

Cerebellar Ataxia: Torque Deficiency or Torque Mismatch Between Joints?

A. J. BASTIAN,^{1,2} K. M. ZACKOWSKI,^{1,2} AND W. T. THACH¹⁻³

¹Program in Physical Therapy, ²Department of Anatomy and Neurobiology, and ³Department of Neurology and Neurological Surgery, Washington University School of Medicine, St. Louis, Missouri 63108

Bastian, A. J., K. M. Zackowski, and W. T. Thach. Cerebellar ataxia: torque deficiency or torque mismatch between joints? *J. Neurophysiol.* 83: 3019–3030, 2000. Prior work has shown that cerebellar subjects have difficulty adjusting for interaction torques that occur during multi-jointed movements. The purpose of this study was to determine whether this deficit is due to a general inability to generate sufficient levels of phasic torque inability or due to an inability to generate muscle torques that predict and compensate for interaction torques. A second purpose was to determine whether reducing the number of moving joints by external mechanical fixation could improve cerebellar subjects' targeted limb movements. We studied control and cerebellar subjects making elbow flexion movements to touch a target under two conditions: 1) a *shoulder free condition*, which required only elbow flexion, although the shoulder joint was unconstrained and 2) a *shoulder fixed condition*, where the shoulder joint was mechanically stabilized so it could not move. We measured joint positions of the arm in the sagittal plane and electromyograms (EMGs) of shoulder and elbow muscles. Elbow and shoulder torques were estimated using inverse dynamics equations. In the shoulder free condition, cerebellar subjects made greater endpoint errors (primarily overshoots) than did controls. Cerebellar subjects' overshoot errors were largely due to unwanted flexion at the shoulder. The excessive shoulder flexion resulted from a torque mismatch, where larger shoulder muscle torques were produced at higher rates than would be appropriate for a given elbow movement. In the shoulder fixed condition, endpoint errors of cerebellar subjects and controls were comparable. The improved accuracy of cerebellar subjects was accompanied by reduced shoulder flexor muscle activity. Most of the correct cerebellar trials in the shoulder fixed condition were movements made using only muscles that flex the elbow. Our findings suggest that cerebellar subjects' poor shoulder control is due to an inability to generate muscle torques that predict and compensate for interaction torques, and not due to a general inability to generate sufficient levels of phasic torque. In addition, reducing the number of muscles to be controlled improved cerebellar ataxia.

INTRODUCTION

Recent work has shown that cerebellar ataxia during multi-jointed reaching may be due to an inability to adjust for dynamic interaction torques (Bastian et al. 1996; Topka et al. 1998a,b). Interaction torques are the mechanical consequence of moving limb segments that are linked together. During a reach, elbow movement causes an interaction torque to occur at the shoulder, and shoulder movement causes an interaction torque to occur at the elbow. Interaction torques may assist or oppose the desired movement at each joint depending on direction, velocity and acceleration of joint movements. Cerebellar subjects have difficulty offsetting interaction torques

especially during fast, multi-jointed reaching movements (Bastian et al. 1996; Topka et al. 1998b). Specifically, the torque produced by the muscles did not counter the interaction torque appropriately, allowing interaction torques to contribute excessively to the generation of the movement (Bastian et al. 1996). This resulted in an abnormal pattern of reaching, with the elbow and shoulder joints moving at inappropriate rates relative to one another and the fingertip overshooting the target (Bastian et al. 1996; Topka et al. 1998a).

When asked to move slowly and accurately, cerebellar subjects reached using patterns that were either similar to controls (Topka et al. 1998a,b) or were "decomposed" (Bastian et al. 1996). Decomposition means that subjects moved the shoulder while fixing the elbow, then moved the elbow while fixing the shoulder (Holmes 1939). Decomposition was hypothesized to represent a compensatory strategy to improve accuracy by voluntarily fixing all but one joint. The decomposition would have reduced the interaction torques occurring at the moving joint, although interaction torques would still have occurred at the fixed joint (Bastian et al. 1996).

Based on these studies, it has been suggested that a major role of the cerebellum is in the generation of muscle torques that predict and compensate for interaction torques caused by other moving joints (Bastian et al. 1996; Schweighofer et al. 1998a,b). Under this hypothesis, we propose that cerebellar subjects should have difficulty with fast movements that produce greater interaction torques because they cannot precisely activate multiple muscles to offset or accommodate the anticipated interaction torques. Thus cerebellar subjects theoretically could under- or overcompensate for interaction torques. Cerebellar deficits, such as dysmetria, should be also be greatly improved when interaction torques are eliminated.

Others have proposed that this impairment is a result of a generalized inability to quickly generate the appropriate muscle torque levels (Boose et al. 1999). If this second hypothesis is correct, then cerebellar subjects should have a general deficit in producing phasic torques, which would account for their inability to offset interaction torques. Overall, one may expect that cerebellar subjects would consistently produce smaller muscle torques at much lower rates than controls. This reduction in torque rate should account for the types of errors observed during the movement.

Either of these two mechanisms could conceivably explain an inability to adjust for the larger interaction torques generated during fast reaching movements. To distinguish between these possibilities, we studied controls and cerebellar subjects making targeted elbow flexion movements in two different conditions. In the *shoulder free condition*, subjects pointed to a target located at a position that required movement at the

The costs of publication of this article were defrayed in part by the payment of page charges. The article must therefore be hereby marked "advertisement" in accordance with 18 U.S.C. Section 1734 solely to indicate this fact.

TABLE 1. *Subject demographics*

Subject	Age	Lesion	Length of Illness, yr	Ataxia		Tremor	
				Arm	Stance/gait	Arm	Stance/gait
<i>CBL01</i>	60	Pancerebellar cortical atrophy	10	Moderate	Moderate	None	None
<i>CBL02</i>	41	Pancerebellar cortical atrophy	6	Minimal	Moderate	None	Minimal
<i>CBL03</i>	42	Pancerebellar cortical atrophy	5	Moderate	Severe	Minimal	Minimal
<i>CBL04</i>	37	Pancerebellar cortical atrophy	2	Severe	Severe	Moderate	Moderate
<i>CBL05</i>	56	Pancerebellar cortical atrophy	3	Moderate	Moderate	Minimal	None
<i>CBL06</i>	39	OPCA	10	Severe	Severe	Moderate	Moderate
<i>CBL07</i>	32	SCA7	5	Minimal	Moderate	Moderate	Moderate
<i>CBL08</i>	73	SCA6	6	Severe	Severe	Minimal	Minimal

OPCA, olivopontocerebellar atrophy; SCA, spinocerebellar ataxia.

elbow joint only, although the shoulder was free to move. Movements in the *shoulder fixed condition* were to the same target position, and the shoulder was mechanically stabilized so it could not move. In the shoulder free condition, cerebellar subjects over- and undershot the target; in shoulder fixed condition, cerebellar subjects made errors comparable to controls. Cerebellar overshoot errors in the shoulder free condition were primarily due to excessive shoulder flexion. The excessive shoulder flexion resulted from larger shoulder muscle torques produced at higher rates than would be appropriate for a given elbow movement. This mismatch occurred early in the movement (within the 1st 100 ms). These findings are consistent with the first hypothesis, that the cerebellum is involved in generating muscle torques that predict and compensate for interaction torques. It is not consistent with the second hypothesis because cerebellar subjects often produced greater shoulder muscle torques at a similar rate as controls.

Improvement in cerebellar subjects' performance in the shoulder fixed condition was accompanied by a significant reduction in shoulder flexor electromyogram (EMG) compared with controls. Our findings indicate that cerebellar subjects have greater difficulty in moving accurately when they have to control the muscles acting at more than one joint. As a consequence, reducing the number of muscles to be controlled appears to improve cerebellar ataxia. Preliminary results of this work have been previously reported (Bastian et al. 1997).

METHODS

Subjects

Eight control subjects and eight subjects with cerebellar cortical atrophy participated in this study. Presence of cerebellar damage was assessed by neurological examination and magnetic resonance imaging (MRI) scan. In addition, results of genetic testing were sometimes available. A clinical neurological examination was done on each subject to check for involvement of other systems and to assess the severity of ataxia (Table 1). One subject (*CBL02*) showed a Babinski's reflex on the right side, but no other clinical signs of corticospinal involvement (e.g., no increased muscle tone of arms and legs). One subject with olivopontocerebellar atrophy (OPCA) had probable degeneration in the cerebellum, pons, and inferior olive. Two other subjects had hereditary spinocerebellar ataxias (SCA6 and SCA7), which have exclusively or predominantly cerebellar involvement. The subject with SCA7 had no chorea or pyramidal signs. Control subjects were matched for age, sex, handedness, and arm used to perform the movement. Informed consent was acquired prior to testing all subjects.

