

Selection and Coordination of Human Locomotor Forms Following Cerebellar Damage

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Earhart, Gammon M. and Amy J. Bastian. Selection and coordination of human locomotor forms following cerebellar damage. *J Neurophysiol* 85: 759–769, 2001. We have previously shown that control subjects use two distinct temporal strategies when stepping on an inclined surface during walking: one for level and 10° surfaces and another for 20 and 30° surfaces. These two temporal strategies were characterized by systematic shifts in the timing of muscle activity and peak joint angles. We examined whether cerebellar subjects with mild to moderate gait ataxia were impaired in their ability to select these two temporal strategies, adjust peak joint angle amplitudes, and/or adjust one joint appropriately with respect to movements and constraints at another joint. Subjects walked on a level surface and on different wedges (10, 20, and 30°) presented in the context of level walking. In a single trial, a subject walked on a level surface in approach to a wedge, took a single step on the wedge, and continued walking on an elevated level surface beyond the wedge. Cerebellar subjects used two temporal strategies, one for the level and 10° surfaces and another for 20 and 30° surfaces. Cerebellar strategies were similar to those used by controls except for the timing of ankle-joint movement on the steeper wedges. Cerebellar subjects adjusted the peak amplitudes of individual joint angles normally, with the exception of peak ankle plantarflexion. However, they exhibited greater trial-to-trial variability of peak hip and knee joint angles that increased as a function of wedge inclination. The most substantial deficit noted in the cerebellar group was in the relative movement of multiple joints. Cerebellar subjects demonstrated multijoint coordination deficits in all conditions, although these deficits were most pronounced during stance on the steeper wedges. On the 30° wedge, cerebellar subjects showed abnormal relative movement of hip, knee, and ankle joints and the most substantial decomposition of movement. We speculate that to simplify multijoint control, cerebellar subjects decomposed their movement by fixing the ankle joint in a dorsiflexed position on the steepest wedges. Our results suggest that the cerebellum may not be critical in selecting the basic motor patterns for the two temporal strategies because cerebellar subjects produced appropriate timing shifts at most joints. Instead, our data suggest that the cerebellum is most critical for adjusting the relative movement of multiple joints, especially to accommodate external constraints.

INTRODUCTION

Many people with cerebellar damage have gait ataxia, which is characterized by staggering, irregular stepping, veering, and excessively high lifting of the feet above the ground (Gilman et al. 1981). Another typical feature of cerebellar gait ataxia is that the joints of the leg tend to move at abnormal rates relative

to one another, often with extreme variability (Bastian et al. 1998; Hallett and Massaquoi 1993; Hallett et al. 1991; Palliyath et al. 1998). For example, subjects with cerebellar atrophy can have delays in the relative movement of the knee and ankle throughout the gait cycle (Hallett and Massaquoi 1993). Palliyath et al. (1998) noted a similar deficit; subjects with cerebellar cortical and olivopontocerebellar atrophy show delays in the timing of peak knee flexion during the swing phase of walking. Subjects with discrete lesions of the posterior cerebellar vermis show marked coordination deficits within each leg and between the legs when walking in tandem (heel to toe), but minimal deficits in standard walking or hopping on one foot (Bastian et al. 1998). In summary, human studies generally suggest that the cerebellum is important for controlling the relative movement between joints of the legs during locomotion. It is not clear whether these deficits can be accounted for by abnormalities in the timing or scaling of individual joint movements.

Animal studies have helped elucidate the role of different cerebellar regions in the control of walking. During cat locomotion, there is rhythmic neural activity in the fastigial nucleus that modifies descending brain stem pathways that influence spinal cord locomotor generators (Antziferova et al. 1980; Arshavsky et al. 1983; Orlovskii 1972). Lesions of the fastigial nucleus impair stance and the basic gait pattern, with the animals falling to the side of the lesion (Botterell and Fulton 1938a; Sprague and Chambers 1953; Thach et al. 1992b). Lesions of intermediate or lateral cerebellar zones (including interpositus or dentate) do not markedly impair stance or the basic gait pattern but can produce high stepping (Botterell and Fulton 1938b; Thach et al. 1992b) or knee hyperflexion (Udo et al. 1979b, 1980; Yu and Eidelberg 1983). Neural activity in intermediate and lateral cerebellar zones has been shown to relate to locomotion in novel contexts, such as walking on a treadmill (Armstrong and Edgley 1988) or walking across motor-driven ladder rungs (Anderson and Armstrong 1987; Armstrong and Marple-Horvat 1996; Marple-Horvat and Criado 1999). In addition, the ability to adapt to perturbations during treadmill locomotion following nitric oxide deprivation in lobules V and VI of the vermis is impaired (Yanagihara and Kondo 1996). In summary, all cerebellar regions may participate in the control of quadrupedal locomotion, although inter-

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TABLE 1. *Subject information*

Subject	Age, yr	Gender	Lesion	Side Tested	Length of Illness	Gait Ataxia
<i>CBL-1</i>	20	F	Posterior vermal split	Right	6 years	Mild
<i>CBL-2</i>	23	F	Posterior vermal split	Left	2 years	Mild
<i>CBL-3</i>	35	F	Spinocerebellar atrophy type 7	Right	6 years	Moderate
<i>CBL-4</i>	39	M	Pancerebellar cortical atrophy	Right	6 years	Moderate
<i>CBL-5</i>	43	F	Pancerebellar cortical atrophy	Right	12 years	Moderate
<i>CBL-6</i>	67	M	Olivopontocerebellar atrophy	Right	12 years	Moderate
<i>CBL-7</i>	69	F	Left superior cerebellar artery infarct	Left	5 years	Moderate
<i>CBL-8</i>	70	F	Right superior cerebellar artery infarct	Right	2 weeks	Mild

mediate and lateral regions may be more involved in adjusting the walking pattern to novel contexts.

Only one study has examined the ability of people with cerebellar damage to adjust to perturbations during treadmill locomotion (Rand et al. 1998). This study showed that subjects with damage to the cerebellar vermis and/or hemispheres (but minimal damage to the cerebellar nuclei) could adjust to transient changes in treadmill speed but did so in an inconsistent manner compared with control subjects. Rand et al. (1998) suggested that the cerebellum is important for specification of optimal strategies to be used during compensatory or learned movements.

The purpose of the present study was to examine the role of the cerebellum in the coordination of human locomotion over wedges. Subjects walked on a level surface in approach to a wedge, took a single step on the wedge, and continued walking on an elevated level surface beyond the wedge. This task is similar to walking from the street to the sidewalk by taking a single step on a handicapped access ramp. Subjects were tested during level walking and on inclines of 10, 20, and 30°. In a previous study of this task, we demonstrated that control subjects use two distinct temporal strategies to traverse inclines; one strategy is used for level and 10° conditions, another for 20 and 30° conditions (Earhart and Bastian 2000). In the present study, we were interested in determining whether subjects with cerebellar damage show deficits in selection of these temporal strategies, deficits in the amplitude scaling of individual joint angles, and/or a specific inability to adjust one joint appropriately with respect to movements and constraints at another joint. We provide evidence that the subjects tested were able to select two temporal strategies and adjust individual joint angle amplitudes but demonstrated abnormalities in the relative movement of multiple joints. We also discuss whether people with damage of different cerebellar regions show comparable deficits on this task. This is the first study to address the role of the human cerebellum in adjusting locomotion to accommodate different walking surface inclinations.

