticipatory muscle activity across multiple muscles and modifying it in response to changing demands through trial-and-error practice.

INTRODUCTION

The cerebellum has long been implicated in the coordination of movement (Diener and Dichgans 1992; Holmes 1917; Thach et al. 1992). However, what aspects of movement are “coordinated” and how the cerebellum accomplishes this are still not understood. Recent evidence demonstrates that a potential mechanism for multijointed reaching ataxia in cerebellar

subjects is an inability to adjust for complex interaction torques generated between moving joints (Bastian et al. 1996; Topka et al. 1998). Cerebellar subjects also produce abnormal postural reactions to adjust for the mechanical effects of raising their arms (Diener et al. 1989). Based on these studies, it has been proposed that one role of the cerebellum may be to modulate muscle activity across multiple joints in anticipation of the mechanical demands of the movement and ultimately produce movement of the appropriate trajectory and magnitude (Bastian et al. 1996).

To adjust for the mechanical demands of movement, the nervous system must continually vary motor commands to account for the spectrum of mechanical contexts experienced in everyday movements (e.g., varied loads). Slow movements may rely on feedback-dependent processes to make these types of corrections, but fast movements require anticipatory mechanisms. To efficiently anticipate and adjust for changing mechanical demands, the nervous system must have a means of estimating the effect of loads on the motor apparatus. One possibility is that the nervous system makes explicit calculations to adjust a limb movement for complex interactions between joints and/or novel inertial loads. Another possibility is that these adjustments are learned through trial-and-error practice and stored. In support of the latter idea, normal subjects require trial-and-error practice to adapt multijoint limb movements to novel external forces (Gordon et al. 1993; Lackner and Dizio 1994; Shadmehr and Mussa-Ivaldi 1994), novel inertial loading (Sainberg et al. 1995; Sainberg and Ghez 1995), and visual environment shifting via prisms (Harris 1963; Held and Hein 1958; Thach et al. 1992). In all cases, removal of the novel perturbation following practice resulted in a negative aftereffect, such that the movement was deviated in a direction opposite the perturbation. This negative aftereffect appears to be the strongest evidence that the adaptation was stored in the central nervous system.

Based on the architecture of the cerebellar cortex, both Marr (1969) and Albus (1971) proposed that the cerebellum is well suited to learn patterns through trial-and-error practice. Gilbert and Thach (1977) support this idea by showing alterations in the relative activity of simple and complex spikes in cerebellar Purkinje cells related to learning force perturbations of different magnitudes (but see Ojakangas and Ebner 1992, 1994). Further experimental evidence suggests that humans and monkeys with cerebellar damage are impaired in their ability to adapt arm movements to a shifted visual environment (Baizer et al. 1999; Martin et al. 1996; Weiner et al. 1983) and to novel visual-motor gains (Deuschl et al. 1996). This impairment

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generally consisted of slowed adaptation and decreased or absent negative aftereffect, indicating that the adjustment was not stored.

The role of the cerebellum in adapting anticipatory mechanisms of motor control remains unclear. Prior work has shown that cerebellar subjects had difficulty modifying the magnitude of automatic postural responses to different anticipated displacements of a force platform (Horak and Diener 1994). In a subsequent study, the authors attribute this deficit to faulty response gain control, not to an inability to anticipate the magnitude of the perturbation (Timmann and Horak 1997). Other investigators have assessed whether cerebellar subjects could coordinate and scale pinch and lift forces when picking up different loads. They found that cerebellar subjects could adjust their pinch force levels to different loads but were less efficient in adapting peak pinch force rates based on experience (Müller and Dichgans 1994). This study did not distinguish whether the deficit was due to faulty adaptation or variable task performance. Highly variable task performance can give the appearance of an inability to adapt motor behavior. However, deficits in adaptation and performance can be dissociated by modeling the adaptation process (Deuschl et al. 1996; Keating 1994; Martin et al. 1996). We sought to more clearly distinguish the role of the cerebellum in anticipating mechanical demands by using a task that required anticipatory muscle activity prior to a perturbation and allowed a dissociation of task performance from adaptation.

Our experiments were designed to determine whether and how individuals with cerebellar damage adapt to different weights in a multijointed catching task. To make a successful catch, an individual relies primarily on anticipatory muscle activity timed to occur *before* the moment of impact between the hand and the ball and relies little on stretch reflex gain (Bennett et al. 1994). Normal subjects scale their anticipatory muscle activity to the expected ball momentum at impact (Lacquaniti and Maioli 1989). We hypothesized that the cerebellum “adapts” through trial-and-error to make the appropriate anticipatory adjustment to control changing forces. To test this, individuals with and without cerebellar damage were asked to catch balls of different weights. Our results show that cerebellar subjects were not able to adapt or adapted very slowly to the different weights as compared with the control subjects. This behavioral deficit was matched by a specific inability to adapt anticipatory muscle activity differentially across multiple joints.

METHODS

Subjects

Nine subjects with cerebellar lesions and nine control subjects participated in *Experiment 1* (Table 1). Five additional control subjects participated in *Experiment 2*. In the cerebellar group, three subjects had cerebellar hemorrhages, and six subjects were diagnosed with different forms of cerebellar cortical atrophy. Cerebellar lesions were confirmed by imaging [computerized tomography or magnetic resonance imaging (MRI)] and/or by genetic testing. All cerebellar subjects underwent a neurological examination to determine the severity of the movement deficit and to check for signs of involvement of other motor structures. The cerebellar subjects tested in this study presented with limb ataxia, consisting of irregular arm and leg movements (by finger-nose-finger and heel-knee-shin tests), hypermetria, and decreased ability to perform rapid alternating movements of the arm. None of the cerebellar subjects appeared to be hypotonic (e.g., no pendular reflexes, no excessive arm swing during walking). All of the cerebellar subjects walked with a wide-based gait and used an assistive device or were unable to walk on their own. As in other lesion studies, the cerebellar subjects had incomplete lesions, and thus presumably maintained some residual cerebellar function. None of the subjects had any sign of peripheral neuropathy. One cerebellar subject (*CBL-02*) showed hyperreflexia at the ankles. Another cerebellar subject (*CBL-05*) had a small lesion confined to the brachium pontis (middle cerebellar peduncle); MRI and clinical examination showed that this lesion did not extend into the pons. Last, one cerebellar subject (*CBL-09*) had an old ischemic infarct in the right occipital lobe (in addition to the acute hemorrhage in the right cerebellum). This did not impair his ability to see and track the ball during the catching task.

Control subjects were matched by age (60.7 ± 16.8 yr), gender, and hand preference to the cerebellar group. Informed consent was obtained from all subjects before testing.

Paradigm

Subjects were required to catch balls of different weight but identical appearance. Figure 1A illustrates the experimental setup. Each subject held one arm out in $\sim 30^\circ$ of shoulder flexion, 80° of elbow flexion, and neutral wrist position. A vertical pole was positioned next to the subject's outstretched hand, with markings indicating the top, bottom, and middle of a 10-cm “window.” Each subject was instructed to align his hand such that it was level with the middle of the window. We checked to ensure that the subject's hand was in the middle of the window before the start of each trial. When a tone sounded, a ball was dropped from 40 cm directly above the subject's hand. The 40-cm vertical drop height was chosen based on prior work that demonstrated that this drop height allowed sufficient time for anticipatory muscle activity to occur (Lacquaniti and Maioli 1989). Subjects were instructed to catch the ball while attempting to maintain hand position within the window

TABLE 1. Subject information

Subject	Age	Gender	Lesion	Length of Illness	Arm Ataxia	Arm Tremor	Stance/Gait Ataxia
<i>CBL-01</i>	62	M	Pancerebellar cortical atrophy	12 years	Moderate	Present	Moderate
<i>CBL-02</i>	43	F	Pancerebellar cortical atrophy	12 years	Moderate	Present	Moderate
<i>CBL-03</i>	72	M	Right hemispheric hemorrhage	3 weeks	Moderate	Absent	Severe
<i>CBL-04</i>	41	F	OPCA	10 years	Severe	Present	Severe
<i>CBL-05</i>	73	M	Left brachium pontis hemorrhage	1 month	Moderate	Absent	Moderate
<i>CBL-06</i>	74	M	SCA6	6 years	Severe	Present	Severe
<i>CBL-07</i>	67	M	OPCA	11 years	Moderate	Absent	Moderate
<i>CBL-08</i>	35	F	SCA7	5 years	Moderate	Absent	Moderate
<i>CBL-09</i>	70	M	Right hemispheric hemorrhage	3 months	Moderate	Absent	Severe

OPCA, olivopontocerebellar atrophy; SCA6, spinocerebellar atrophy type 6; SCA7, spinocerebellar atrophy type 7.