Paradigm

All subjects performed elbow flexion movements in the sagittal plane under shoulder free and shoulder fixed conditions (Fig. 1). Prior to testing, reflective markers were placed on the tip of index finger, hand (index metacarpalphalangeal joint), wrist (dorsal joint surface), elbow (lateral epicondyle), and shoulder (tip of acromion process). Bipolar, surface EMG electrodes were placed over the anterior deltoid (AD), posterior deltoid (PD), biceps (BI), brachioradialis (BR), and triceps (TRI). Two 4-cm ball markers suspended from flexible plastic wires indicated the start and target positions for the reaching movements under both conditions.

SHOULDER FREE CONDITION. Subjects were seated with their back supported and the arm positioned at the side with the shoulder in 0–10° of flexion, elbow in 80–90° flexion, and forearm in neutral pronation-supination (Fig. 1A). The target was located at a position that required 35° of elbow flexion and no shoulder movement. During this condition the shoulder was free to move. All subjects were asked to reach out and touch the target as fast as they could, while trying to stop on the target. Subjects were instructed to "move only the arm without leaning forward." Subjects were not specifically told to hold the shoulder joint steady. Three to 5 practice trials were given prior to 10 test trials.

SHOULDER FIXED CONDITION. This condition was identical to the shoulder free condition, except that the elbow rested on a flat surface (Fig. 1B). This position served to mechanically constrain movement at the shoulder and allow only the elbow to move. Three to 5 practice trials were given prior to 10 test trials.

Data collection

All movements were videotaped at 60 fields per second. The video camera was positioned to record the movement in the sagittal plane

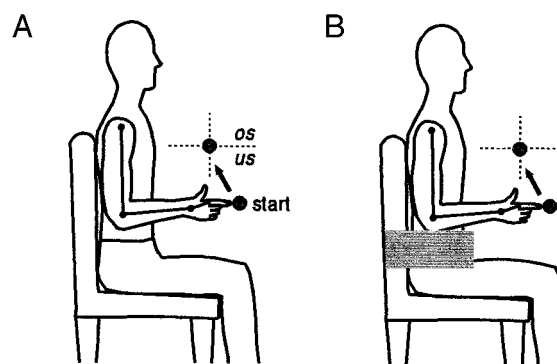


FIG. 1. Diagram of the task. *A*: shoulder free condition. *B*: shoulder fixed condition. Endpoint error was classified as an overshoot (OS) when the finger traveled above a horizontal line through the target and an undershoot (US) when the finger fell below a horizontal line through the target.

and zoomed in to provide the largest image possible while maintaining all markers in the field of view. Video data were digitized at 60 Hz to determine marker displacements (Peak Performance Technologies). EMG signals were time locked to the video data, amplified to obtain a strong signal, and collected at 600 Hz.

Analysis

KINEMATICS. We analyzed five test trials from each subject moving in each condition. Equal numbers of movements were used from each control and cerebellar subject in the overall analysis. When possible, we chose the first five trials because these would be the trials least likely affected by fatigue. During digitization of the trials, we occasionally found a movement that was clearly out of the sagittal plane, and this movement was then rejected. This was determined by measuring the apparent length change of the forearm during the movement. Apparent length changes of up to 10% were considered acceptable for this type of analysis (Hoy et al. 1985). Few trials were rejected for this reason (<7 in both groups across both conditions).

Marker displacement data were smoothed using a fourth-order Butterworth filter (low-pass) with a cutoff frequency of 5 Hz. Linear and angular displacements at the shoulder, elbow, and wrist were calculated from the marker positions. Linear and angular displacement data were numerically differentiated to calculate velocity and acceleration. The “start of movement” was defined as the time and position at which the wrist tangential velocity exceeded 5% of its peak. The “end” of the first phase of movement was defined as the time and position at which the wrist tangential velocity dropped below 5% of its peak. In a few trials, the wrist tangential velocity did not drop down to 5% of its peak prior to subsequent peaks (corrective movements). In these cases, the end of movement was taken as the lowest point following the peak. All reaching movements were then analyzed from the start of the movement to the end of the first phase of the movement, prior to any corrective movements.

Each trial performed by cerebellar subjects was classified into different “error categories” by assessing the magnitude and direction of endpoint error as No Error (comparable to control subject errors), an overshoot (OS) or an undershoot (US). The magnitude of the endpoint error was measured as the absolute distance between the tip of the index finger and the center of the target at the end of movement. A cerebellar trial was classified as No Error if the absolute magnitude of endpoint error was within the mean \pm 2 SD of control errors for that condition. If a trial was out of this limit, it was classified as an OS if it fell above the horizontal axis running through the target or as an US if it fell below the horizontal axis running through the target (Fig. 1). Additional kinematic and kinetic analysis were then done to compare the different patterns of movement associated with the error categories.

Kinematic measures of interest were 1) errors of elbow and shoulder angle and 2) peak tangential wrist velocity. Errors of the elbow and shoulder angles were calculated by taking the difference between the actual joint angle at the end of movement and the “ideal” joint angle required to hit the target (0° of movement for the shoulder, 35° flexion for the elbow). Peak tangential wrist velocity was chosen between the start and end of the movement and was our measure of reach velocity.

We used a repeated measures ANOVA to assess differences in endpoint error magnitude between groups, conditions, and to test for group \times condition interactions. One-way ANOVAs were used to test for differences in errors of joint angle and reach velocity associated with the various categories of error (OS, US, or No Error). Post hoc comparisons were made using Duncan’s test.

TORQUE ANALYSIS. Using videotaped kinematic data together with estimates of the inertial mass of the different limb segments (Winter 1990), we employed inverse dynamics equations to calculate the time series of the 1) net torque, 2) gravitational torque, 3) dynamic interaction torque, and 4) muscle (residual) torque about both the elbow

and shoulder joints (Bastian et al. 1996). We define these terms in the following way.

The *net torque* is defined as the sum of all of the torques acting at a joint. We estimated the net torque by taking the product of the moment of inertia of the involved segments (including the segment under consideration and all segments distal to it) and the angular acceleration around a given joint. The *gravitational torque* is the torque produced by the force of gravity acting on the limb segments of the arm. Note that this torque was due to the force of gravity acting on *all* limb segments distal to the joint in question. The *dynamic interaction torque* is the “passive” mechanical torque generated when two or more linked segments move on one another. The *muscle (residual) torque* is produced by muscle and passive tissues; it is calculated by taking the difference between the net torque and the sum of the interaction and gravitational terms. For a full mathematical description of the inverse dynamics equations used for this calculation, see Bastian et al. (1996).

In this study, we describe only shoulder and elbow torques because 1) movement of these two joints primarily determined the course of the reaching movement and 2) wrist movement was inconsistently present, and most often absent, during the first phase of the movement. However, we included movement of the wrist joint in the calculation of torques occurring at the elbow and shoulder joints by use of a three-segment inverse dynamics model (Bastian et al. 1996).

Prior to analysis, we aligned all torque traces on the start of movement and normalized individual subject’s torques to their body weight. Normalization allowed us to directly compare the magnitude of the different torque components produced by subjects of different size. Measures of interest for both shoulder and elbow joints were 1) the magnitude of all torque components at the time of the peak flexor muscle torque, 2) the time of the peak flexor muscle torque and peak extensor interaction torque, and 3) the rate of flexor muscle torque production. We measured torque magnitudes to determine whether the peak muscle torque was too little or too large relative to interaction and gravitational torques at that time. The relative time of the peak flexor muscle torque and peak extensor interaction torque was used to assess timing deficits in torque generation. The rate of torque production was calculated by dividing the change in muscle torque magnitude from start to the flexor peak by the time it took to reach the peak. Differences in kinematic and kinetic measurements associated with the various categories of error (OS, US, or No Error) were tested using one way ANOVAs. Post hoc comparisons were made using Duncan’s test.