METHODS

Subjects

Eight subjects with cerebellar damage and eight control subjects participated in this study. Cerebellar damage was confirmed by imaging (computed tomography or magnetic resonance imaging). In addition, results of genetic testing were sometimes available. Neurological examination of cerebellar subjects revealed that all had mild instability in quiet stance and mild to moderate gait ataxia. Subjects who normally walked independently without an assistive device were considered to have mild ataxia, while those who normally used an assistive device were considered to have moderate ataxia. Two cere-

bellar subjects had surgical splits of the posterior vermis secondary to tumor removal, four had cerebellar atrophy, and two had superior cerebellar artery infarcts (Table 1). For subjects with unilateral infarcts, data were recorded from the side of the body ipsilateral to the lesion. For all other subjects, data were recorded from the most affected side of the body. None of the subjects had sensory loss in the legs or feet. *Subject CBL-1* underwent surgical resection of a medulloblastoma, resulting in damage to lobules VI–X; *subject CBL-2* underwent surgical resection of an astrocytoma, resulting in extensive vermal damage. This subject (*CBL-2*) exhibited nystagmus and had facial nerve involvement but was not hyperreflexive and did not show a Babinski's sign. One subject with pancerebellar cortical atrophy (*CBL-5*) had mild hyperreflexia at the ankle on the side that was not recorded. All cerebellar subjects were able to walk without an assistive device, but with human assistance if necessary, during participation in the study. Cerebellar subjects ranged in age from 20 to 70 (mean \pm SD = 45.75 \pm 20.45) years.

Neurologically unimpaired control subjects were matched with cerebellar subjects on the basis of age and gender. Control subjects ranged in age from 22 to 71 (mean: 45.25 \pm 20.97) years. All subjects gave informed consent prior to participation in the study.

Paradigm

The locomotor task used has been described previously (Earhart and Bastian 2000). Subjects walked at a comfortable pace on a level surface approaching the wedge, took a single step on the wedge, and continued walking on an elevated level surface beyond the wedge (see Fig. 1 of Earhart and Bastian 2000). The wedge system had a height-adjustable platform providing an elevated level surface beyond the wedge that was even with the height of the wedge peak for all inclinations. All wedges were 0.98 m (3 ft) wide and 0.66 m (2 ft) long. Subjects performed three to eight trials on each of the conditions: level walking, 10, 20, and 30° wedges. One cerebellar subject (*CBL-7*) was unable to traverse the 30° wedge and walked only in the level, 10 and 20° conditions. All trials on a given wedge were performed as a block, and blocks were presented in random order to all subjects. Cerebellar subjects wore a gait belt and were accompanied by a physical therapist at all times. Subjects were allowed to rest as needed throughout testing.

Data collection

Kinematic and electromyographic (EMG) data were recorded from one side of the body. Although patterns on the opposite side of the body were undoubtedly changing during this task, equipment limitations prevented us from recording contralateral patterns. We collected kinematic data (100 Hz) using the OPTOTRAK three-dimensional motion-analysis system (Northern Digital, Waterloo, Ontario). Infrared-emitting diodes were placed over the following anatomical landmarks: shoulder (acromion process), pelvis (iliac crest), hip (greater trochanter), knee (lateral femoral epicondyle), ankle (lateral malleolus), and foot (5th metatarsal head). Joint angles were calculated based on the positions of these

TABLE 2. Basic gait parameters

	Stride Length, m	Stride Duration, s	Gait Velocity, m/s	Foot Height, mm (peak height – height at HS)
Level				
Cerebellar	0.99 ± 0.11*	1.32 ± 0.06*	0.75 ± 0.12*	15.29 ± 2.99
Control	1.22 ± 0.04	1.17 ± 0.03	1.04 ± 0.04	17.70 ± 0.82
10°				
Cerebellar	1.01 ± 0.09*	1.66 ± 0.11*	0.63 ± 0.09*	16.11 ± 2.19
Control	1.30 ± 0.04	1.29 ± 0.03	1.01 ± 0.04	10.91 ± 1.19
20°				
Cerebellar	0.97 ± 0.07*	1.76 ± 0.13*	0.57 ± 0.07*	7.39 ± 2.13
Control	1.26 ± 0.06	1.29 ± 0.04	0.99 ± 0.06	4.13 ± 0.64
30°				
Cerebellar	1.01 ± 0.04*	2.02 ± 0.25*	0.54 ± 0.08*	5.64 ± 2.91
Control	1.26 ± 0.06	1.29 ± 0.04	0.99 ± 0.04	1.61 ± 0.48

Values are means ± SE for cerebellar and control groups. HS, heel strike. *, significant group effect ($P < 0.01$).

markers (see Fig. 1 of Earhart and Bastian 2000). Hip angle (the angle between the thigh and the pelvis) increased in the positive direction with hip flexion and increased in the negative direction with hip extension. A hip angle of 0° indicated that the pelvis and thigh were colinear. Knee angle (the angle between the thigh and the shank) increased with knee flexion. A knee angle of 0° represented full extension. Ankle angle (the angle between the shank and the foot) increased in the positive direction with dorsiflexion and increased in the negative direction with plantarflexion. A neutral ankle angle (0°) was defined as the angle at which the foot was flat on the floor and the shank aligned vertically. This definition of ankle neutral corrected for the vertical offset of the ankle marker relative to the foot marker.

EMG data (collected at 1 kHz) were obtained using silver-silver chloride surface electrodes (Therapeutics Unlimited, Iowa City, IA). EMG activity was recorded from the following muscles: rectus femoris (RF), gluteus maximus (GM), vastus lateralis (VL), lateral hamstring (LH), anterior tibialis (AT), and gastrocnemius (GA).

Analysis

Automatic synchronization of kinematic and electromyographic data was done with the OPTOTRAK system. Heel strike (HS) and toe off (TO) times were selected manually from animated stick figure representations of each trial. The accuracy of HS and TO selections was assessed by collecting several walking trials during which HS and TO were registered on a force plate. HS and TO times selected manually were always within 10 ms (1 frame) of the times indicated by the force plate data. All subjects struck the walking surfaces with the heel first or with a flat foot rather than with toes first. We noted no differences in the patterns used for trials with heel-first versus foot-flat contact. We used the term HS to denote the time of contact with the incline whether that contact was made with the heel or with a flat foot.

For each subject, three strides from each condition were analyzed. A stride was defined from TO prior to the wedge through TO from the wedge, thus including swing approaching and stance on the wedge. For each stride, we calculated stride duration, stride length, and gait velocity.