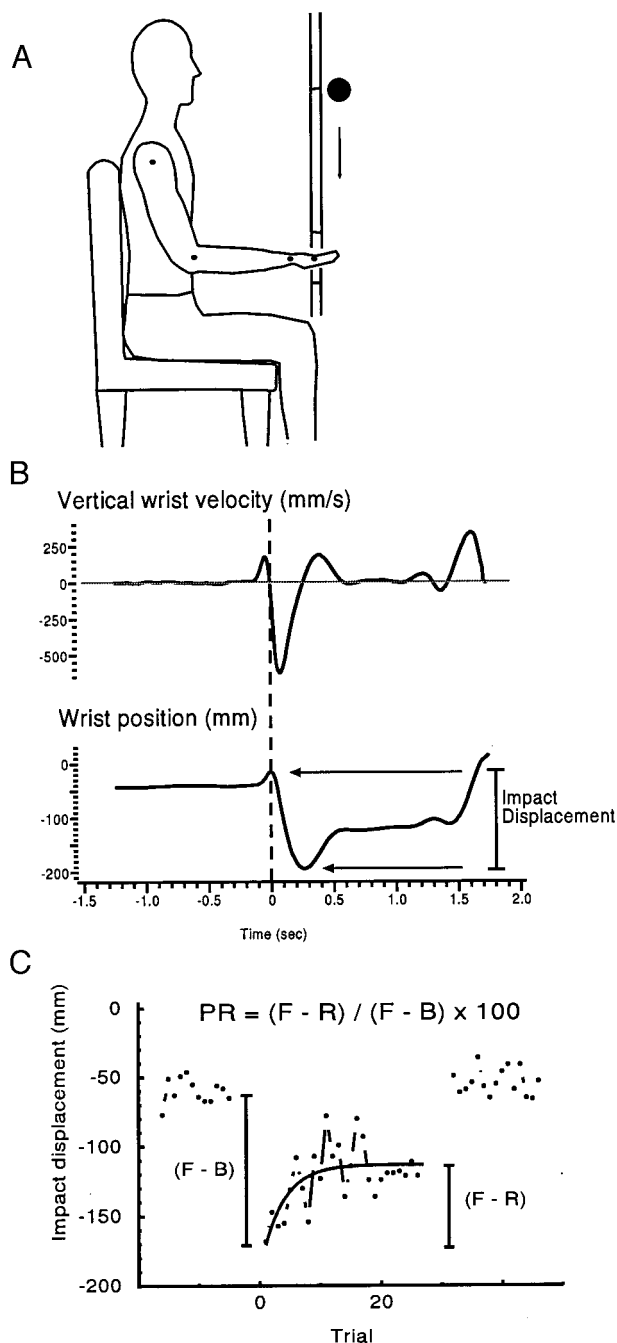


FIG. 1. A: schematic of the catching task. B: typical vertical wrist velocity and position traces demonstrating impact selection and impact displacement calculation. The trial is aligned on impact, marked by the dashed vertical line. (The changes in position and velocity that occur after 1.50 s are due to the subject handing the caught ball to the investigator.) C: calculation of percent return from impact displacement values. Impact displacement is plotted as a function of trial. Trials before 1 constitute the *baseline phase*, trials 1–25 constitute the *adaptation phase*, and trials after 25, the *postadaptation phase* for this individual (CBL-08). The small lines connecting the impact displacement values are provided to show sequential order of the trials. The large curvilinear line through the values in the adaptation phase represents the exponential curve fit. PR, percent return; F, impact displacement in the 1st trial of the adaptation phase; R, mean impact displacement of the last 10 trials in the adaptation phase; B, mean impact displacement of the baseline phase.

as they caught it. Each subject was given verbal feedback on every third trial (approximately) about whether they were successful at maintaining hand position within the window. Additionally, all

subjects reported that they were aware of trials in which their hand was out of the window during the catch.

The balls were ~12 cm diam and were easy to grasp. The balls were made of rubber latex outside and filled with various granular materials to produce the necessary weights. Each subject repeatedly caught two different balls, one “light” and one “heavy.” The weight of the light ball was either 320 or 545 g, depending on the subject’s size or body weight. The heavy ball was always 450 g heavier than the light ball. The duration of the ball drop was ~286 ms, and the momentums at impact were 896 g * m/s (320-g ball), 1,527 g * m/s (545-g ball), 2,157 g * m/s (770-g ball), and 2,787 g * m/s (995-g ball). Ten to 15 unrecorded practice trials with the light ball were given to familiarize the subject with the instructions and with catching in the window.

Recorded trials were blocked as follows. For *Experiment 1* (light-heavy-light), the *baseline phase* consisted of 10–20 trials with the light ball, the *adaptation phase* consisted of 25–35 trials with the heavy ball, and the *postadaptation phase* consisted of 15 additional trials with the light ball (to test for storage in the form of an aftereffect). All subjects participated in *Experiment 1*. For *Experiment 2* (heavy-light-heavy), the baseline phase consisted of 15–40 trials with the heavy ball, the adaptation phase consisted of 25–35 trials with the light ball, and the postadaptation phase consisted of 15 additional trials with the heavy ball. We had subjects complete more trials in the baseline phase in *Experiment 2* because the cerebellar subjects often had more difficulty catching the heavy ball within the window. Five of the nine cerebellar subjects and five additional control subjects participated in *Experiment 2* after completing *Experiment 1*. For both experiments, subjects were informed that they would be catching balls of various weights but were not told when the balls would be switched. Subjects were required to rest their arm after every seven trials to avoid fatigue.

After completing all of the catching trials, subjects were tested to determine whether they could perceive the weight difference between the two balls. They were serially given each ball to hold in the catching hand and then asked to judge which ball was heavier. Additionally, upper extremity strength was measured in all subjects using a MicroFET2 hand-held dynamometer (Hogan Health Industries, Draper, UT). Maximum isometric force of the flexors and extensors of the shoulder, elbow, and wrist joints was determined by stereotypic placement of the dynamometer. Three measurements of each muscle group were taken in the sitting position, and the average of the three measurements was used as the maximum isometric force.

Data collection

Arm movement was recorded in three dimensions (3-D) using infrared emitting markers placed on the subject’s arm (OPTOTRAK System, Northern Digital, Waterloo, ON). Markers were placed on each subject’s 1) hand (lateral surface of the 1st metacarpophalangeal joint), 2) wrist (radial surface), 3) elbow (lateral epicondyle), and 4) shoulder (lateral surface of the head of the humerus). Three additional markers defined the window and the start position of the balls. Silver-silver chloride bipolar surface electrodes with on-site preamplification were used to collect simultaneous EMG from six muscles: 1) anterior deltoid, 2) posterior deltoid, 3) biceps, 4) triceps, 5) wrist flexors, and 6) wrist extensors. Marker position data and EMG data were time locked and collected simultaneously at 100 and 1,000 Hz, respectively.

Data analysis

KINEMATIC ANALYSIS. Position data were low-pass filtered at 10 Hz. Marker positions, velocities, and joint angles were calculated using OPTOTRAK software. Custom software was used for the following analyses. First, we determined impact (time of ball contact with the hand) using the vertical velocity of the wrist. Often the

subjects made a very small upward movement of the arm just before catching the ball. Impact was marked as the time when the vertical wrist velocity crossed the zero line. Figure 1B illustrates selection of impact on a typical vertical wrist velocity trace and the corresponding wrist position trace from a cerebellar subject. Impact times were confirmed by visual inspection of a stick figure animation of the arm catching the ball. All trials were aligned on impact for subsequent analysis. We then calculated, for each trial, a value of impact displacement. Impact displacement was defined as the vertical distance traveled by the wrist from impact to the first reversal in direction. Figure 1B illustrates impact displacement for a typical trial.