EMG. Raw EMG signals from all muscles were rectified and low-pass filtered at 100 Hz. A preliminary analysis was then done to determine whether each muscle was active during a given trial. A muscle was considered active if it increased 2 SD above a baseline measure (taken 500 ms prior to the go signal) for more than 40 ms. Further analysis determined differences in the muscle activity levels of the AD, BI, and BR between shoulder free and shoulder fixed conditions. We limited our analysis to these three muscles because 1) the BI and BR are the agonists for elbow flexion and were active 100% of the time, 2) the AD must be co-active with BI and BR to counteract shoulder interaction torques caused by early elbow flexion in the shoulder free condition, and 3) the PD and TRI (antagonists) were inconsistently active in both groups for both conditions (Table 2, see RESULTS). We integrated EMG signals in the AD, BI, and BR for the first 100 ms of movement, and then calculated the percent change in muscle activity from the shoulder free to the shoulder fixed conditions. Student’s *t*-tests were used to test whether the percent change in muscle activity was different between the control and cerebellar groups.

RESULTS

Endpoint error

Endpoint errors for control and cerebellar groups are shown in Fig. 2. Control subjects made small endpoint errors that were

TABLE 2. EMG activity

	Percent of Trials With EMG Activity			TRI
	AD	PD	BI/BR	
Shoulder free				
Control	100	53	100	53
Cerebellar	90	18	100	18
Shoulder fixed				
Control	68	52	100	63
Cerebellar	34	8	100	15

AD, anterior deltoid; PD, posterior deltoid; BI, biceps; BR, brachioradialis; TRI, triceps.

similar in both conditions. Cerebellar subjects often produced large endpoint errors in the shoulder free condition, but were able to perform at nearly control levels in the shoulder fixed condition. There was a significant group \times condition interaction for endpoint error magnitude ($P < 0.0005$).

The majority of cerebellar errors in the shoulder free condition were overshoots. The cerebellar group produced 40% OS errors, 17% US errors, and 43% classified as No Error. Note that three of eight cerebellar subjects produced both over- and undershoot errors. These subjects tended to overshoot the target on the first trial and then undershoot the target on the subsequent trial. Cerebellar endpoint errors were dramatically reduced in the shoulder fixed condition, with only 15% OS errors, 7% US errors, and 78% classified as No Error.

Movement patterns associated with errors

KINEMATICS. Figure 3A shows elbow (■) and shoulder (□) angular errors made in the shoulder free condition and Fig. 3B shows elbow angular errors made in the shoulder fixed condition. Angular errors at both joints were normalized so that the desired end angle was 0°. Flexor deviations from the desired angle are represented as positive values, and extensor deviations are represented as negative values. In the shoulder free condition, control subjects flexed the shoulder slightly even though movement of this joint was not required (Fig. 3A, □). Cerebellar subjects flexed the shoulder excessively when they made OS errors, but were comparable to controls in all other categories. Shoulder flexion movements in the OS category were significantly larger than those made in all other categories (all $P < 0.0005$). Figure 4 shows that shoulder flexion began concurrently with elbow flexion and was not a reaction to an early shoulder extension movement. At the elbow, cerebellar subjects often flexed too much when they made OS errors and flexed too little when they made US errors (Fig. 3A, ■). Elbow movements in the OS and US categories were significantly different from each other, and from all other categories (all $P < 0.0005$). In the shoulder fixed condition, cerebellar subjects flexed the elbow too much when they made OS errors and too little when they made US errors (Fig. 3B, ■). Elbow movements in the OS and US categories were significantly different from each other, and from all other categories (all $P < 0.0005$).

Figure 3, C and D, shows peak tangential wrist velocities produced for each error category. The overall peak tangential wrist velocity was not significantly different in the shoulder free condition compared with the shoulder fixed condition for either group. Velocity magnitudes for the different error cate-

gories are also comparable across both conditions. Cerebellar subjects moved faster than controls when they made OS errors, slower than controls when they made US errors, and slower than controls when they made No Errors. In the shoulder free condition, peak tangential wrist velocity for OS errors was significantly different from for US errors and No Error ($P < 0.05$), but not different from control values. In the shoulder fixed condition, peak tangential wrist velocity for OS errors was significantly different from for US errors ($P < 0.05$) only.

TORQUE ANALYSIS. The cerebellar group produced the most abnormal torque magnitudes in the shoulder free condition. The major abnormality was at the shoulder joint when they made OS errors. Figure 5 shows single trials of torques (normalized to body weight) and EMG from a control subject and a cerebellar subject in the OS category. Trials are matched for peak tangential wrist velocity. The control subject's shoulder muscle torque is nearly equal and opposite the interaction torque in shape, and offset by an amount that counteracts the nearly constant gravitational torque (Fig. 5A). Because of this, the control subject produced a fairly flat net shoulder torque. The cerebellar subject produced a peak shoulder muscle torque that was too large and slightly too late to counter the interaction torque (Fig. 5B). This abnormality occurred within the first 100 ms of the movement. Because of this early imbalance, cerebellar subjects produced a substantial net shoulder torque, and flexed excessively at the shoulder. Also illustrated in Fig. 5 are the EMG patterns recorded from the same trials. Note that the control subject produced an initial burst of activity in the AD that coincided in time with the initial burst of activity in the BI and BR (Fig. 5C). The cerebellar subject produced more tonic levels of AD activity that did not coincide with

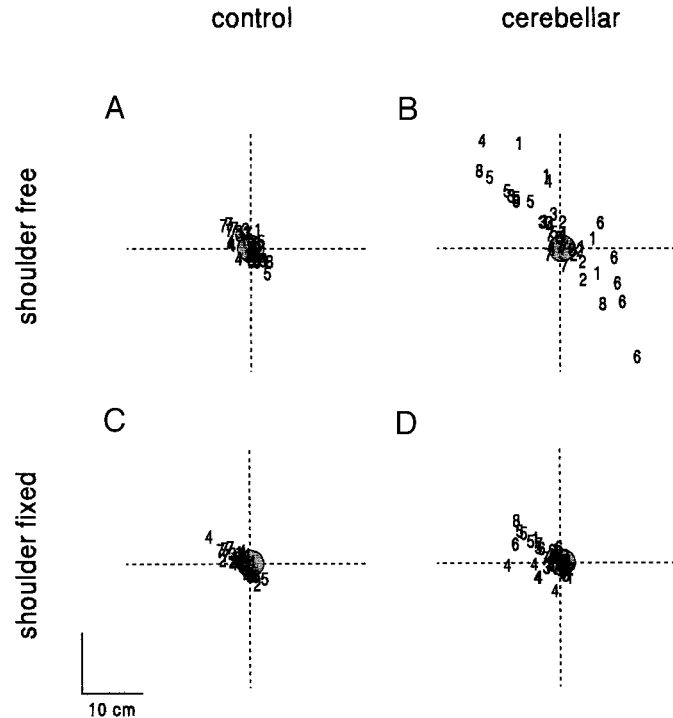


FIG. 2. Endpoint error for the control group, shoulder free condition (A); cerebellar group, shoulder free condition (B); control group, shoulder fixed condition (C); cerebellar group, shoulder fixed condition (D). Index finger endpoints are plotted in a parasagittal plane, relative to the target (gray ball). Each number represents the endpoint location a different subject; 5 trials for each subject are shown.