Kinematic data were represented graphically in joint angle time series plots and joint angle-angle plots. We measured: 1) joint angles at HS, 2) peak joint angles at the hip, knee, and ankle, 3) joint angle variance across trials for swing and stance phases, 4) timing of peak joint angles within the stride cycle, and 5) the peak foot height above the contact surface. Joint angles at HS and peak joint angles were used to determine if the cerebellar group showed similar scaling of joint-movement amplitudes relative to the control group. The timing of peak joint angles was used to determine whether cerebellar subjects used temporal strategies similar to those used by control subjects. Peak foot height was measured to determine if cerebellar subjects

showed hypermetric lifting of the lower extremity above the walking surface.

Interjoint coordination was assessed using joint angle-angle plots, a decomposition index, and the overall configuration of the limb at different joint angle maxima. We focused particularly on ankle versus knee angles because our previous work indicated that the ankle-knee relationship showed the greatest and most consistent pattern change between the two forms (Earhart and Bastian 2000). Decomposition of movement refers to movement patterns where one joint is held fixed while another joint is moving (Bastian et al. 1996). Decomposition index values were the percentage of stance time during which movement was decomposed. A joint was considered to pause when its angular velocity dropped below 5°/s. Decomposition index values were calculated for all pairs of joint-joint combinations: hip versus ankle, knee versus hip, and ankle versus knee.

Interjoint coordination was also assessed by comparing the relative values of the hip, knee, and ankle angles at time points when one of the joints was maximally flexed or extended. During swing, limb configuration was assessed at the times of peak hip flexion, peak knee flexion, peak knee extension, and peak plantarflexion. During stance, limb configuration was assessed at the times of peak knee flexion and peak ankle dorsiflexion.

EMGs were rectified, low-pass filtered at 70 Hz (DataPac, Run Technologies, Laguna Hills, CA) and aligned on HS. EMG onsets were selected as the time when muscle activity was twice that of baseline for at least 50 ms. EMG offsets were selected as the time when muscle activity fell below twice that of baseline for at least 50 ms. Baseline was defined as the activity level during quiet stance. EMG onset and offset times were expressed as a percentage of the stride period with HS defined as zero. Events occurring during the swing phase had negative values, those occurring at HS had a value of zero, and those occurring during stance had positive values.

We used a repeated-measures analysis of covariance (ANCOVA) to compare group differences across wedge conditions for each variable. Gait velocity was used as a covariate for all analyses because the cerebellar and control groups walked at different speeds. Statistical significance was set at $P < 0.01$ and Duncan's multiple range post hoc comparisons were performed for all significant F values.

RESULTS

Basic gait parameters

Cerebellar subjects walked significantly more slowly than controls, taking shorter strides with longer stride durations (Table 2). As wedge inclination increased, stride duration increased substantially in the cerebellar group but only slightly in the control group. In addition, gait velocity decreased sub-

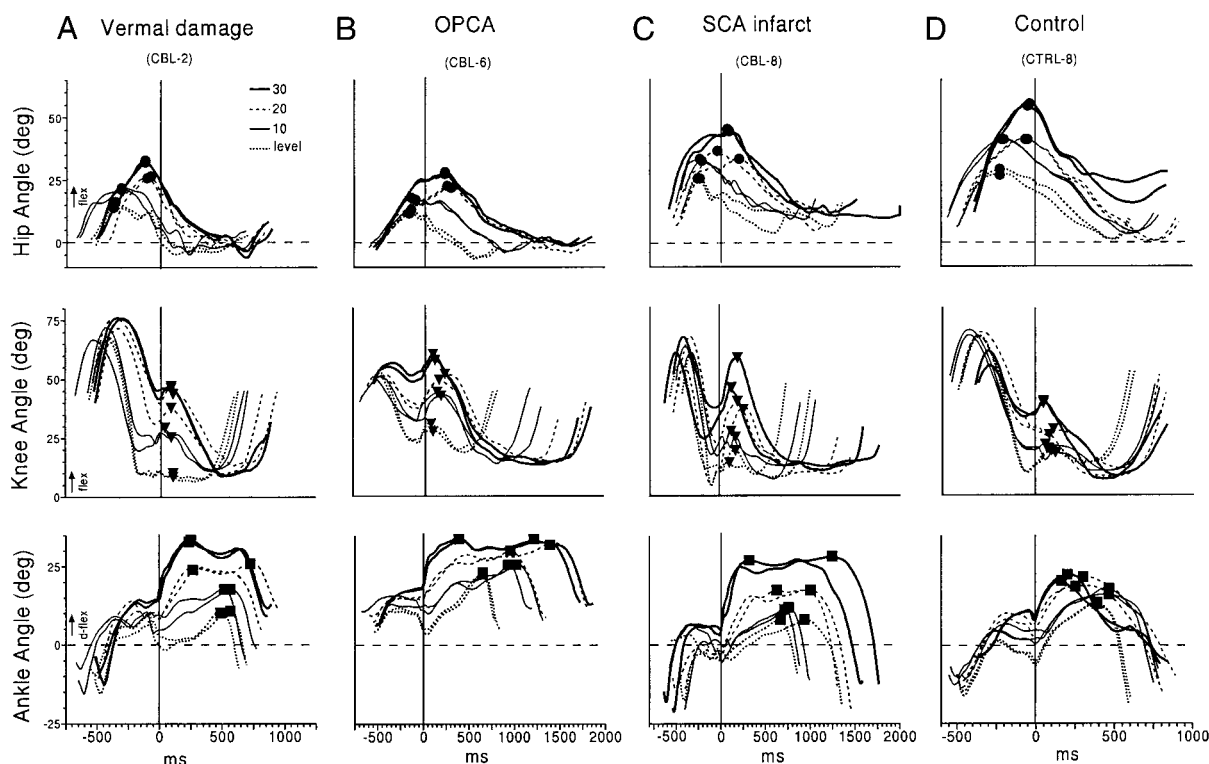


FIG. 1. Plots of joint angles vs. time for a control subject (*D*) and 3 subjects (*A–C*) with different types of cerebellar damage (OPCA, olivopontocerebellar atrophy; SCA, superior cerebellar artery). Hip (*top*), knee (*middle*), and ankle (*bottom*) angles for level and 10, 20, and 30° conditions are shown. Two trials for each wedge condition are drawn from toe off prior to the wedge through toe off from the wedge, thus depicting swing approaching and stance on the wedge. Heel strike is denoted by vertical lines drawn through 0. Markers denote peak hip flexion (●), peak stance knee flexion (▼), and peak ankle dorsiflexion (■). Note that the time scales for the cerebellar subjects vary and are longer than that for the control subject.

stantially in the cerebellar group and slightly in the control group. Stride duration and gait velocity were significantly different across wedge conditions, and there was a significant group by wedge interaction for stride duration.

Joint angle amplitudes

Cerebellar subjects adjusted most joint angle amplitudes appropriately with wedge inclination, although they demonstrated greater variability than controls. Cerebellar subjects increased joint angles at HS as wedge inclination increased but demonstrated slightly greater knee flexion than controls across all conditions. There were no significant differences between groups for individual joint angles at HS. For both groups, all joint angles were significantly different across wedge conditions (all $P < 0.01$).