Three behavioral indices were derived from the impact displacement values: the adaptation coefficient, the performance coefficient, and the percent return. The adaptation coefficient was used as the measure of the rate of adaptation, or how quickly a subject's impact displacement values changed after repeated exposures to the new ball during the adaptation phase. The performance coefficient was used as the measure of performance variability, or how variable impact displacement values were from trial to trial within the adaptation phase. The percent return was used as the measure of the extent of the adaptation, or the magnitude of change in impact displacement values by the end of the adaptation phase.

To obtain the adaptation and performance coefficients, impact displacement was plotted as a function of trial for each subject individually. Then, the adaptation process for each individual was mathematically modeled by an exponential decay function (Deuschl et al. 1996; Keating 1994; Martin et al. 1996). Impact displacement values from the trials in the baseline phase were used as the baseline for this model. The rate of change in the impact displacement during the adaptation phase was modeled by fitting the data to an exponential decay function of the form

$$y = a - b * e^{-tc} \quad (1)$$

where a is the final value that the exponential decay function approaches, b is the magnitude of the adaptation required from the first adaptation trial to the value a , t is the trial number, and c (the decay constant) is the rate at which the adaptation takes place. In this model, the exponential decay constant, c , is the number of catches that it would take to obtain $(1 - e^{-1})$ or $\sim 63.2\%$ of the final adaptation. This value, c , was taken as the adaptation coefficient or measure of the rate of adaptation (Deuschl et al. 1996; Keating 1994; Martin et al. 1996). Thus our measure of the rate of adaptation is an estimation of the number of trials for a subject to proceed approximately two-third of the way through the adaptation process. All curve fits were generated using CoStat software (CoHort Software, Berkeley, CA).

We used trial-to-trial variability of the catch as a measure of performance variability. After the impact displacement values were fitted to the exponential curve, the residuals of the curve fit showed how variable a subject's performance was from one trial to the next in the adaptation phase. In statistical terms, the residuals of the curve fit are the differences between the predicted values (from the curve fit) and the actual values. Highly variable performance would result in residuals that were widely scattered around the curve; consistent performance would result in residuals that were narrowly distributed around the curve. The standard deviation of the residuals of the curve fit was taken as performance coefficient or the measure of performance variability (Keating 1994). It is important to note that the performance coefficient measures the trial-to-trial variability of the catching but does not measure the success (hand in the window) or failure (hand out of the window) of each trial.

To obtain the percent return, the extent of the adaptation by the end of the adaptation phase was measured. The percent return was defined as the relative return of impact displacement during the adaptation phase compared with the impact displacement in the baseline phase. Specifically, percent return was calculated using the following equation

$$\text{percent return} = (F - R)/(F - B) \times 100 \quad (2)$$

where F is the impact displacement in the first trial of the adaptation phase, R is the mean impact displacement of the last 10 trials in the adaptation phase, and B is the mean impact displacement of the baseline phase. Figure 1C illustrates how the percent return was calculated.

We were able to fit exponential curves to the behavior of all but two cerebellar subjects (*CBL-04* and *CBL-09*). We could not fit their behavior to the exponential function because they did not change their behavior at all during the adaptation phase, although they were aware of making errors. They were subsequently excluded from the statistical analysis of adaptation coefficients because no measure of rate of learning could be calculated and their performance coefficient was calculated as the standard deviation of the impact displacement values in the adaptation phase.

We confirmed the results of the exponential decay function using two simple analyses on data from *Experiment 1*. First, we calculated the number of trials in the adaptation phase that it took each subject to return consistently (3 trials in a row) to the window. Seven of nine cerebellar subjects were unable to return to keeping their hand in the window consistently or at all. For these subjects, the number of trials was considered the total number of trials in the adaptation phase. We performed a linear regression of adaptation coefficients on number of trials. The seven cerebellar subjects who never returned to the window were not included in this first analysis because the number of trials was limited by the number of trials tested. We found that the rate of adaptation was predictive of the number of trials to return to the window ($R^2 = 0.941$, $P < 0.0001$). Second, we performed a one-way ANOVA to see whether the same statistical conclusions would be obtained from testing number of trials between groups as from testing adaptation coefficients between groups. Results of the ANOVA on number of trials yielded the same statistical conclusions as the results of the ANOVA on the adaptation coefficients.

Statistica software (StatSoft, Tulsa, OK) was used for all statistical analyses, and the criterion level for statistical significance was set at $P < 0.05$. One-way ANOVA tests were used to assess differences between the control and cerebellar groups for adaptation coefficients, performance coefficients, and percent returns. As we were concerned about making a distinction between adaptation and performance variability, we calculated a linear regression of the adaptation coefficients on the performance coefficients to determine whether performance variability could predict rate of adaptation.

Additionally, a within-subjects repeated measures ANOVA was used to test for differences between groups for the magnitude of impact displacement data on selected trials. We compared impact displacement values for the last baseline trial, the first adaptation trial, the last adaptation trial, the first postadaptation trial and the last postadaptation trial. We used this analysis to test for storage of the adaptation (a negative aftereffect). Specifically, we tested to see whether the impact displacement of the first postadaptation trial was a mirror image (equal and opposite) of the impact displacement in the first adaptation trial.

MUSCLE ANALYSIS. Electromyographic (EMG) analyses were performed on data from *Experiment 1* (light-heavy-light) only. EMG data were amplified, full-wave rectified, and low-pass filtered at 70 Hz. EMG analysis includes data from eight cerebellar subjects because we were unable to obtain EMG recording on *CBL-09*. Trials were aligned on time of impact and ensemble averaged in two groups: 1) the last 10 baseline trials and 2) the last 10 adaptation trials. For both groups of trials, the averaged EMG was integrated (IEMG) over two periods: 1) anticipatory IEMG (the 100 ms before impact) and 2) postimpact IEMG (the 100 ms after impact). To compare IEMG values between subjects and groups, both anticipatory and postimpact IEMG were normalized. Normalization was done by dividing the IEMG values by the IEMG values of the first 500 ms of the grouped trials. This was the time period before the tone sounded and before the ball dropped. The

muscle activity during this period was only that necessary to hold the arm in position against gravity; this activity level did not change in either group from baseline to adaptation trials, as tested using a repeated-measures ANOVA. Thus the normalized anticipatory and postimpact IEMG values express the amount of muscle activity during these time periods as a function of the amount of muscle activity required to hold the arm in the start position.

Comparisons between control and cerebellar groups were done using a within-subjects repeated measures ANOVA for both the anticipatory IEMG and the postimpact IEMG. The repeated measures were baseline phase and late adaptation phase. We were specifically interested in interactions between group and phase. We performed this analysis on all six muscles recorded (3 flexors and 3 extensors). Anticipatory and postimpact IEMG from the extensor muscles (posterior deltoid, triceps, and wrist extensors) were usually present, but inconsistently modulated in both groups between the baseline and late adaptation phases. We could not detect differences in antagonist modulation between the light and heavy balls, nor between the control and cerebellar groups.

We have focused our analysis on the flexor muscles (anterior deltoid, biceps, and wrist flexors) because they were consistently modulated in the control group and because it is the combined action of the flexors that works directly to oppose the momentum of the ball. Additionally, the combined action of the flexors implies that either a change in the activity of a single flexor muscle, two flexor muscles, or all three could control the momentum of the ball. Therefore we calculated the sum of the normalized IEMG of the three flexor muscles for both the anticipatory and postimpact time periods in each individual. We used these sums as unitary measures of the ability to coordinate muscle activity across all three joints (shoulder, elbow, and wrist).

Last, maximal isometric force measurements of the tested muscles were compared between the groups using a multiple ANOVA. This was done to determine whether weakness in particular muscles could account for any deficits in catching.

RESULTS

Kinematic data

All subjects were able to catch the ball at least 90% of the time throughout the entire study. Most control subjects never dropped the ball. Three of nine cerebellar subjects dropped the ball four times or less, and only one cerebellar subject (*CBL-07*) dropped the ball eight times. Occurrence of ball drops was not specific to whether subjects were catching a light or heavy ball. Trials in which the ball was dropped were excluded from further analyses.