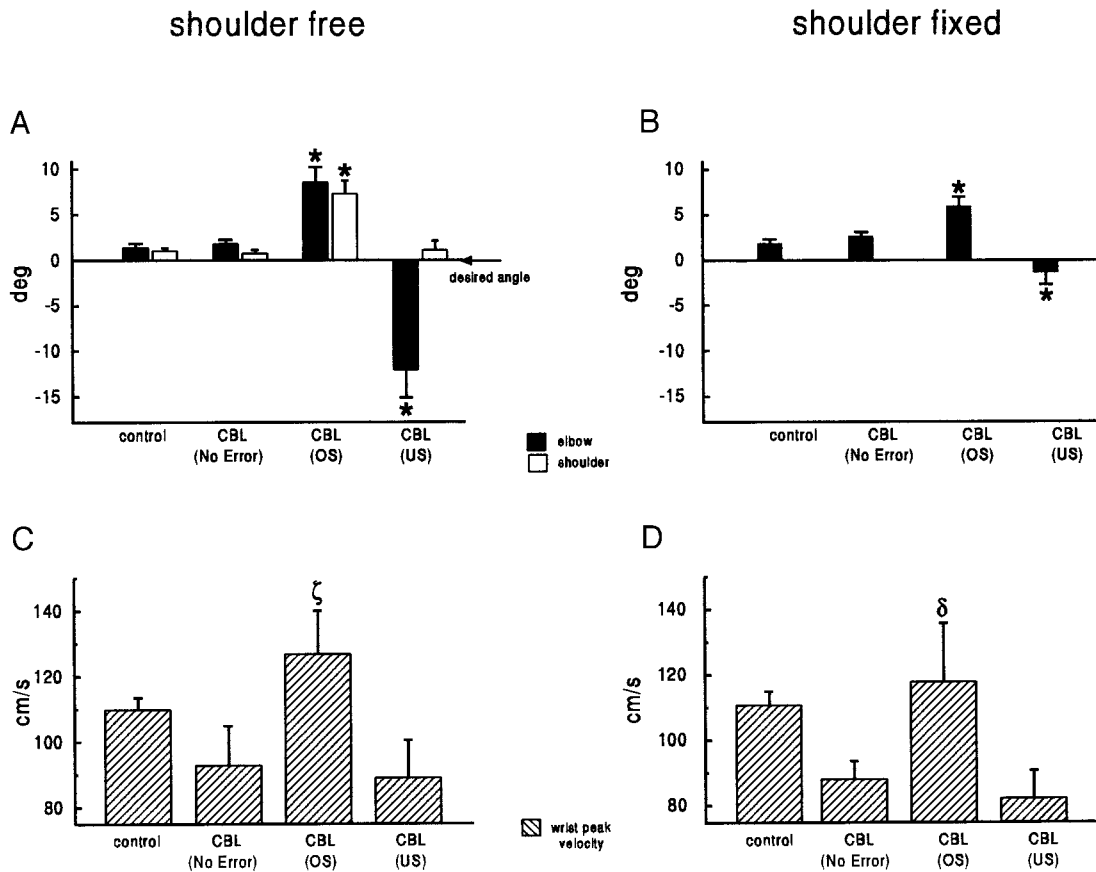


FIG. 3. *A* and *B*: joint angular errors for the different categories in the shoulder free (*A*) and shoulder fixed conditions (*B*). Bars represent mean \pm SE. The angular position of both joints has been normalized to make the desired end angle equal to 0°. Flexor deviations from the desired angle are represented as positive values, and extensor deviations are represented as negative values. * Significant difference from all other categories. *C* and *D*: peak tangential wrist velocities for the different categories in the shoulder free (*C*) and shoulder fixed conditions (*D*). ζ , significant difference between the OS category and the US and No Error categories only. δ , significant difference between the OS and US categories only.

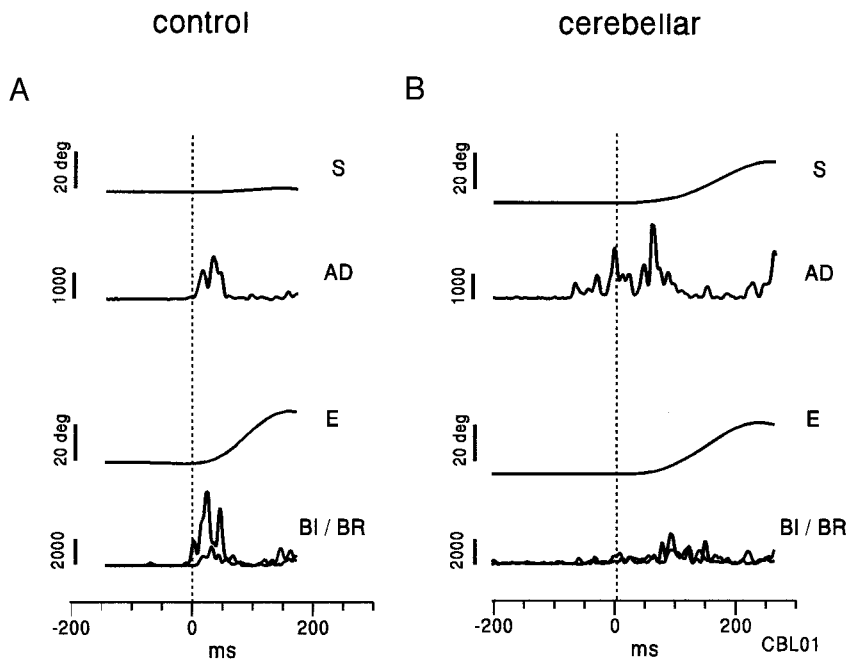


FIG. 4. Single trial of elbow and shoulder joint movements and electromyogram (EMG) for a control subject (*A*) and a cerebellar subject (*B*) who overshoot the target. Both trials are from the shoulder free condition. Labels are as follows: S, shoulder angle; E, elbow angle; AD, anterior deltoid; BI, biceps; BR, brachioradialis. Flexor direction joint movement is in the upward direction. Data are aligned on start of movement (*time 0*) and are from movements of similar peak tangential wrist velocity (control 162 cm/s; cerebellar 189 cm/s). Note that the cerebellar subject begins to move the shoulder in the flexor direction concurrently with the elbow flexion movement. Shoulder flexion late in the movement is not a response to an initial extensor direction shoulder movement.

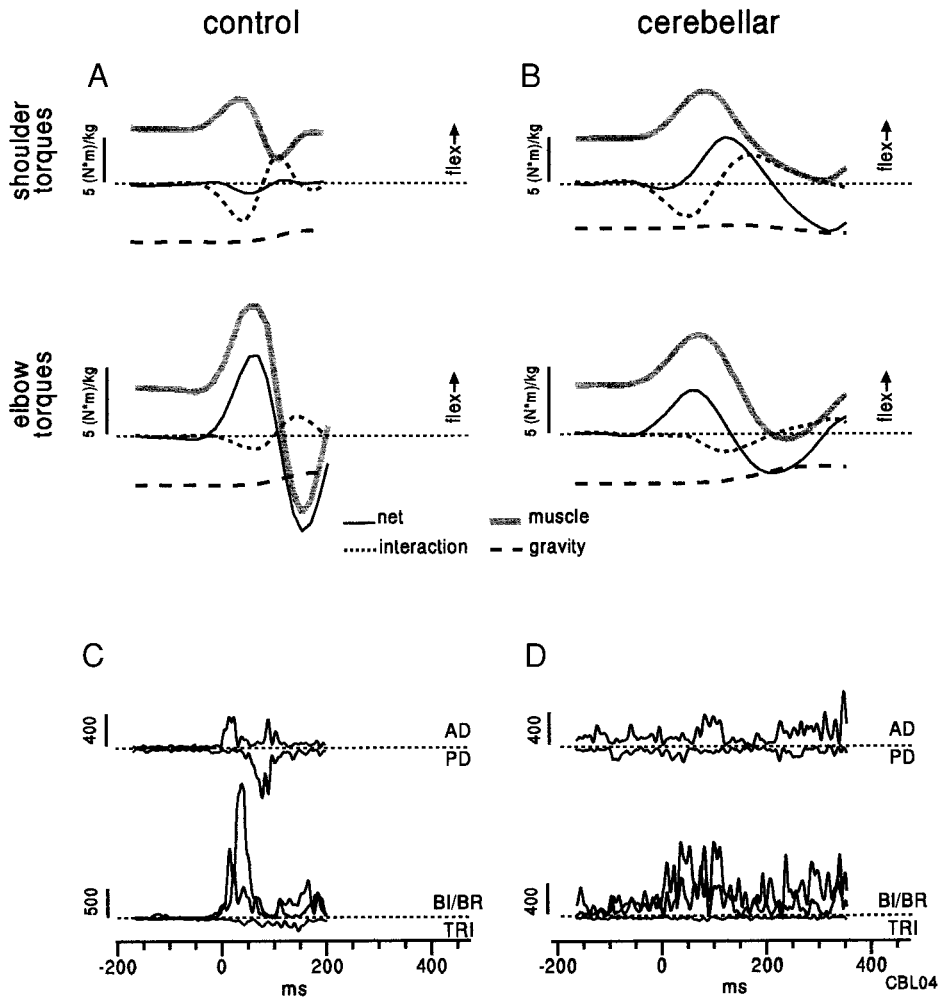


FIG. 5. Single trial of normalized elbow and shoulder torque components for a control (A) and a cerebellar subject (B), both moving in the shoulder free condition. The cerebellar subject shown overshoot the target in this trial. Data are from movements of similar peak velocity (control 161 cm/s, cerebellar 167 cm/s). EMG traces for the same trial are shown in C and D. Trials are aligned on the start of movement (*time 0*) and drawn to the end of movement, prior to corrections.

the initiation of BI/BR activity (Fig. 5D). Analysis of EMG changes from the shoulder free to shoulder fixed conditions will be presented in a later section.