Cerebellar subjects also adjusted most peak joint angles similarly to controls (Fig. 1). Peak stance knee flexion (Fig. 1, ▼) was greater in the cerebellar than in the control group, although this difference was not quite significant ($P = 0.053$). Average peak ankle plantarflexion was significantly reduced in the cerebellar group compared with controls for all conditions. Peak plantarflexion varied among cerebellar subjects. Three of eight cerebellar subjects did not plantarflex but instead remained in a dorsiflexed position throughout the gait cycle (Fig. 1B). These subjects represented each of the three types of cerebellar damage: vermal split, atrophy, and SCA infarct. The other five cerebellar subjects demonstrated varying amounts of

peak plantarflexion, and some were comparable to controls on this variable (Fig. 1, A and C).

Although cerebellar subjects adjusted most peak joint angle amplitudes appropriately, their joint angle profiles were more variable than those of control subjects. The cerebellar group showed greater variance of hip and knee movement as a function of wedge inclination ($P < 0.01$). This difference was greatest for stance on the steeper wedges. Ankle angle variances for the stance phase were similar between cerebellar and control groups across all conditions as were variances of all the joints for the swing phase.

As another means of assessing joint-angle amplitude, we determined the maximum foot height above the contact surface. This height is determined by the combination of hip, knee, and ankle angles used during swing in approach to the wedge. Maximum foot height tended to decrease with increasing wedge inclination in both groups (Table 2). Cerebellar subjects (regardless of lesion type) showed slightly higher foot heights in approach to the wedges than control subjects, but these differences were of only a few millimeters in magnitude and were not statistically significant.

Timing of peak joint angles

We previously showed that control subjects use two distinct temporal patterns to traverse the wedges (Earhart and Bastian 2000). Specifically, when controls walked on the steeper wedges (20°/30°), peak hip flexion was delayed and peak ankle dorsiflexion was time advanced. In this study, cerebellar sub-

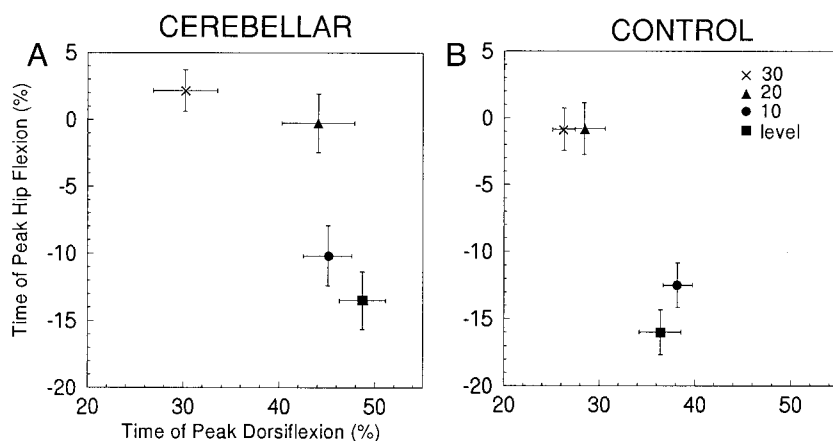


FIG. 2. Peak hip flexion time vs. peak ankle dorsiflexion time for the cerebellar (A) and control (B) groups. Values are expressed as a percentage of the gait cycle; heel strike (HS) is at *time 0*, swing phase has negative values from approximately -40 to 0% , and stance extends from 0% to approximately 60% . Times given are means \pm SE and are expressed as a percentage of the gait cycle. Note the much greater variability of peak dorsiflexion timing in the cerebellar group, particularly on the 20° and 30° conditions.

jects also showed delayed hip flexion on the steeper wedges but did not show time-advanced ankle dorsiflexion. Peak hip flexion occurred in mid-swing for the level and 10° conditions but was significantly delayed toward HS for the 20° and 30° conditions in both groups (Fig. 2). In contrast, peak ankle dorsiflexion did not shift to an earlier time for the cerebellar group as compared with the control group (Fig. 2). The cerebellar group showed highly variable timing of peak ankle dorsiflexion for the $20^\circ/30^\circ$ conditions. This variability reflected the tendency of cerebellar subjects to fix the ankle in dorsiflexion during a large portion of stance in the $20^\circ/30^\circ$ conditions (Fig. 1, ■). Note the plateau of ankle dorsiflexion in the $20^\circ/30^\circ$ conditions for the cerebellar subject in Fig. 1C. The actual time of peak dorsiflexion often varied within this plateau.

Interjoint coordination

We initially assessed interjoint coordination by constructing joint-joint angle plots of the stance phase. We focused our analysis on plots of ankle versus knee angle because these plots have shown the greatest and most consistent pattern change between the forms in control subjects (Earhart and Bastian 2000). In the current study, both control and cerebellar groups showed general differences in the ankle versus knee plot patterns for level/ 10° surfaces versus $20^\circ/30^\circ$ surfaces. For both groups, the ankle versus knee plot on the level and 10° surfaces showed several discrete changes in direction due to rapid reversals of angular movement at one or both joints (Fig. 3, 10° condition). Ankle versus knee patterns on the 20° and 30° surfaces did not show such distinct directional changes (Fig. 3, 30° condition). On these steeper inclinations, control subjects produced a C-shaped curvilinear pattern due to continuous changes in angular movement at both joints. Cerebellar subjects produced a pattern that was roughly C shaped but was composed of more linear horizontal and vertical portions.

Horizontal and vertical linear regions of the ankle versus knee plots represent times in which one joint was moving while the other was held fixed, and were seen in plots from subjects with all types of cerebellar damage. We quantified this using a decomposition index, which reflects the percentage of stance during which one joint was fixed while the other continued to move. Decomposition index measures showed that movements of cerebellar subjects were decomposed a greater percentage of stance across all conditions but particularly in the $20^\circ/30^\circ$

conditions. Cerebellar subjects tended to hold the ankle fixed relative to either the hip or the knee but moved the hip and knee together. Figure 4 shows average decomposition index values for hip versus ankle, knee versus hip, and ankle versus knee joints. ANCOVA, with walking velocity as a covariate, showed significant differences between groups for hip versus ankle (Fig. 4A) and ankle versus knee (Fig. 4C) but no statistical difference for knee versus hip (Fig. 4B). Decomposition of hip versus ankle and ankle versus knee showed similar trends, increasing significantly in the cerebellar group for the $20^\circ/30^\circ$ conditions ($P < 0.01$).

Abnormalities in interjoint relationships were also apparent when we assessed the configuration of the entire lower extremity at specific time points in the gait cycle. These "snapshots" of limb configuration provided an indication of the relationships between the average angular positions of the three joints as well as the variability of these relationships. Snapshots were taken during swing at the times of peak ankle plantarflexion, peak hip flexion, peak knee flexion, and peak knee extension and during stance, at the times of peak knee flexion and peak ankle dorsiflexion. Each set of stick figures at the top of Fig. 5 shows snapshots from a trial on the 30° condition. Stick figure sets on the *left* and *middle* depict two trials from the same cerebellar subject. The stick figure set on the *right* depicts a trial from a matched control subject. During swing, relative configurations of the joints were not different between groups for any wedge condition.