Figure 2 shows, for selected trials, plots of wrist position versus time for a control subject and two cerebellar subjects in *Experiment 1* (light-heavy-light). All subjects were able to catch the ball within the window for the baseline phase and were displaced out the bottom of the window on the first adaptation trial. By the last adaptation trial, control subjects were no longer displaced out of the bottom of the window with the heavy ball. This was not true for most of the cerebellar subjects. Note that one cerebellar subject (*CBL-03*) had returned to the edge of the window by the last adaptation trial, while the other cerebellar subject (*CBL-08*) had not. The control subject (Fig. 2A) shows a negative aftereffect for the first postadaptation trial. The first postadaptation trial (light ball, large dashed line) is the mirror opposite of the first adaptation trial (heavy ball, dotted line). The cerebellar subjects do not show a negative aftereffect. In Fig. 2, B and C, the first postadaptation trial is not the mirror opposite of the first

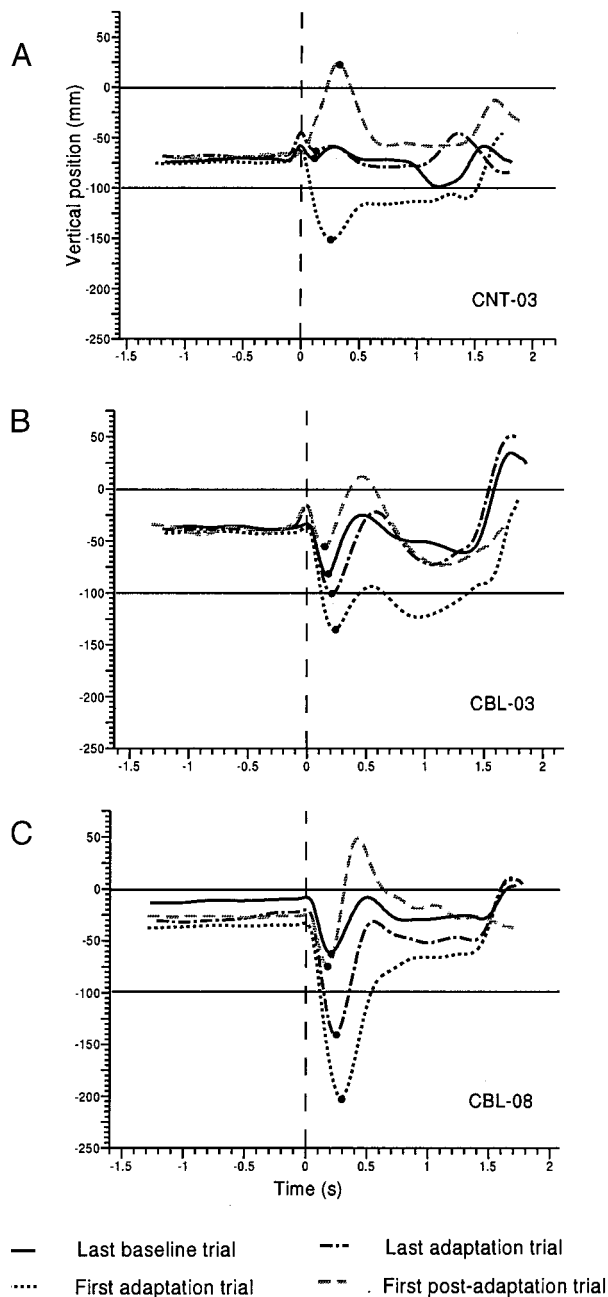


FIG. 2. Wrist displacement over time for selected trials in *Experiment 1* (light-heavy-light). Trials are aligned on impact, marked by the dashed vertical lines. Horizontal solid lines represent the 10-cm window. Shaded circles represent the 1st reversal after the catch. Examples from a control subject (A) and 2 cerebellar subjects (B and C).

adaptation trial. The cerebellar traces for the first postadaptation trials go out the top of the window, but this occurs after they have already been displaced downward.

Cerebellar subjects either did not adapt or adapted more slowly than control subjects, even though their performance variability was similar. Figure 3 shows examples of impact displacement plotted as a function of trial and the corresponding adaptation and performance coefficients (see METHODS) from *Experiment 1* (light-heavy-light). Data in Fig. 3, A and B, show the same subjects as Fig. 2, A and B. Figure 3A is an example of a typical control subject. Figure 3B is an example

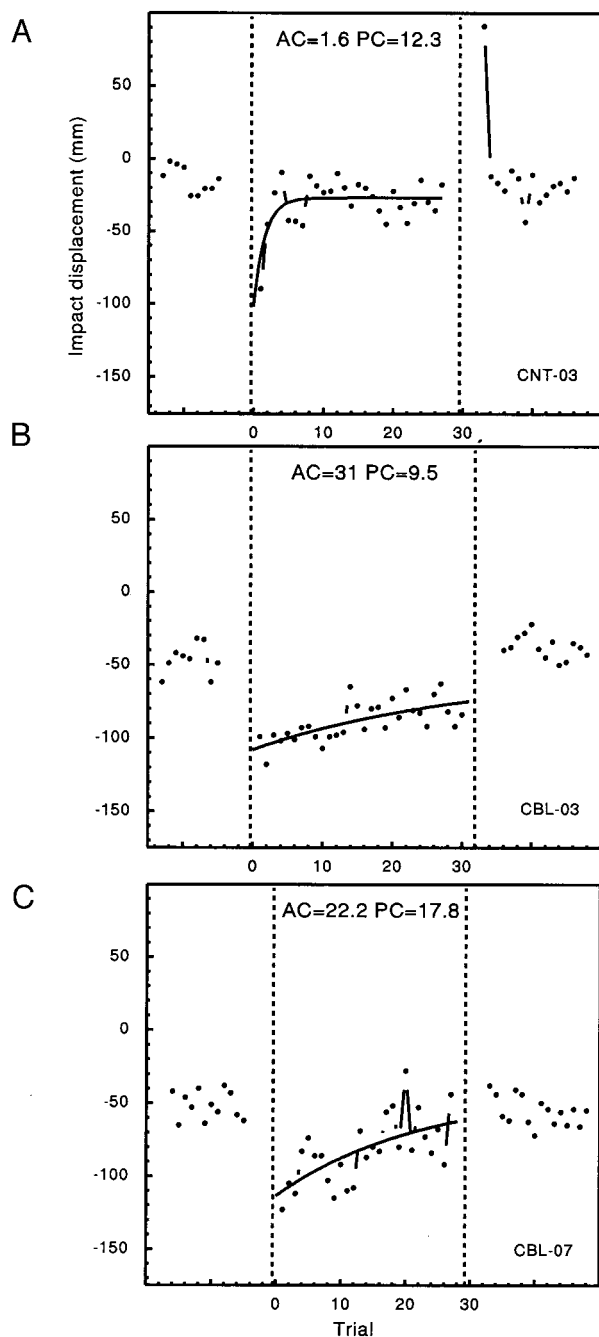


FIG. 3. Impact displacements plotted as a function of trial for *Experiment 1* (light-heavy-light). Negative impact displacements indicate displacement in the downward direction, whereas positive values indicate displacement upward. Vertical dashed lines separate baseline, adaptation, and postadaptation phases. Small lines connecting the impact displacement values are provided to show sequential order of the trials. Large solid line through the data points in the adaptation phase represents the exponential curve fit from which the adaptation coefficient (AC) and performance coefficient (PC) were calculated. See *Data analysis* for explanation of curve fitting. Examples from a control subject (A) and 2 cerebellar subjects (B and C) with their respective coefficients.

of a cerebellar subject with poor (slow) adaptation but with good performance variability (small scatter of trials around the curve fit). Figure 3C is an example of a cerebellar subject with poor (slow) adaptation and poor performance variability (greater scatter of trials around the curve fit). For *Experiment 1*,

TABLE 2. Measures of adaptation and performance for all subjects in *Experiment 1*

Subject	AC	PC	PR	Subject	AC	PC	PR
CBL-01	66.2	9.7	50	CNT-01	2.9	15.3	92
CBL-02	35.5	23.2	99	CNT-02	1.7	15.0	67
CBL-03	31.1	17.5	52	CNT-03	1.6	12.3	83
CBL-04*		13.0	18	CNT-04	1.6	14.4	71
CBL-05	7.0	21.5	88	CNT-05	1.4	12.7	74
CBL-06	51.6	12.1	44	CNT-06	0.9	13.4	83
CBL-07	22.2	17.8	80	CNT-07	1.4	16.7	75
CBL-08	2.5	17.2	51	CNT-08	2.0	16.1	74
CBL-09*		12.5	35	CNT-09	1.9	15.1	89

AC, adaptation coefficient; PC, performance coefficient; PR, percent return. * Subjects did not adapt; thus we were unable to fit a curve to these subjects.

adaptation coefficients (number of trials required to complete 62.3% of the adaptation) were an order of magnitude higher for the cerebellar group (30.9 ± 8.7 , mean \pm SE) than for the control group (1.7 ± 0.2 , $P < 0.002$). Adaptation coefficients, performance coefficients, and percent returns for all subjects are given in Table 2. ANOVA showed that there was no difference in performance coefficients between the control (14.6 ± 0.5) and cerebellar groups (15.2 ± 1.7 , $P = 0.729$). In addition, performance coefficients were *not* predictive of the rate of adaptation ($R^2 = 0.067$, $P = 0.334$). Finally, the percent return (see *METHODS*) was greater for the control group ($79 \pm 3\%$) than the cerebellar group ($57 \pm 8.8\%$, $P < 0.035$).