To assess torque magnitude differences across error categories, we measured each torque component (muscle, interaction, and gravitational torques) at the time of the peak flexor muscle torque for each joint. This allowed us to determine whether the peak muscle torque was inadequate or excessive relative to the levels of interaction and gravitational torques occurring at that time. Figure 6, A and B, shows the magnitudes of the muscle, interaction, and gravity torques produced at the shoulder and elbow joints in the shoulder free condition. We found that the shoulder muscle torque was larger in the OS category than in all other categories (all $P < 0.02$). Shoulder interaction and gravitational torques were not different between categories. The elbow muscle torque was slightly larger in the OS category (Fig. 6B), although this difference was not statistically significant. However, the interaction torque at the elbow was larger in the OS category than in all other categories (all $P < 0.02$). Larger elbow interaction torques are not surprising, since the shoulder moved excessively when cerebellar subjects produced OS errors. Finally, elbow gravitational torques were larger than control values in the OS and No Error categories (all $P < 0.01$). This difference likely reflects the fact that, at this time point, the forearm was oriented in slightly different positions with respect to gravity.

Figure 6C shows the magnitudes of the muscle and gravity torques produced at the elbow joint in the shoulder fixed condition. The elbow muscle torque was slightly larger and more variable in the OS category, although this difference was not statistically significant. There was no difference in the magnitude of the gravitational torque between the groups.

The timing of peak muscle and interaction torques were assessed in the shoulder free condition only; the shoulder fixed condition was not included in this analysis since the mechanical constraint prevented all shoulder torques and interaction torques at the elbow. Table 3 shows the time of the peak flexor muscle torque, peak extensor interaction torque (aligned on start of movement), and the difference between the two. At the shoulder, the time of the peak flexor muscle torque was within the first 100 ms for all groupings, but slightly later for the cerebellar OS and No Error groups compared with controls ($P < 0.01$), but the time of the peak interaction torque was not different between groups. As a result, the muscle-interaction time difference was nearly doubled in the cerebellar OS group compared with controls. At the elbow, the time of the peak flexor muscle torque was slightly later for the cerebellar OS group compared with controls ($P < 0.05$), but the time of the peak interaction torque and muscle-interaction difference was similar between the groups.

We analyzed the rate of flexor muscle torque production for both groups moving in both conditions. Figure 7A shows the

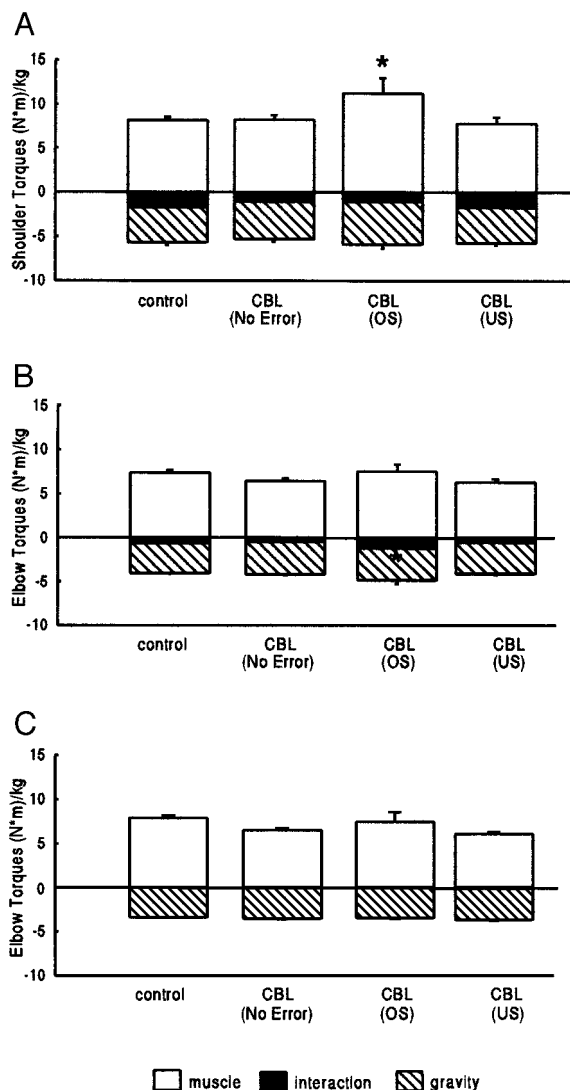


FIG. 6. Magnitude of torque components occurring at the time of the peak flexor muscle torque for each joint. Shoulder (A) and elbow torque components (B) are shown for the shoulder free condition. C: elbow muscle and gravitational torques for the shoulder fixed condition. Means \pm SE are shown for trials in each error category.

rate of flexor torque produced at the elbow and shoulder joints in the shoulder free condition. At the shoulder (\square), the cerebellar OS group demonstrated the highest rates of torque production. However, there was no statistical difference between the different categories due to the variability in the cerebellar OS group. At the elbow (\blacksquare), there was a significant effect of

category, with controls producing higher torque rates than the other groups ($P < 0.05$). Thus the most striking difference between the elbow and shoulder torque rates. The control group produced comparable torque rates at the elbow and shoulder joints (Fig. 7A). The cerebellar groups produced higher rates of torque at the shoulder compared with the elbow. Statistical comparison of the difference between shoulder and elbow torque rates showed that only cerebellar OS group was significantly different from controls ($P < 0.05$). This was due to the fact that the cerebellar OS group tended to produce slightly higher shoulder torque rates and lower elbow torque rates compared with controls.

Figure 7B shows the rate of flexor torque produced at the elbow in the shoulder fixed condition. There was a significant effect of error category, with higher torque rates in the control group compared with the US category ($P < 0.05$).

Torque magnitude and rate as a function of velocity

It is possible that our analysis of torques by error category could have failed to uncover a velocity-dependent impairment of torque generation in the cerebellar group. To test this, we compared the 1) peak flexor muscle torque magnitudes and 2) flexor muscle torque rates of control and cerebellar subjects moving at comparable velocities. We used the peak tangential wrist velocity as an index for dividing cerebellar and control trials into five different velocity categories: 40–70 cm/s, 71–100 cm/s, 101–130 cm/s, 131–160 cm/s, and 161+ cm/s. We then determined whether the elbow and shoulder torque magnitudes and rates were comparable in the different velocity categories across groups.

Figure 8A shows that control subjects scaled elbow and shoulder muscle torque magnitudes similarly with increasing velocity. Figure 8B shows that cerebellar subjects generated greater muscle torques at the shoulder compared with the elbow across velocity categories. This was most pronounced in the higher velocity categories, with the peak shoulder muscle torque greatly exceeding the peak elbow muscle torque. ANOVA showed that the difference between elbow and shoulder torque magnitudes was greater in the cerebellar group compared with controls ($P < 0.001$). Two findings were responsible for elbow-shoulder torque mismatch: 1) cerebellar subjects produced greater shoulder muscle torques than controls ($P < 0.005$) and 2) cerebellar subjects produced lower peak elbow muscle torques than controls ($P < 0.05$).

Figure 8C shows that control subjects also scaled elbow and shoulder muscle torque rates similarly with increasing

TABLE 3. Condition 1: peak torque times

	Shoulder			Elbow		
	Flexor muscle torque	Extensor interaction torque	Difference	Flexor muscle torque	Extensor interaction torque	Difference
Shoulder free						
Control	71 \pm 3	50 \pm 2	21 \pm 2	69 \pm 3	97 \pm 5	-27 \pm 3
Cerebellar, OS	97 \pm 6	59 \pm 4	38 \pm 5	88 \pm 5	122 \pm 13	-34 \pm 11
Cerebellar, US	85 \pm 2	62 \pm 5	23 \pm 3	78 \pm 3	100 \pm 4	-22 \pm 6
Cerebellar, No Error	94 \pm 6	58 \pm 4	36 \pm 4	78 \pm 4	104 \pm 8	-26 \pm 7

Values are means \pm SE in ms. OS, overshoot; US, undershoot.