During stance, relative configurations of the three joints were not different between groups for the level and 10° and 20° conditions. However, there were group differences in the relative configurations of the joints during stance in the 30° condition. First, at the time of peak stance knee flexion, the cerebellar group showed much greater joint angle variability for the 30° condition than did control subjects. Second, at the time of peak ankle dorsiflexion, the cerebellar group limb configuration was different from the control group. In Fig. 5, A–C, the bold stick figure within each set shows the snapshot taken at the time of peak ankle dorsiflexion. Note that the configurations in A and B figures are different from one another, although they are from the same cerebellar subject traversing the same wedge. At this time point, the control group average joint angle values (Fig. 5E) were highest for the ankle and lowest for the hip, with the knee angle value falling between those of the ankle and hip.

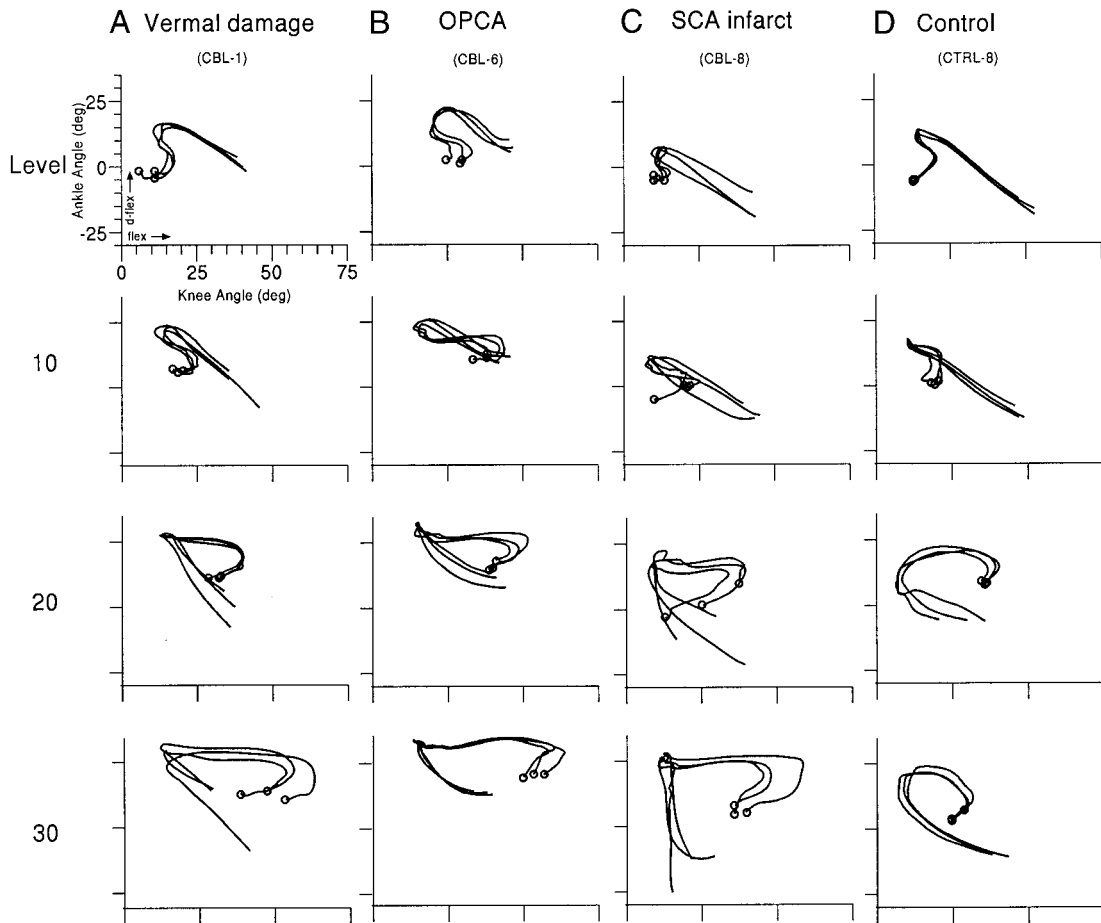


FIG. 3. Plots of ankle angle vs. knee angle during stance on the wedge for 3 subjects with different types of cerebellar damage (A–C) and a control subject (D). HS is denoted by \circ and represents the starting point of each plot. Note that both subjects use 1 basic pattern for level/10° and a different pattern for 20°/30°. Also note the 30° plots for the cerebellar subjects have linear, horizontal and vertical portions, indicating that 1 joint was held fixed as the other joint moved during different portions of the stance phase.

The cerebellar group average joint angle values were highest for the knee and lowest for the hip, with the ankle angle value falling between those of the knee and hip (Fig. 5D). The cerebellar group also showed much greater joint angle variability than the control group.

EMG timing

Onsets and offsets of EMG activity were measured to determine if cerebellar subjects used temporal patterns of muscle activity similar to those of controls. Muscle offsets were some-

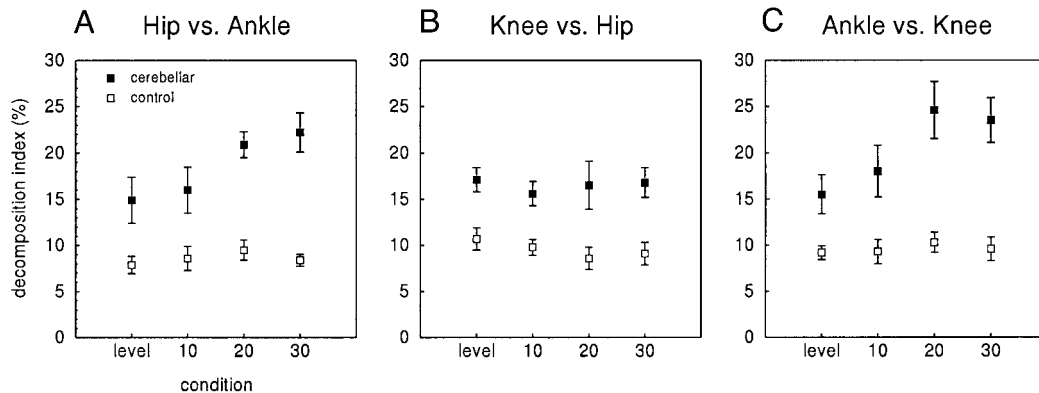


FIG. 4. Plots of average decomposition index values \pm SE for hip vs. ankle (A), knee vs. hip (B), and ankle vs. knee (C). Note the similarity of the hip vs. ankle and ankle vs. knee data, both of which show increased decomposition of movement in the cerebellar group, particularly in the 20°/30° conditions. Differences between groups were significant for both hip vs. ankle and ankle vs. knee decomposition as were group by wedge interactions ($P < 0.01$).