Figure 4 shows group summary data of impact displacement values for *Experiment 1*. Data points indicate the mean impact displacement values for the last baseline, first adaptation, last adaptation, first postadaptation, and last postadaptation trials. This figure illustrates two main points. First, the control group showed less displacement than the cerebellar group for the last adaptation trial ($P < 0.004$), signifying that the cerebellar group did not adapt to the same extent. This agrees with the above finding that the percent return in cerebellar subjects was

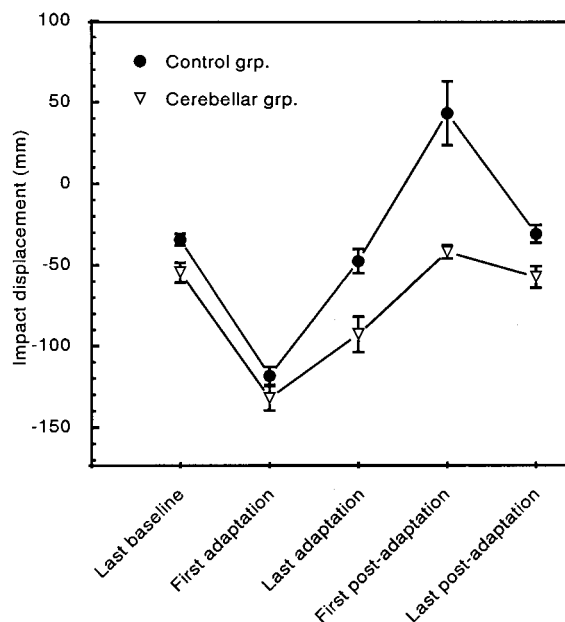


FIG. 4. Group averages (means \pm SE) of impact displacement data for selected trials in *Experiment 1* (light-heavy-light). ●, control group; ▽, cerebellar group.

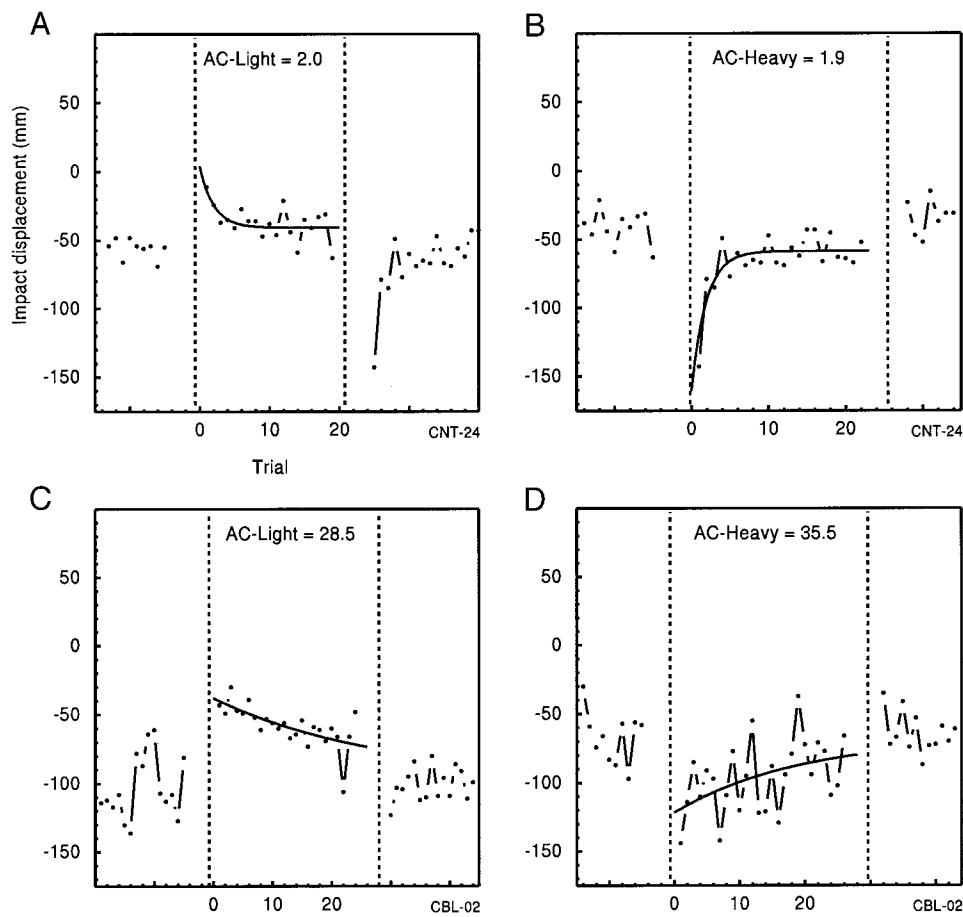


FIG. 5. Impact displacements plotted as a function of trial for *Experiment 2* (heavy-light-heavy) vs. *Experiment 1* (light-heavy-light). See legend of Fig. 3 for explanation. Examples from a control subject adapting to a light ball (*Experiment 2*; A) and heavy ball (*Experiment 1*; B) and a cerebellar subject adapting to a light ball (*Experiment 2*; C) and heavy ball (*Experiment 1*; D) with their respective adaptation coefficients.

less than the percent return in controls. Second, the control group was different from the cerebellar group for the first postadaptation trial ($P < 0.001$), signifying that only the control group had large negative aftereffects (approximately equal and opposite to the original displacement in the adaptation phase).

Adaptation to light versus heavy weights

Five individuals in the cerebellar group and five additional control subjects also participated in *Experiment 2* (heavy-light-heavy; see METHODS). In this experiment, we were interested in whether subjects would have similar rates of adaptation with the light ball as they had with the heavy ball. All of the control subjects were able to catch the heavy ball (baseline phase) within the window with practice. We allowed extra trials with the heavy balls in the baseline phase for the cerebellar subjects in an attempt to make their behavior as consistent as possible; two cerebellar subjects (*CBL-02* and *CBL-05*) could catch the heavy ball in the window and three could not. Despite failing to catch the heavy ball in the window, we could still measure the rate of adaptation to the light ball using impact displacement values. Figure 5 shows examples of impact displacement plotted as a function of trial and the corresponding adaptation coefficients for a control and cerebellar subject adapting to the light and the heavy ball. Data in Fig. 5, A and B, are from a control subject; her adaptation coefficient for the light ball (*Experiment 2*: heavy-light-heavy) was 2.0 trials, and her adaptation coefficient for the heavy ball (*Experiment 1*: light-

heavy-light) was 1.9 trials. Data from Fig. 5, C and D, are from a cerebellar subject; her adaptation coefficient for the light ball was 28.5 trials and her adaptation coefficient for the heavy ball was 35.5 trials. It should be noted that this cerebellar subject (*CBL-02*) was able to catch the heavy ball within the window in the baseline and postadaptation phases.