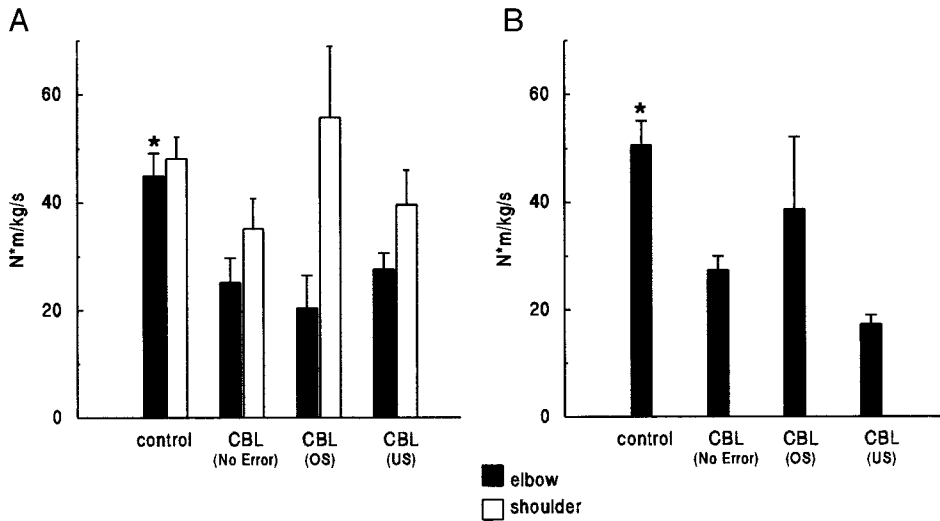


FIG. 7. Rate of flexor muscle torque production in each error category. A: shoulder free condition. B: shoulder fixed condition. ■, elbow muscle torque rates; □, shoulder muscle torque rate. Means ± SE are shown.

velocity. In contrast, Fig. 8D shows that cerebellar subjects produced higher torque rates at the shoulder compared with the elbow, and this difference increased with increasing velocity. ANOVA showed that the difference between elbow and shoulder torque rates was greater in the cerebellar group compared with controls ($P < 0.0001$). This was due to the fact that cerebellar subjects produced lower elbow

torque rates than controls ($P < 0.001$), and shoulder torque rates that were slightly higher or comparable to controls.

EMG differences between shoulder free and shoulder fixed conditions

As stated earlier, cerebellar endpoint error was reduced to near control levels when they moved in the shoulder fixed

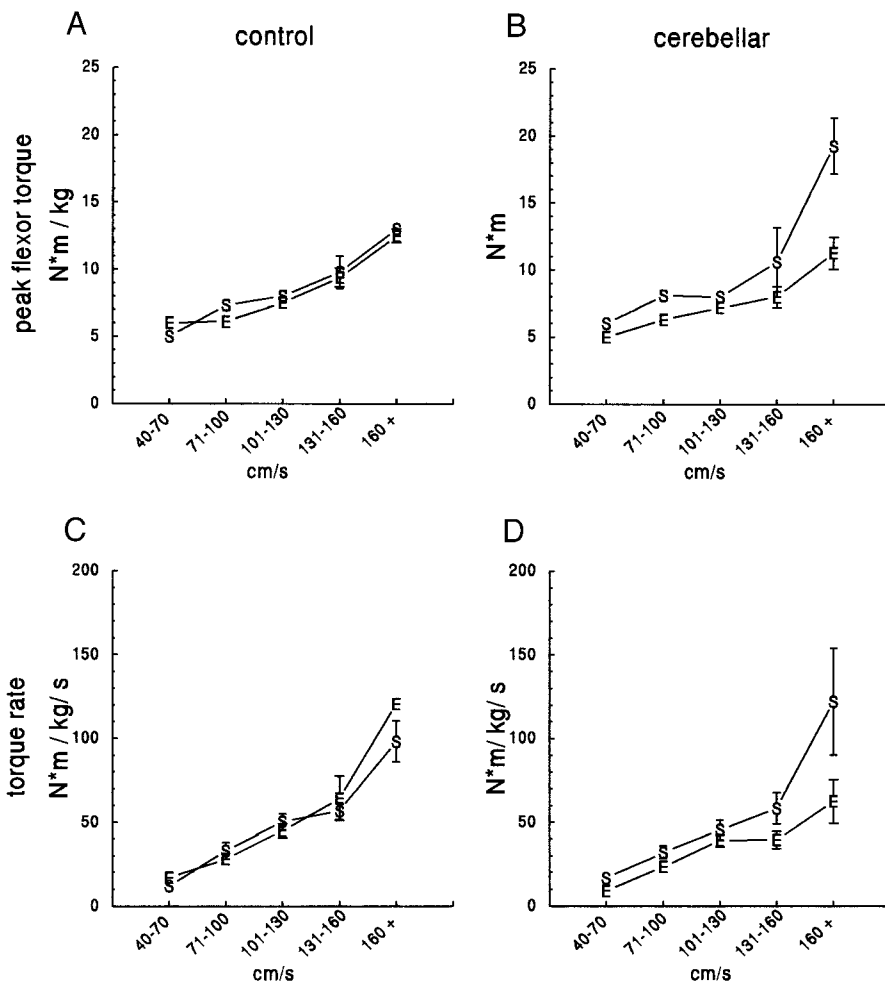


FIG. 8. Rate and magnitude of flexor muscle torque for different velocity movements. All movements are shown for the shoulder free condition. Peak flexor torque magnitudes at the shoulder (S) and elbow (E) from the control group (A) and the cerebellar group (B). Peak flexor torque rate at the shoulder (S) and elbow (E) from the control group (C) and the cerebellar group (D). Means ± SE are shown for each velocity category.

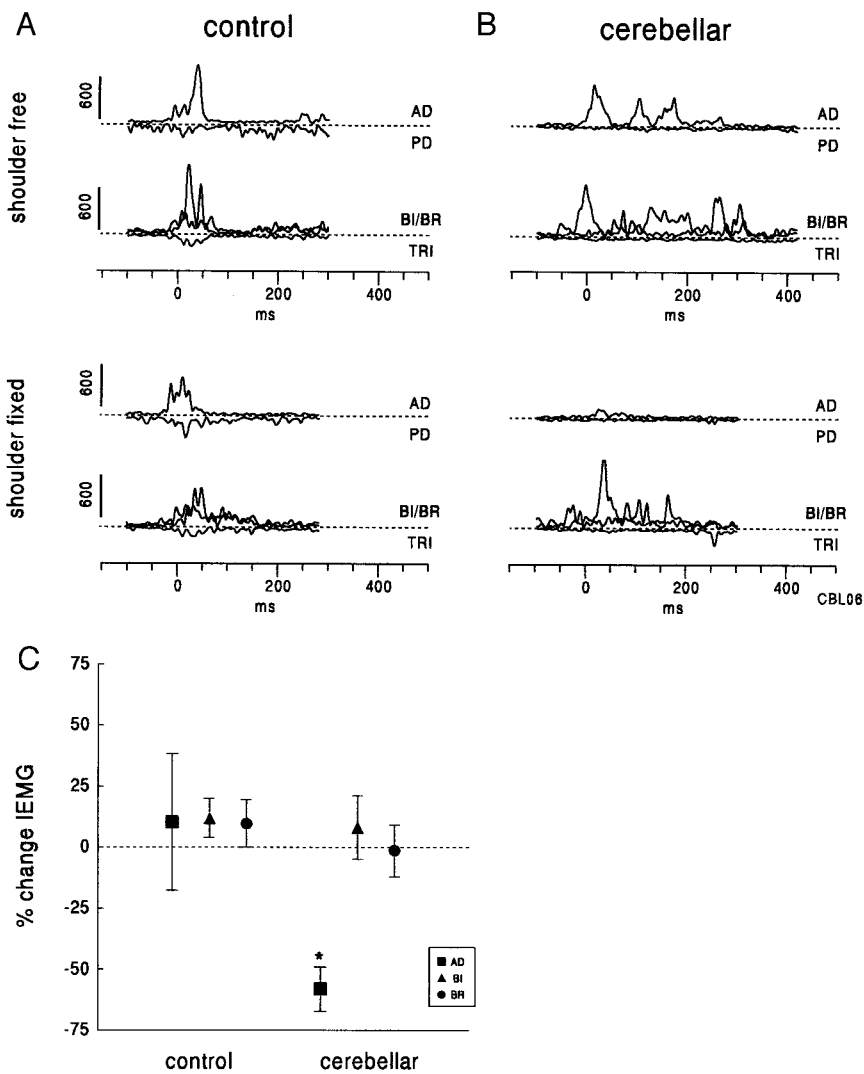


FIG. 9. Single trial of EMG from control (A) and cerebellar subjects (B), moving under the shoulder free condition (top) and shoulder fixed condition (bottom). Trials are aligned on the start of movement (time 0) and drawn to the end of movement, prior to corrections. All trials are matched for peak tangential wrist velocity (control, shoulder free = 103 cm/s; control, shoulder fixed = 101 cm/s; cerebellar, shoulder free = 99 cm/s; cerebellar, shoulder fixed = 116 cm/s). C: percent change (mean ± SE) in integrated EMG from the shoulder free to the shoulder fixed conditions. Negative changes indicate decreased muscle activity in the shoulder fixed condition.