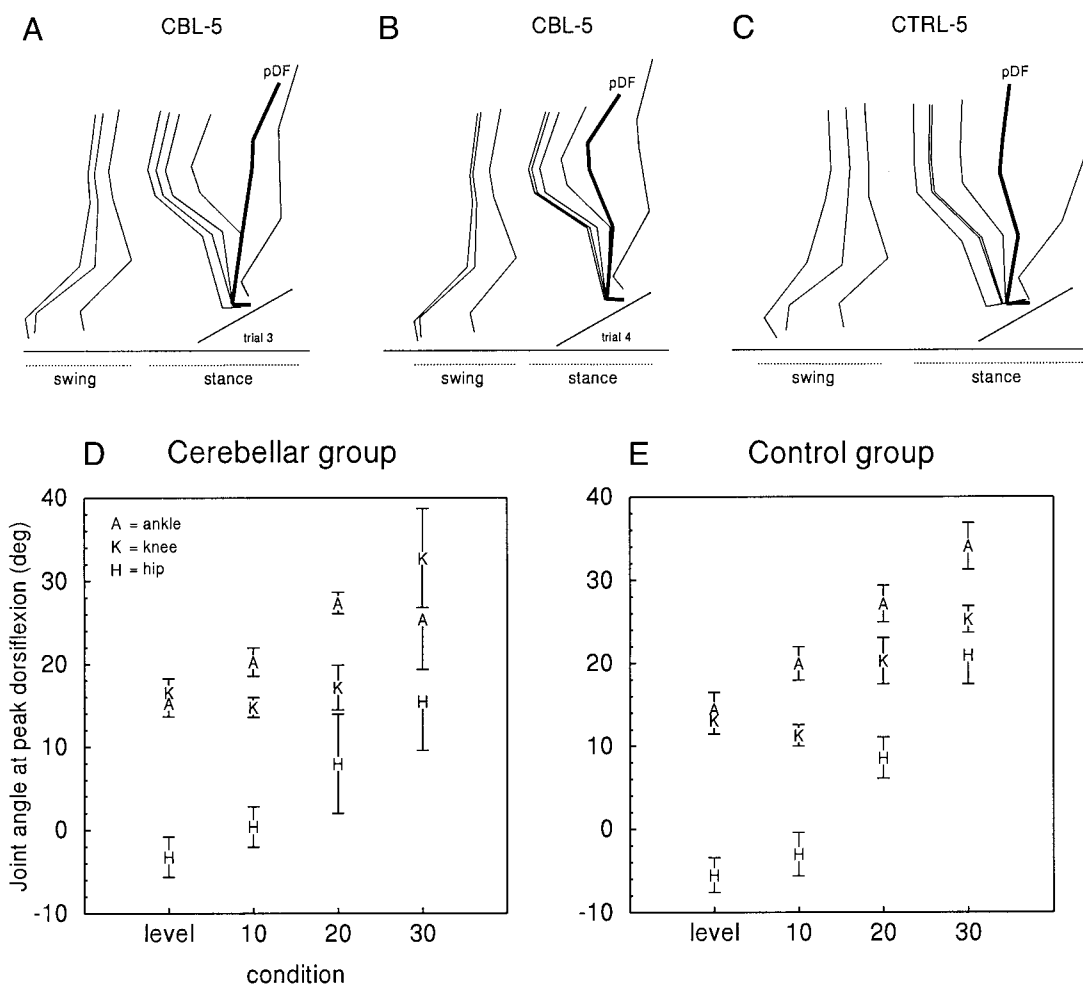


FIG. 5. The sets of stick figures in *A* and *B* show 2 30° trials from the same cerebellar subject; the set of stick figures in *C* shows a 30° trial from a matched control subject. Stick figures show marker locations rather than joint angles. The ankle joint appears to be plantarflexed in many of the stick figures because of the vertical offset between the ankle and foot markers, whereas joint angles indicate that the ankle was dorsiflexed throughout much of stance. This is because the definition of neutral ankle angle corrected for this offset (see METHODS). Each set of stick figures depicts snapshots taken at the times of: toe off, peak ankle plantarflexion, peak swing knee flexion, peak swing knee extension, HS on the wedge, peak stance knee flexion, peak ankle dorsiflexion (bold, labeled pDF), and toe off from the wedge. Graphs at the bottom show average joint angles \pm SE at the time of peak ankle dorsiflexion for cerebellar (*D*) and control (*E*) groups. Note the differences between cerebellar and control groups at 30° , where the cerebellar group shows a different relative configuration of the 3 joints as well as increased variability.

what delayed in the cerebellar group. Figure 6 shows EMG records from a cerebellar subject and a matched control subject for one trial of level walking (Fig. 6A) and one trial in the 30° condition (B). Note that the time scales for level walking and the control subject's 30° trial are the same, but the time scale for the cerebellar subject's 30° trial is much longer. In both subjects, RF, GM, and VL onsets occurred before HS during level walking, but at the time of HS in the 30° condition. In the control subject, AT offset occurred just after HS in level walking but well after HS in the 30° trial. In the cerebellar subject, AT activity was prolonged relative to the control subject with AT offset occurring well after HS in level walking and the 30° trial.

Figure 7 shows group average times \pm SE for onsets of GM and VL and for offset of AT across conditions. Time is expressed as a percentage of the gait cycle with 0% corresponding to HS. These particular muscles were selected for illustration based on our former study, which showed grouping of temporal features in the level/ 10° and $20^\circ/30^\circ$ degree strategies (Earhart and Bastian 2000). In the current study, both the cerebellar and the control

group showed two distinct temporal patterns of muscle activity: one pattern for level/ 10° and a second pattern for $20^\circ/30^\circ$. For both groups, onset times for GM (Fig. 7A) and VL (B) and offset times for AT (C) fell into two distinct groupings: the patterns for level and 10° were similar, as were those for 20 and 30° , but differences between level/ 10° and $20^\circ/30^\circ$ were significant.

Although not statistically significant, there was a noteworthy trend of prolonged AT activity (Fig. 7C) among cerebellar subjects. AT offsets in cerebellar subjects were well after HS for level/ 10° , whereas AT offsets in control subjects were at or near the time of HS for level/ 10° . Both cerebellar and control groups showed later AT offsets in $20^\circ/30^\circ$ than in level/ 10° conditions with the cerebellar group offsets occurring later than those of the control group.

DISCUSSION

During this task, we found that cerebellar subjects walked slower than controls and showed increased joint-angle variabil-