The adaptation coefficients for the control subjects for *Experiment 2* (heavy-light-heavy) were within one trial of their adaptation coefficients for *Experiment 1* (light-heavy-light). The adaptation coefficients for the cerebellar subjects for the two experiments were more disparate but were of the same order of magnitude. One cerebellar subject that did not adapt to the heavy ball (*CBL-04*) also did not adapt to the light ball. Table 3 gives the adaptation coefficients for the cerebellar and control subjects who participated in *Experiment 2* (heavy-light-heavy) and *Experiment 1* (light-heavy-light) as well as group means and standard errors. Statistically, the adaptation coefficients for *Experiment 2* were not different from those in *Experiment 1* (repeated measures ANOVA: main effect of heavy-light-heavy vs. light-heavy-light experiments $P = 0.364$; interaction effect of group vs. experiment $P = 0.385$).

Muscle data

Maximal isometric strength values for six muscles were compared between the control and cerebellar groups. No differences existed between the two groups for any of the muscles tested ($P = 0.759$). Subjects in both groups were able to

TABLE 3. Comparison of adaptation to the light ball versus adaptation to the heavy ball

Subject	AC, Light	AC, Heavy
CBL-01	20.6	66.2
CBL-02	28.5	35.5
CBL-03	44.1	31.1
CBL-04*		
CBL-05	5.0	7.0
Mean \pm SE	24.6 \pm 8.1	35.0 \pm 12.2
CNT-20	2.0	1.4
CNT-21	1.7	2.1
CNT-22	1.3	1.8
CNT-23	1.1	2.1
CNT-24	2.0	1.9
Mean \pm SE	1.6 \pm 0.2	1.9 \pm 0.1

AC, Light; adaptation coefficient when subject adapted to the light ball (*Experiment 2*); AC, Heavy, adaptation coefficient when subject adapted to the heavy ball (*Experiment 1*). * Subject did not adapt to either the heavy ball or the light ball; thus we were unable to fit a curve to this subject. This subject was omitted from the statistical analysis between experiments.

perceive the difference in weight between the balls used in their series of catching trials.

EMG analysis was performed on data from *Experiment 1* (light-heavy-light) only. Averaged EMG traces in Fig. 6 for a control and two cerebellar subjects illustrate that cerebellar subjects had difficulty adapting their anticipatory muscle activity to changed weights. Figure 6A shows a control subject who increased her anticipatory EMG (time -100 to 0 ms) primarily in the anterior deltoid and the wrist flexors from the baseline phase to the late adaptation phase. Control subjects demonstrated considerable variability in regard to which muscles had increased anticipatory EMG, although increases were most commonly seen in two or three of the flexor muscles. Figure 6B shows a cerebellar subject who did not demonstrate any increases in her anticipatory EMG when catching the heavy ball. Some cerebellar subjects could increase anticipatory activity in one muscle; this is shown in Fig. 6C. This cerebellar subject increased his anterior deltoid activity but not his biceps or wrist flexor activity.

As a group, cerebellar subjects were unable to increase their anticipatory muscle activity differentially across three joints to successfully keep their hand within the window. Figure 7 shows group averages for normalized anticipatory IEMG for baseline and late adaptation phases. A value of one (for normalized anticipatory IEMG) means that the muscle activity for the period 100 ms before impact was of the same magnitude as the muscle activity required to hold the arm in the start position. Cerebellar subjects were unable to increase anticipatory activity of all muscles to the same extent as controls. When looking at each muscle separately, the anterior deltoid ($P = 0.048$) and the wrist flexors ($P = 0.033$) showed significant interactions between group (control vs. cerebellar) and phase (baseline vs. late adaptation). The interaction for the biceps was not quite significant at the $P < 0.05$ level ($P = 0.052$). When we compared the linear sum of these three muscles, a significant interaction existed ($P = 0.011$) between group (control vs. cerebellar) and phase (baseline vs. late adaptation). These results demonstrate that, although cerebellar subjects were sometimes able to increase their anticipatory EMG in one

muscle, they were unable to increase their anticipatory EMG across multiple muscles.

The averaged EMG traces in Fig. 6 illustrate that cerebellar subjects were able to increase their postimpact EMG from the baseline to the late adaptation phase. The control subject and the cerebellar subjects shown in Fig. 6 all increased their postimpact EMG (time 0 , 100 ms) from the baseline to the late adaptation phase. Figure 8 shows group averages for normalized postimpact IEMG data. The control group had large increases in postimpact IEMG from baseline to late adaptation, whereas the cerebellar group had only moderate increases. The increases in postimpact IEMG from baseline to late adaptation were smaller in the cerebellar group compared with the control group, although the differences (interactions between group and phase) were not quite statistically significant (anterior deltoid, $P = 0.076$; biceps, $P = 0.095$; wrist flexors, $P = 0.084$). The change in the linear sum of the three flexor muscles was significantly different ($P = 0.039$) between groups.

Prior knowledge and visual cues

For seven control subjects, we varied the paradigm to provide prior knowledge about the switch in weight. In this variation, subjects were allowed to hold and feel the light and heavy balls before the catching trials. The subjects were told explicitly when the switch to a heavier or lighter ball was about to occur. Data from these variations were analyzed by calculating a value of impact displacement for each trial, fitting these values to an exponential curve and calculating adaptation and performance coefficients (see METHODS, *Data analysis*). Even with prior knowledge of the heavier ball weight and the time of the switch, impact displacement values for the first adaptation trial (107 ± 12 mm downward) more than doubled with respect to the magnitude of the baseline impact displacement values (41 ± 4 mm downward). After a few trials, the impact displacement values in the adaptation phase returned to values similar to those in the baseline phase. The rate of adaptation and the performance variability of these subjects was not different ($P = 0.184$, $P = 0.189$, respectively) than those of the control subjects in *Experiment 1* (without prior knowledge; control values given in Table 2). Means \pm SE of the adaptation and the performance coefficients are given in Table 4.

Additionally, we were interested in how vision might affect this task. Six control subjects performed the experiment twice, once with eyes open and once with eyes closed (using only the tone that sounded at the drop). Absence of vision did not change the general rate of adaptation but did cause a slight detriment in performance variability. Means \pm SE for the visual conditions are also given in Table 4. The results from these alternative conditions imply that this catching task requires trial-and-error learning but not necessarily visual cues.

DISCUSSION

Catching a ball requires anticipatory muscle activity that is scaled to the expected momentum of the ball (Lacquaniti and Maioli 1989). We found that control subjects could easily catch balls of different weight on the first trial, but

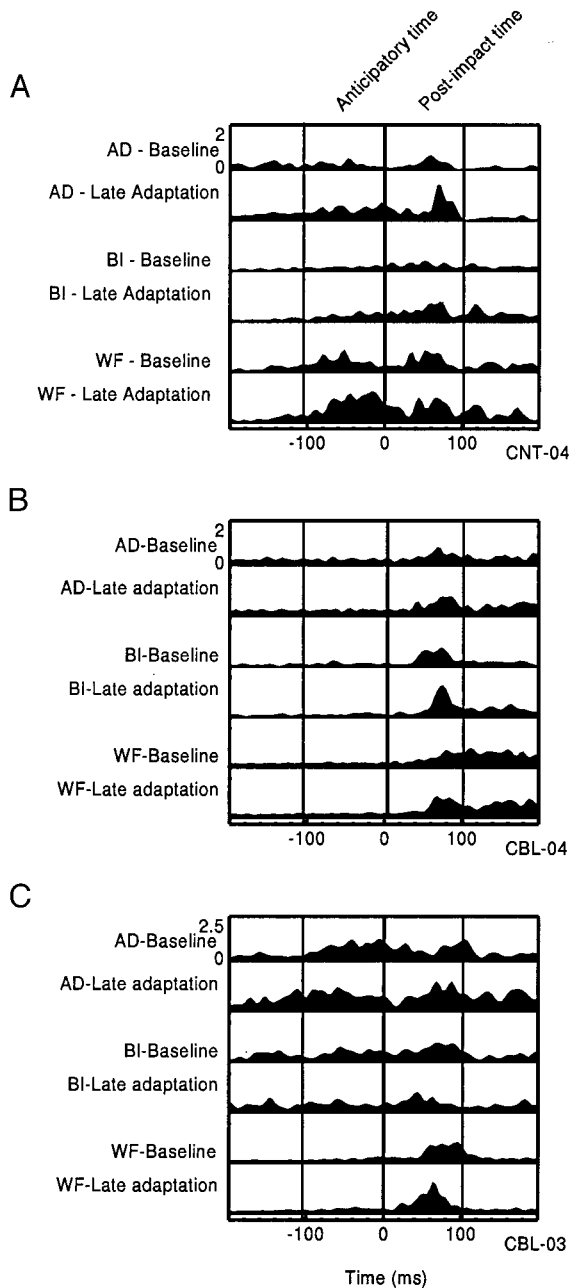


FIG. 6. Averaged electromyogram (EMG) for 3 muscle groups during the baseline phase and the late adaptation phase from *Experiment 1*. Each panel is arranged with the anterior deltoid on the *top*, the biceps in the *middle*, and the wrist flexors on the *bottom*. AD, anterior deltoid; BI, biceps; WF, wrist flexors. EMG data are aligned on impact (*time 0*). Anticipatory integrated EMG (IEMG) time period is -100 to 0 ms, and postimpact IEMG time period is 0 to 100 ms. Examples are shown from a control subject (A) and 2 cerebellar subjects (B and C).