condition (78% of trials were within control variability). Cerebellar subjects also showed a striking difference in the magnitude of EMG activity in the AD muscle. Figure 9A shows a single EMG trace from a control subject and a cerebellar subject moving in both conditions. All traces are aligned on start of movement (time 0) and matched for peak tangential wrist velocity. The cerebellar subject shown is highly representative of the group, with prolonged AD activity (either tonic or multiple phasic bursts) in the shoulder free condition, and nearly absent AD activity in the shoulder fixed condition. Control subjects strongly activated the AD in both conditions, despite shoulder fixation in the second condition. Figure 9C shows the percent change in integrated EMG activity of the AD, BI, and BR from the shoulder free to shoulder fixed conditions. Averages are shown for the control group and cerebellar group. Note that the control subjects increased the magnitude of activity in all three muscles. Cerebellar subjects increased EMG magnitude in the BI muscles, showed little change in the BR, and greatly decreased activity in the AD. AD activity was decreased by 58% in cerebellar group and increased by 10% in the control group ($P < 0.05$). Reduced AD activity did not appear to be due to a general slowing of the reach in the cerebellar group. The average peak tangential wrist

velocity for the entire cerebellar group decreased by about 12% from the shoulder free (105 ± 12 cm/s) to shoulder fixed (92 ± 10 cm/s) conditions. Controls produced comparable peak wrist velocities in both conditions (shoulder free 110 ± 7 cm/s; shoulder fixed 110 ± 9 cm/s).

We did not systematically analyze the magnitude of PD and TRI activity because these muscles were inconsistently active in both groups moving under both conditions (Table 2). We speculate that these extensor muscles were not active in many trials because the force of gravity provided a sufficient extensor torque to decelerate the limb at the end of the flexor movement. We also found that the BI and BR (elbow flexors) were the only muscles active for the majority (75%) of the cerebellar trials with no errors in the shoulder fixed condition. In contrast, more muscles (in addition to the elbow flexors) were active for five of six of the overshoot trials in the shoulder fixed condition.

DISCUSSION

Many studies have investigated potential mechanisms for cerebellar limb ataxia (Bastian et al. 1996; Brown et al. 1990; Flament and Hore 1986; Goodkin et al. 1993; Hallett et al.

1975; Hallett and Massaquoi 1993; Hore et al. 1991; Manto et al. 1994; Massaquoi and Hallett 1996; Topka et al. 1998a,b). One strategy has been to study a specific aspect of ataxia (e.g., hypermetria, dysdiadochokinesia) using a single jointed movement paradigm (Brown et al. 1990; Flament and Hore 1986; Hallett et al. 1975, 1991; Hore et al. 1991; Manto et al. 1994). Studies of single jointed movements have revealed several parameters that the cerebellum may control, including timing and/or amplitude scaling in either the agonist, antagonist, or both muscle groups. However, no single deficit uncovered can be generalized across studies to explain the different features of ataxia. Instead, cerebellar damage appears to disrupt different aspects of a movement depending on task conditions. The one common feature across these studies is that the movement deficits were always attributed to an imbalance of agonist-antagonist activity.

An important feature of cerebellar control may be in predictively scaling the relative activities of different muscles in relation to the mechanical demands (e.g., inertia, interaction torques) during the movement. If this is the case, then cerebellar ataxia should be more pronounced during movements that require coordination of many muscles (Thach et al. 1992a) and/or have complex mechanical demands (Bastian et al. 1996; Massaquoi and Hallett 1996; Topka et al. 1998b). Evidence from both humans and monkeys indicate that cerebellar damage impairs multi-jointed movements to a greater extent than would be expected based on single jointed deficits (Goodkin et al. 1993; Thach et al. 1992a,b). Goodkin and colleagues reported a cerebellar subject who could make relatively normal single-jointed wrist movements, but had impaired multi-jointed reaching movements (Goodkin et al. 1993). Similarly, monkeys with inactivations of the cerebellar dentate nucleus have been shown to have mild impairments of single-jointed wrist movements, but gross impairments of reaching movements (Thach et al. 1992b).

In the present study, we found that cerebellar subjects can make nearly normal arm movements when they were constrained to move at only one joint in the shoulder fixed condition. This error reduction could not be explained by an appreciable slowing of the movement. Reduction of the most prevalent error type (overshoot) was due to a decrease in unwanted shoulder flexion movements caused by over-compensation for interaction torques. Reduction of undershoot errors was due to an increase in elbow flexion movements. We also found that, unlike controls, cerebellar subjects substantially reduced shoulder muscle activity when moving in the shoulder fixed condition. We speculate that the improvement in the cerebellar groups performance in the shoulder fixed condition could be due to the absence of interaction torques, reduction of the number of muscles to be controlled, or a combination of these two factors.

Interaction torques: under- versus over-compensation

Interaction torques are a mechanical complexity inherent in multi-jointed movements. It has been suggested that the role of the cerebellum may be in generating muscle torques that predict and compensate for interaction torques (Bastian et al. 1996; Schweighofer et al. 1998a,b). This is supported by the finding that subjects with cerebellar damage have difficulty adjusting their motor output to offset interaction torques during

fast, reaching movements (Bastian et al. 1996; Topka et al. 1998b). In these studies, cerebellar subjects produced muscle torques that under-compensated for the interaction torques that occurred late in the movement. Because of this under-compensation, interaction torques contributed more to the net torque and determined the course of the movement. This resulted in target overshoot and abnormalities in elbow-shoulder coordination (Bastian et al. 1996).

When compared with controls, cerebellar subjects also are impaired when volitionally stabilizing the shoulder joint while moving the elbow joint (Boose et al. 1999). In a recent study, subjects were asked to flex only the elbow to a remembered final position (no visual target) with both elbow and shoulder joints unconstrained. Both controls and cerebellar subjects were unable to volitionally maintain a static shoulder position. This was especially true of cerebellar subjects who tended to move the shoulder more than controls. Unwanted shoulder movement increased with higher velocities of elbow movement. When compared with controls, cerebellar subjects also had a lower correlation between the unwanted shoulder movement and the interaction torque occurring at the shoulder (Boose et al. 1999). The authors speculated that the cerebellar subjects could not generate sufficient phasic shoulder muscle torque, based on the velocity dependence of the unwanted shoulder movement. However, the magnitude and rate of muscle torque production was not assessed. If the cerebellar subjects' only problem was insufficient muscle torques in countering interaction torques (i.e., under-compensation), one would have expected a higher correlation between the unwanted shoulder movement and the interaction torque. This did not appear to be the case.

We have found that cerebellar subjects can over- or under-compensate for interaction torques, depending on the demands of the task. During our previous study of fast reaching, the deficit that we observed was that of under-compensation for interaction torques (Bastian et al. 1996). In the present study, we have observed that, compared with controls, cerebellar subjects often over-compensate for interaction torques at the shoulder, producing excessive shoulder flexion. This was due to the fact that, compared with controls, cerebellar subjects produced larger shoulder muscle torques at higher rates than those produced at the elbow. This torque "mismatch" became most apparent at higher velocities of movement and appeared to be the primary cause of the deficit. This mismatch also occurred early in the movement (within the 1st 100 ms). Finally, we found timing differences for peak muscle and interaction torques at the shoulder joint between the control and cerebellar groups. These results indicate that the primary cerebellar deficit may not simply be deficiency of phasic muscle torques. Instead, we suggest that the deficit is an inability to produce the appropriate muscle torque for the expected interaction torques. It remains to be determined why over-compensation for interaction torques occurs during some movements, and under-compensation during others.

Control of multiple muscles

Cerebellar subjects produced irregular EMG patterns, often with prolonged muscle activity that was great or lesser in magnitude compared with controls. One of the striking findings in the present study was the difference in EMG patterns pro-

duced by controls and cerebellar subjects moving in the shoulder fixed condition. For controls, mechanically stabilizing the shoulder did not dramatically change the pattern of muscle activity. Control subjects typically activated the shoulder flexor (AD) concurrently with the elbow flexors (BI, BR) in both the shoulder free and shoulder fixed conditions. In contrast, for the cerebellar subjects, mechanically stabilizing the shoulder greatly altered the pattern of muscle activity used by the cerebellar subjects. In the shoulder fixed condition, cerebellar subjects reduced or ceased activity in the shoulder flexor (AD), but continued to strongly activate the elbow flexors (BI, BR). The reduction in AD activity was most often to resting baseline levels, and almost always present on the first test trial. We do not know whether it was diminished on a trial-by-trial basis during the three to five unrecorded test trials in the shoulder fixed condition.