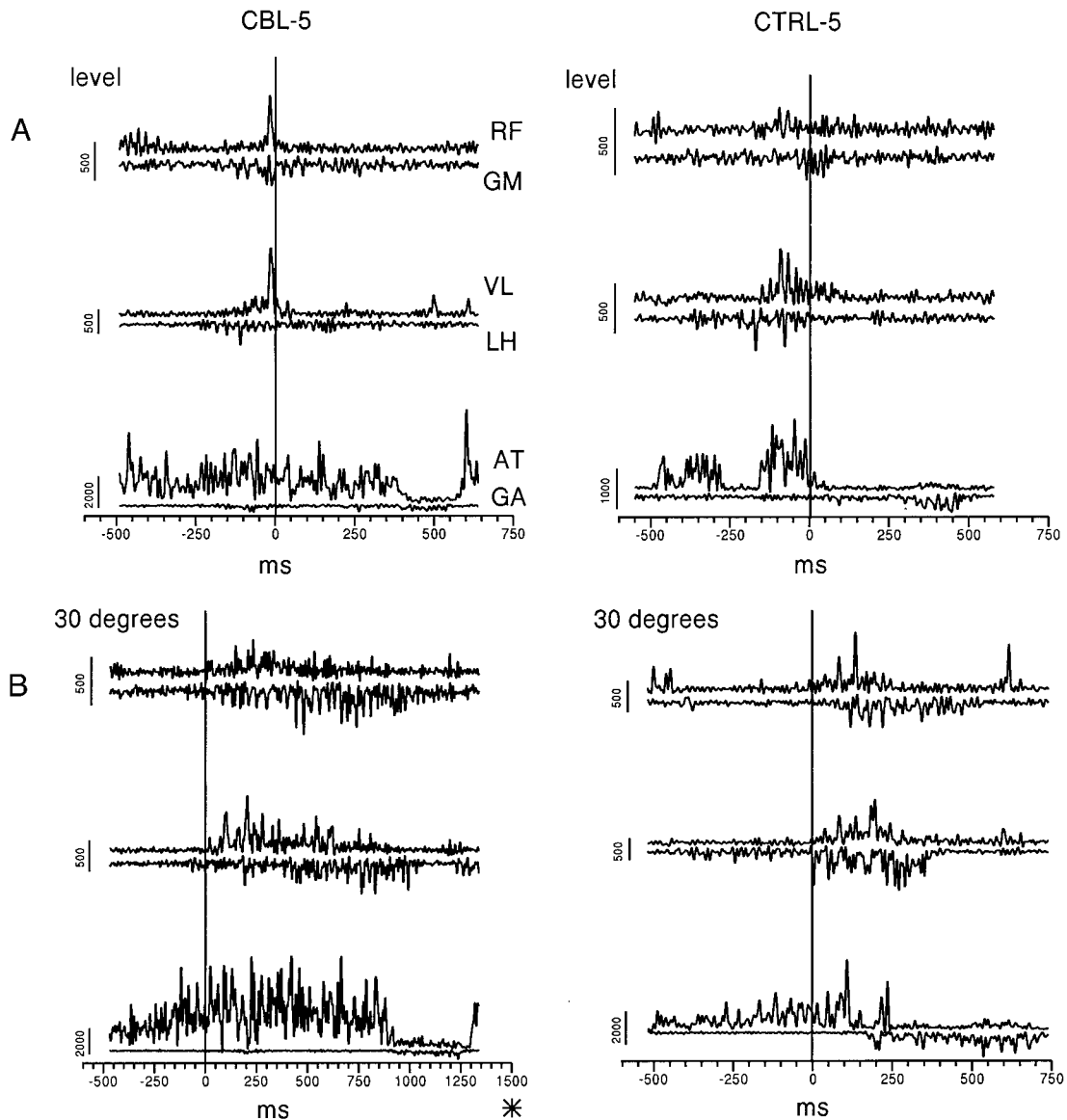


FIG. 6. Electromyographic (EMG) records from trials of level (A) and 30° (B) for a cerebellar subject (left) and a matched control subject (right). Plots are drawn from toe off prior to the wedge through toe off from the wedge, thus depicting swing approaching and stance on the wedge. HS is denoted by the vertical lines. Vertical scales are not constant across conditions, as the aim of the figure was to allow for accurate comparison of EMG timing rather than EMG amplitude. The time scale for the 30° cerebellar trial (*) is longer than that for the other 3 trials. RF, rectus femoris; GM, gluteus maximus; VL, vastus lateralis; LH, lateral hamstring; GA, gastrocnemius; AT, anterior tibialis.

ity that became more extreme on higher wedges. They decomposed their leg movement during stance on all surface inclinations, but this was most pronounced on the higher inclinations. Despite these deficits, cerebellar subjects were able to produce temporal shifts of muscle activity and of hip joint kinematics when walking on the 20°/30° wedges compared with the level/10° conditions. Though not identical to controls, major features of these strategy shifts were preserved.

We were surprised to find that subjects with different types of cerebellar damage all showed similar deficits. This lack of difference may be due to the small number of subjects per lesion category and the fact that all subjects showed mild to moderate locomotor deficits. Based on these limitations, it is not appropriate to assume that the absence of particular deficits indicates a total lack of cerebellar participation in control of

these preserved features or that all types of cerebellar damage influence locomotion in a similar manner.

In a previous study, we demonstrated that healthy neurologically normal subjects use two distinct temporal strategies to traverse wedges of different inclinations (Earhart and Bastian 2000). One strategy was used for level and 10° conditions and the other strategy for 20 and 30° conditions. In the present study, cerebellar subjects showed evidence that some, but not all, features of these two strategies were preserved. Cerebellar strategies were similar to those of control subjects in terms of time shifts for peak hip flexion, time shifts for muscle activity in the GM, VL, and AT, and in the general shape of ankle versus knee angle stance plots. Differences between cerebellar subjects and controls were also present, such that there was no clear time shift of peak ankle dorsiflexion, prolonged AT

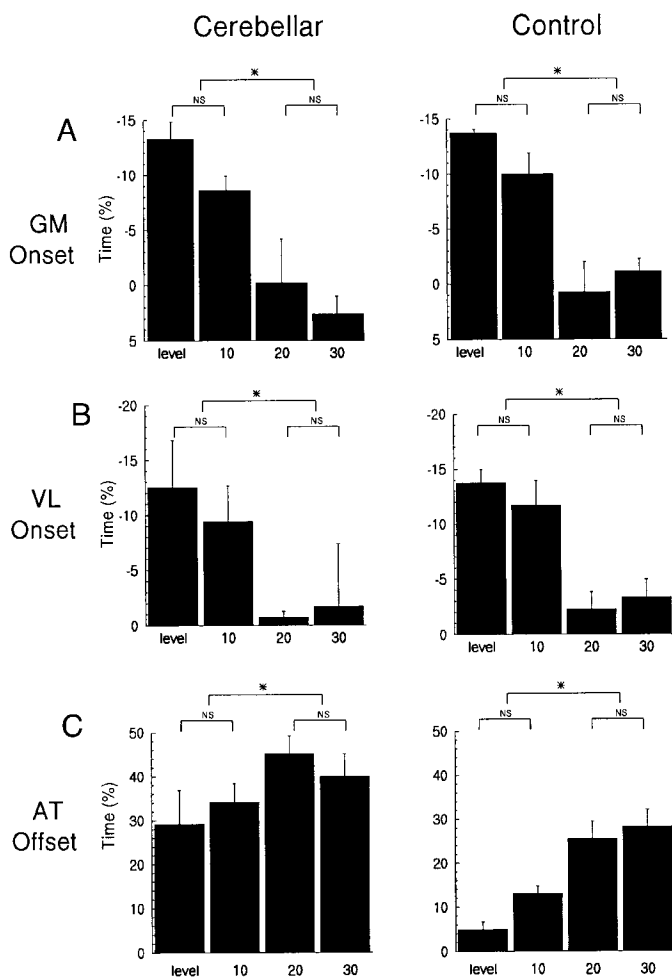


FIG. 7. Average onsets for GM (A) and VL (B), and offsets for AT (C) across conditions for the cerebellar and control groups. Values are means \pm SE expressed as a percentage of the gait cycle; HS is at 0%, swing phase values are negative, and stance phase values are positive. For both groups, onset times for GM and VL and offset times for AT fell into 2 distinct groupings: the patterns for level and 10° were similar (NS, not significant; $P > 0.05$) as were those for 20° and 30°, but differences between level/10° and 20°/30° were significant (*, significant, $P < 0.01$). Also note the trend of prolonged AT activity in the cerebellar group.

muscle activity, and much greater variability of performance. Because many features of the two strategies observed previously in control subjects were preserved in cerebellar subjects, we think it is likely that the basic motor programs for the two strategies are not generated within the cerebellum, a conclusion consistent with that of Dichgans and Fetter (1993). What, then, is the role of the cerebellum in coordination of these walking patterns?