could not maintain their hand in the spatial window until after they had performed the first few trials. The trial dependence of this adjustment did not change when we explicitly told subjects that we were switching the weights. A rather interesting finding was that control subjects could not correctly catch the ball in the window on the first trial even after they had held both weights in their hand before beginning the catching paradigm. This finding may be explained by the fact that anticipatory muscle activity is scaled to the

expected momentum of the ball, which depends on the drop height as well as the ball weight.

Cerebellar subjects were unable to adapt their catch at all or adapted it more slowly when compared with controls. This was demonstrated by increased adaptation rates and by lack of negative aftereffects. The deficit in adaptation could not be explained by poor performance variability. However, our measurement of performance variability was not a measure of success (able to catch in the window) or failure (unable to catch

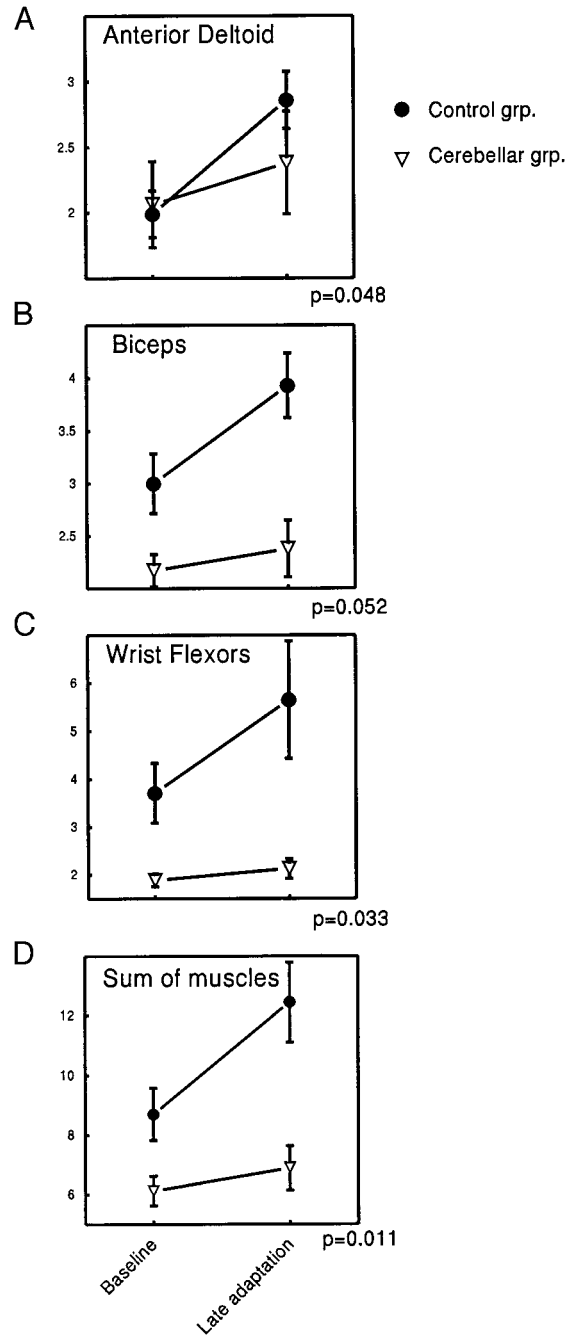


FIG. 7. Group averages (means \pm SE) of normalized anticipatory IEMG for the baseline and late adaptation phases from *Experiment 1*. ●, control group; ▽, cerebellar group. Three muscles: anterior deltoid (A), biceps (B), wrist flexors (C), and their linear sum (D). *P* values given are for the interaction between phase (baseline vs. late adaptation) and group (control vs. cerebellar).

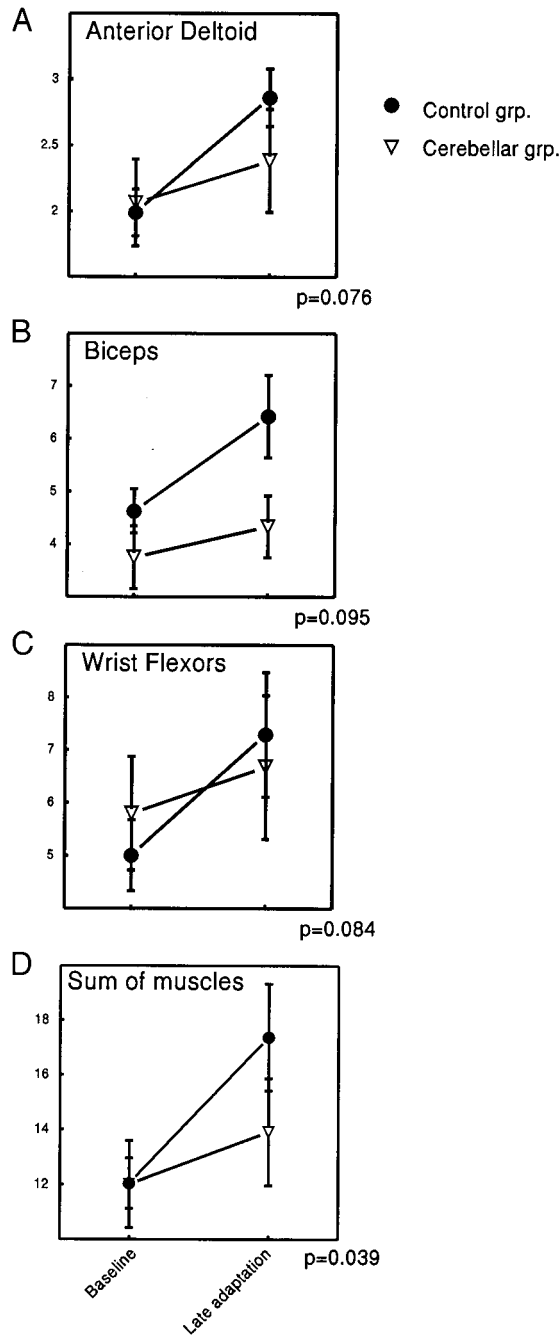


FIG. 8. Group averages (means \pm SE) of normalized postimpact IEMG data for the baseline and late adaptation phases from *Experiment 1*. ●, control group; ▽, cerebellar group. Three muscles: anterior deltoid (A), biceps (B), wrist flexors (C), and their linear sum (D). *P* values given are for the interaction between phase (baseline vs. late adaptation) and group (control vs. cerebellar).

in the window). Cerebellar subjects were not successful at keeping their hand within the window when catching the heavy balls as shown by the greater number of trials it took to return to the window (see METHODS) in *Experiment 1*. Additionally, we noted that the cerebellar group tended to make more extreme and variable corrective movements following the impact displacement. The variability of the postimpact corrective movements were partially due to tremor in some subjects (Table 1), as evidenced by frequency components in the 3- to 5-Hz range.