We suspect that the reduced shoulder muscle activity observed in the shoulder fixed condition could be explained by two possible mechanisms. One possibility is that the reduced activity of shoulder muscles represents a strategy used by cerebellar subjects to further simplify control of the reaching movement. During reaching, multiple joints and multiple muscles must be controlled to hit the target. We imposed one simplification on the reaching movement by constraining the number of joints to be controlled in the shoulder fixed condition. However, the pattern of muscle activity was still free to vary, even though activity at the shoulder would have no effect on the movement. We speculate that the cerebellar subjects may have reduced activity of all muscles except the elbow flexors to further simplify control of the reach in the shoulder fixed condition. However, they would still have to combine the appropriate levels of activity in the two elbow flexors that were active (BI and BR). Errors in this combination could lead to endpoint errors in the shoulder fixed condition.

A second possibility is that cerebellar subjects had a loss of associated muscle activity due to the cerebellar disease (Holmes 1939). If this were the case, the shoulder free condition would require cerebellar subjects to attempt a volitional muscle activation at the shoulder to compensate for the interaction torques. This volitional mechanism may not be appropriate to account for the interaction torques at the shoulder, and the cerebellar subjects' attempted shoulder stabilization would fail. In the shoulder fixed condition, the loss of associated shoulder muscle activity would not interfere with the performance of the elbow movement, and the movement would be performed more normally.

Given the time course of our subject's disease, we expect that we are measuring both the movement deficits and compensatory behavior in these individuals. Because of this, it is difficult to know whether the reduction in shoulder muscle activity during the shoulder fixed condition is a result of a learned compensation strategy, or the primary deficit. Future studies of subjects with very acute lesions may help to more clearly distinguish between these two possibilities.

Cerebellar contributions to arm movement

Based on this and other studies, we suggest that the cerebellum is involved in scaling the relative activities of multiple muscles to account for various forces (including inertial and

interaction torques) that oppose or assist the intended movement (Bastian et al. 1996; Schweighofer et al. 1998a,b). This contribution would be critical to ensure that the limb segments are moved at the correct rate and extent relative to one another. This type of control would also have to be predictive in nature, given that many movements are too fast for feedback-dependent processes to operate. Several lines of evidence are consistent with these ideas. First, cerebellar subjects have difficulty scaling the relative activities of agonist and antagonist muscles to move a single segment (Flament and Hore 1986; Hallett et al. 1975; Hore et al. 1991). These single joint deficits worsen when inertia is added to the moving segment (Manto et al. 1994). Second, much more striking deficits are observed when multiple muscles are used to control a multi-jointed reaching movement. Multi-jointed movement deficits are more than one would expect given the single joint deficits, implying that the single joint deficits are not simply summing. We believe that the additional mechanical complexity of interaction torques during multi-jointed movements may account for this difference. Finally, we suggest that a more general inability to produce phasic muscle activity cannot explain these deficits. The current study demonstrates that cerebellar errors in multi-jointed movements can be due to excessive muscle torques produced at comparable rates compared with controls. Thus cerebellar patients can produce muscle torques that under- or over-compensate for interaction torques during multi-jointed movements.

We thank E. Connor and C. Lang for helpful comments and discussions. We also thank A. Dromerick, J. Mink, J. Perlmutter, and B. Racette for patient referral.

This work was supported by National Institute of Neurological Disorders and Stroke Grants NS-01199 to A. J. Bastian and NS-12777 to W. T. Thach. Address for reprint requests: A. J. Bastian, Program in Physical Therapy, Washington University School of Medicine, 4444 Forest Park Blvd., Rm. 1101, Campus Box 8502, St. Louis, MO 63108.

Received 15 September 1999; accepted in final form 15 February 2000.

REFERENCES

- BASTIAN, A. J., MARTIN, T. A., KEATING, J. K., AND THACH, W. T. Cerebellar ataxia: Abnormal control of interaction torques across multiple joints. *J. Neurophysiol.* 76: 492–509, 1996.
- BASTIAN, A. J., ZACKOWSKI, K. M., AND THACH, W. T. Cerebellar incoordination in reaching: effects of mechanical vs. voluntary joint fixation. *Soc. Neurosci. Abstr.* 23: 4, 1997.
- BOOSE, A., DICHGANS, J., AND TOPKA, H. Deficits in phasic muscle force generation explain insufficient compensation for interaction torque in cerebellar patients. *Neurosci. Lett.* 26: 53–56, 1999.
- BROWN, S. H., HEFTER, H., MERTENS, M., AND FREUND, H.-J. Disturbances in human arm movement trajectory due to mild cerebellar dysfunction. *J. Neurol. Neurosurg. Psychiatry* 53: 306–313, 1990.
- FLAMENT, D. AND HORE, J. Movement and electromyographic disorders associated with cerebellar dysmetria. *J. Neurophysiol.* 55: 1221–1233, 1986.
- GOODKIN, H. P., KEATING, J. G., MARTIN, T. A., AND THACH, W. T. Preserved simple and impaired compound movement after infarction in the territory of the superior cerebellar artery. *Can. J. Neurol. Sci.* 20, Suppl. 3: S93–S104, 1993.
- HALLETT, M., BERARDELLI, A., MATHESON, J., ROTHWELL, J., AND MARSDEN, C.D.S. Physiological analysis of simple rapid movements in patients with cerebellar deficits. *J. Neurol. Neurosurg. Psychiatry* 53: 124–133, 1991.
- HALLETT, M. AND MASSAQUOI, S. Physiologic studies of dysmetria in patients with cerebellar deficits. *Can. J. Neurol. Sci.* 20 Suppl. 3: S83–S92, 1993.
- HALLETT, M., SHAHANI, B. T., AND YOUNG, R. R. EMG analysis of patients with cerebellar deficits. *J. Neurol. Neurosurg. Psychiatry* 38: 1163–1169, 1975.

- HOLMES, G. The cerebellum of man. The Hughlings Jackson memorial lecture. *Brain* 62: 1–30, 1939.
- HORE, J., WILD, B., AND DIENER, H.-C. Cerebellar dysmetria at the elbow, wrist, and fingers. *J. Neurophysiol.* 65: 563–571, 1991.
- HOY, M., ZERNICKE, R. F., AND SMITH, J. L. Contrasting roles of inertial and muscle moments at knee and ankle during paw-shake response. *J. Neurophysiol.* 54: 1282–1284, 1985.
- MANTO, M., GODAUX, E., AND JACQUY, J. Cerebellar hypermetria is larger when the inertial load is artificially increased. *Ann. Neurol.* 35: 45–52, 1994.
- MASSAQUOI, S. AND HALLETT, M. Kinematics of initiating a two-joint arm movement in patients with cerebellar ataxia. *Can. J. Neurol. Sci.* 23: 3–14, 1996.
- SCHWEIGHOFER, N., ARBIB, M. A., AND KAWATO, M. Role of the cerebellum in reaching movements in humans. I. Distributed inverse dynamics control. *Eur. J. Neurosci.* 10: 86–94, 1998a.
- SCHWEIGHOFER, N., SPOELSTRA, J., ARBIB, M. A., AND KAWATO, M. Role of the cerebellum in reaching movements in humans. II. A neural model of the intermediate cerebellum. *Eur. J. Neurosci.* 10: 95–105, 1998b.
- THACH, W. T., GOODKIN, H. G., AND KEATING, J. G. The cerebellum and the adaptive coordination of movement. *Annu. Rev. Neurosci.* 15: 403–442, 1992a.
- THACH, W. T., KANE, S. A., MINK, J. W., AND GOODKIN, H. P. Cerebellar output: multiple maps and motor modes in movement coordination. In: *The Cerebellum Revisited*, edited by R. Llinas and C. Sotelo. New York: Springer-Verlag, 1992b, p. 283–300.
- TOPKA, H., KONCZAK, J., AND DICHGANS, J. Coordination of multi-joint arm movements in cerebellar ataxia: analysis of hand and angular kinematics. *Exp. Brain Res.* 119: 483–492, 1998a.
- TOPKA, H., KONCZAK, J., SCHNEIDER, K., BOOSE, A., AND DICHGANS, J. Multi-joint arm movements in cerebellar ataxia: abnormal control of movement dynamics. *Exp. Brain Res.* 119: 493–503, 1998b.