Gain control?

Several studies have suggested that the cerebellum is important for regulating response magnitude. Studies using lesions or cooling of the cat cerebellum have shown hyperflexion of the knee and elbow following cerebellar inactivation (Udo et al. 1979a,b, 1980; Yu and Eidelberg 1983). Dysmetric limb movements have also been reported during upper extremity tasks and walking in humans with cerebellar damage (Gilman et al. 1981; Manto et al. 1994; Topka et al. 1998). The anterior

cerebellum has been implicated in regulation of the magnitude of postural responses (Horak and Diener 1994). Subjects with cerebellar damage have difficulty regulating postural response gain despite their ability to predict perturbation amplitudes (Timmann and Horak 1997).

In the present study, we did not find evidence for impaired control of response magnitude on average. Cerebellar subjects appropriately adjusted joint angles and foot height in response to changes in surface inclination, suggesting that they were able to anticipate contact with the incline. During stance, cerebellar subjects demonstrated normal amplitude scaling of joint angles (except ankle plantarflexion) when each joint was examined in isolation. However, problems with relative scaling among the joints were noted in the snapshots considered for the 30° condition.

Timing?

Another proposed function of the cerebellum is in the timing or sequencing of movements. Ivry et al. (1988) have proposed a critical role for the cerebellum in the central timing of movements. Timmann et al. (1999, 2000) have related throwing inaccuracy of cerebellar patients to variable timing of finger opening. Upper extremity dysmetria in humans with cerebellar damage, and in monkeys following cooling of the dentate, is associated with abnormal timing relationships between agonists and antagonists (Flament and Hore 1986; Hore et al. 1991). However, other studies using inactivation of cerebellar nuclei indicate no disruption in the sequencing of forelimb movements (Milak et al. 1997). Our data suggest reasonably normal timing and sequencing of the walking pattern in the cerebellar subjects. Timing of hip and knee peak angles and onsets of muscle activity in the cerebellar group were similar to the control group. Changes in timing of peak ankle dorsiflexion in the cerebellar group on the 20°/30° conditions resulted from the presence of a dorsiflexion plateau rather than a sharp dorsiflexion peak. Prolonged AT activity in the 20°/30° conditions may be related to this plateau and may be a compensatory mechanism rather than an inherent timing deficit (see *Decomposition of movement*).

Multijoint coordination?

Damage to the cerebellum results in impaired multijoint coordination, while single joint movements may be relatively preserved (Bastian et al. 2000; Goodkin et al. 1993; Thach et al. 1992a,b). Recordings from the interposed nuclei of monkeys showed that cells there fire strongly during multijoint movements but fire little in response to single-joint movements (van Kan et al. 1993). Electrical stimulation in some regions of the baboon dentate elicits compound, but not simple, movements (Rispoli-Padel et al. 1982). Inactivation of each of the deep cerebellar nuclei in monkeys caused greater impairments in compound than in simple movements (Thach et al. 1992a). These results, along with our own, support the concept that the cerebellum is critical for multijoint coordination.

The cerebellar subjects in this study demonstrated abnormalities in the relative movement of multiple joints during walking, particularly during stance on the steeper inclines. The relative positions of the three joints at the time of peak dorsiflexion during stance on the 30° wedge were highly variable

and, on average, showed a different relationship in the cerebellar group than noted for the control group. Cerebellar subjects were unable to consistently reproduce the same relationship among the joints across trials. We propose that these deficits during stance on the steepest wedges reflect a deficit in the ability to appropriately coordinate multiple joints when specific constraints are imposed at one joint and adjustments must be made at other joints to accommodate these constraints. This result is consistent with the hypothesis that the cerebellum plays a role in accounting for the influences of movement at one joint on other joints in the limb (Bastian et al. 1996).

Decomposition of movement

Similar deficits in producing consistent patterns across repeated trials have been noted for throwing and reaching movements (Becker et al. 1990, 1991). One means of improving accuracy of reaching may be decomposition of movement, where one joint is fixed while another is moved relative to it (Bastian et al. 1996; Goodkin et al. 1993). Decomposition is thought to be a compensatory mechanism rather than an inherent deficit because it is most pronounced when cerebellar subjects attempt to move accurately (Bastian et al. 1996). In the present study, cerebellar subjects demonstrated more decomposition of movement than control subjects across all conditions. Differences between groups were most pronounced in the 20 and 30° conditions, where cerebellar subjects showed dramatic increases in decomposition index values for ankle versus hip as well as ankle versus knee, but not for knee versus hip. Decomposition was achieved by fixing the ankle at a plateau level of dorsiflexion during a substantial portion of stance on the steeper wedges. We think that the decomposition noted in the 20 and 30° conditions was likely a compensatory response. First, the decomposition involved fixation primarily at the ankle joint. There is no evidence to indicate that the cerebellum would exert greater influence on the control of distal joints rather than proximal joints (Hore et al. 1991). As such, it is unlikely that ankle control would be selectively impaired following cerebellar damage so the changes seen in the ankle pattern may be compensatory. Second, there are several reasons why it would be advantageous to fix the ankle joint rather than the knee or hip joints when employing a compensatory strategy. The ankle is already partially constrained by the inclined surface, so stabilization may be easier at this joint than the other freely moving joints. The ankle is the joint located furthest from the center of mass, so that a small movement at the ankle would result in a large movement of the center of mass. Fixation of the ankle may help to reduce potential instability associated with large movements of the center of mass. Finally, fixation of the foot in dorsiflexion during stance may allow for the foot to remain flat on the surface for a larger portion of stance. This full-sole contact would provide a larger and more stable contact area between foot and floor.

Summary

Many features of the two basic strategies used by healthy subjects to traverse inclined obstacles were preserved in these mildly to moderately ataxic subjects, suggesting that the cerebellum may not be essential for selection of the basic pattern

for these strategies. However, cerebellar subjects demonstrated abnormal interjoint coordination during this task, showing increased decomposition of movement and greater variability, particularly during stance on the steepest wedges. Our results suggest that the cerebellum may play a key role in coordinating the activity across multiple joints within the limb during walking. A subject with impaired multijoint coordination may employ decomposition as a means of simplifying movement control when environmental demands exceed the individual's residual coordinative abilities. In the case of walking over inclines, fixation of the ankle, rather than the hip or knee, is employed as a means of reducing movement complexity.

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