Contributions of anticipatory versus reflex muscle activity

The primary reason for the slowed adaptation in the cerebellar group was their inability to adjust anticipatory muscle activity. To make a successful catch (minimizing displacement), control subjects rely primarily on the anticipatory muscle activity timed to occur before ball impact (Bennett et al. 1994). Bennett and colleagues found that elbow deflections countered only by reflex muscle activity were 300% larger than elbow deflections countered by anticipatory and reflex muscle activity. The controls in our study used a wide variety of anticipatory muscle patterns to control the displacement of the arm during catching, most often increasing anticipatory activity in two or three muscles. Some cerebellar subjects could increase anticipatory activity in an individual muscle. However, the majority of cerebellar subjects could not increase anticipatory activity across the three joints to control the initial impact of the ball. This deficit in the magnitude of anticipatory muscle activity was not due to abnormal timing of the anticipatory EMG. We saw no evidence of time shifting of the anticipatory response, as has been reported in studies of preparatory leg EMG related to arm lifting (Diener et al. 1989). We can only speculate that this discrepancy may be due to differences in the type of task performed. In the arm lifting task, preparatory leg EMG had to be scaled to the anticipated onset of the arm muscles. It is known that cerebellar damage can disrupt the relative timing of activity across multiple muscles (Hallett et al. 1975; Hore et al. 1991). In our catching task, activity of arm flexors had to occur in response to an external stimulus (ball drop). We saw no delays during this task, although anticipatory muscle activity was most often markedly reduced or absent. Presumably, the cerebellum is important in the generation of anticipatory EMG in both tasks, although its role may vary with the demands of the task.

Normal subjects rely to a much lesser extent on reflex activity to control arm displacement during catching (Bennett et al. 1994). We quantified reflex activity as postimpact EMG, which likely incorporated both short- and long-latency components. The increases in postimpact EMG that we saw in *Experiment 1* were likely due to larger reflexes elicited by the heavier ball, but may also have been due to increased gain of the long latency stretch reflex. Cerebellar subjects were able to increase their postimpact EMG, although not as effectively as controls. It has been previously shown that cerebellar subjects demonstrate abnormalities in the magnitude of the long-latency stretch reflex of muscles of the hand (Friedemann et al. 1987). These abnormalities varied with the location and extent of the lesion, such that people with hemispheric lesions showed increased long-latency reflexes and people with cerebellar le-

TABLE 4. Adaptation and performance coefficients under alternative conditions

Condition	AC	PC
Somatosensory and verbal cues* (<i>n</i> = 7)	2.3 \pm 0.4	12.8 \pm 1.3
With vision	1.8 \pm 0.2	14.7 \pm 1.3
Without vision (<i>n</i> = 6)	2.4 \pm 0.7	17.3 \pm 1.5

Values are means \pm SE. AC, adaptation coefficient; PC, performance coefficient. * Subjects were provided with somatosensory feedback (allowed to feel and hold the balls before the trials) and verbal cueing regarding when the balls were switched.

sions extending to the midline or with diffuse cerebellar atrophy showed decreased or variable responses.

Mechanisms of the anticipatory deficit

Several possible reasons could be advanced for the cerebellar group's inability to adapt their anticipatory EMG. One reason might be that cerebellar subjects were unable to detect the weight differential between the two balls. This is not the cause of slowed adaptation in our study because all subjects, including the entire cerebellar group, were easily able to perceive the weight difference between the two balls. More subtle deficits in kinesthesia could also contribute to the adaptation deficit. Cerebellar subjects have been shown to have slight deficits in the perception of velocity (difficulty distinguishing differences of $<15^\circ/\text{s}$) of finger displacements (Grill et al. 1994). Although we cannot rule out the possibility that very subtle kinesthetic deficits were present in our subjects, we speculate that they were not a primary cause of the adaptation deficit due to the large amplitude and velocity of joint movement during impact displacement. Switching from the light weight to the heavy weight tended to cause each of the three joints to move at velocities $>100^\circ/\text{s}$ (unpublished observations).

A second possibility is that the cerebellar subjects were weaker and thus unable to generate the necessary force to control the heavier ball. The cerebellar subjects we tested were able to generate peak forces comparable to control subjects in each of the six arm muscles tested. However, we did not test the rate of force production from these muscles. Others have suggested that cerebellar movement deficits may be due to decreases in the phasic components of force production (Topka et al. 1998). An inability to generate phasic muscular forces could theoretically contribute to the cerebellar group's increased adaptation coefficients and decreased percent returns when they had to adapt to heavier weights in *Experiment 1* (light-heavy-light) but would not explain the increased adaptation coefficients when they had to adapt to lighter weights in *Experiment 2* (heavy-light-heavy). Overall, the adaptation coefficients were not statistically different between *Experiments 1* and *2*. However, one cerebellar subject (*CBL-01*) had an adaptation coefficient that was much higher for *Experiment 1* versus *Experiment 2* (66.2 vs. 20.6, see Table 3) and a percent return value of only 50% for *Experiment 1*. This subject illustrates a potential additive effect of the two deficits; abnormalities in the generation of phasic muscle activity may have occurred in combination with an inability to adjust muscle activity through trial-and-error practice in adapting to the heavy weight.

A third potential explanation for the slowed adaptation is that cerebellar subjects had difficulty estimating when the ball was going to hit their hand. This could be due to an inability to perceive velocity and time (Ivry and Diener 1991; Ivry and Keele 1989; Nichelli et al. 1996), or to abnormal smooth pursuit eye tracking. These deficits could cause impaired performance of ball catching but would not necessarily alter the subject's ability to adapt when the ball was switched. Impairments in velocity and time perception would result in difficulty catching the ball in all *phases*, given that the balls were dropped from the same height (thus at the same velocity), regardless of weight. Our results show that the performance

variability of the cerebellar subjects was not different from that of control subjects for this task. Smooth pursuit deficits should not have contributed appreciably because subjects in both groups tended to fix their eyes on their catching hand rather than track the falling ball.

Cerebellar contributions to catching

We suggest that the cerebellum is involved in adapting anticipatory muscle activity to compensate for the effects of external forces on a voluntary limb movement. The cerebellum may be of particular importance in modulating the activity of multiple muscles crossing linked joints (Bastian et al. 1996; Thach et al. 1992). Single-cell recordings in awake, behaving monkeys show that cerebellar contributions to anticipatory processing are plausible. Dugas and Smith (1992) demonstrated increased simple spike activity of Purkinje cells in anticipation of a downward perturbation to a hand-held device. Preparatory activity in these neurons appeared gradually with predictable repetition of the same perturbation and did not disappear immediately when the perturbations were withdrawn. It is also known that activity in the dentate nucleus can occur before movement and before motor cortical activity, especially when a limb movement is triggered by a visual input (Lamarre et al. 1983; Thach 1975, 1978). Early dentate activity can be modified by the motor set of the animal (Strick 1983). Lesions of the dentate can produce delays in the onset of motor cortex cell activity (Meyer-Lohman et al. 1975; Spidalieri et al. 1983). This delay appears to be due to loss of phasic rather than tonic dentate activity (Meyer-Lohman et al. 1975).

Our idea that the cerebellum is involved in anticipating the effects of external forces is in contrast to findings from a study of automatic postural responses (Timmann and Horak 1997). Timmann and Horak (1997) propose that the cerebellum controls the gain of the postural response, but does not predict the magnitude of the postural perturbation. However, the cerebellum may play very different roles in adjusting automatic postural responses versus adjusting voluntary limb movements. Cerebellar damage does not disrupt sequencing of muscle activity in postural responses to perturbations (Horak and Diener 1994), but does disrupt sequencing and magnitude of muscle activity during voluntary limb movements (Hore et al. 1991; Manto et al. 1994). It may be that the organization of postural responses is hardwired in brain stem and spinal structures (Timmann and Horak 1997), whereas the organization of voluntary limb movements is under some cerebellar influence.

We have shown that adjusting anticipatory muscle activity to different external forces normally requires trial-and-error practice and the adaptation is stored, as demonstrated by a negative aftereffect. Explicit information about the time and magnitude of the weight switch was not sufficient to allow control subjects to catch the ball within the window on the first trial. Cerebellar subjects were unable to differentially increase the anticipatory muscle activity across three joints to catch balls of different weight. This resulted in slowed adaptation and little storage. Together, these findings have two implications. First, subjects require practice of this task to make the correct anticipatory adjustment. Control subjects could not estimate the effect of the dropped ball on the limb [for the 1st adaptation trial(s)] using stored information about the weight of the ball only. Second, the cerebellum is necessary for adapting anticipatory

muscle activity across multiple joints and for storage of this adaptation. However, these data do not show whether the storage occurs within or outside the cerebellum. We suggest that the cerebellum plays an important role in combining the appropriate anticipatory activity across several muscles and modifying that muscle activity in response to changing demands through trial-and-error practice.

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