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RESEARCH ARTICLE

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Spatio-temporal and kinematic analysis of pointing movements performed by cerebellar patients with limb ataxia

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Abstract Three patients with cerebellar limb ataxia and three age-matched controls performed arm-pointing movements towards a visual stimulus during an experimental procedure using a double-step paradigm in a three-dimensional space. Four types of trajectories were defined: P1, single-step pointing movement towards the visual stimulus in the initial position S1; P2, double-step pointing movement towards S1; P3, double-step straight pointing movement towards the second position S2; and P4, double-step pointing movement towards S2 with an initial direction towards S1. We found that the cerebellar patients, as well as the controls, were able to modify their motor programs, but with impaired timing, severe anomalies in the direction and amplitude of the changed movement trajectories and alteration of the precision of the pointing movements.

Key words Cerebellar patients · Double-step paradigm · Pointing movement · Kinematic analysis · Human

Introduction

Cerebellar lesions can result in a number of abnormalities in voluntary limb movements directed towards a visual target. These are characterized by errors in the direction, range and velocity of movement and have been described in the literature using terms such as decomposition of movement, dysmetria and dyssynergia (Holmes 1917). There is evidence to suggest that one of the roles of the cerebellum is to coordinate the timing of different components of complex voluntary movements, and some of the abnormalities observed following cerebellar damage could be interpreted as deficits in timing mechanisms. For example, in a study involving patients with motor dysfunction due to a variety of different causes including parkinsonism, peripheral neuropathy, cerebellar lesions and lesions of cortical motor areas, only the cerebellar patients showed deficits in both the production and the perception of tasks requiring precise timing (Ivry et al. 1988; Ivry and Keele 1989).

The timing of the triphasic pattern of electromyographic (EMG) activation during rapid, accurate single-joint movements is disrupted in patients with cerebellar lesions (Hallett et al. 1975, 1991; Hore et al. 1991). More recently, Manto et al. (1994, 1995) showed that both the timing and the amplitude of the antagonist activity used to brake a voluntary movement was abnormal in subjects with cerebellar dysfunction.

During relatively simple pointing movements to a target, there is prolongation of both reaction time (RT) and movement time (MT) in cerebellar patients. A more demanding task, which requires precise timing of the different components of the movement, involves the use of double-step (ds) visual stimuli. In this paradigm, a second visual stimulus at a different location is presented during the RT following an initial stimulus. This requires the subject to modify the planned trajectory after the initial preparation for the movement has commenced. A number of studies have shown that both monkeys and normal human subjects are capable of timing these modifications so that the finger arrives accurately at the second target with very little delay in the movement (Gottsdanker 1973; Megaw 1974; Georgopoulos et al. 1981; Gielen et al. 1984a, b; Massey et al. 1986; van Sonderen et al. 1988, 1989, 1991).

In this study we used a ds paradigm to examine the kinematics of target-directed arm movements in a group of patients with cerebellar disease causing in-coordination and dysmetria of the limbs. We did not analyse the eye movement during the arm movement, because the aim of our study was not to demonstrate that there is a disruption in the eye-hand motor system in cerebellar patients, as it

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Table 1 This table shows the main clinical features of the two groups of subjects; the controls and cerebellar patients. *C1, C2, C3* cerebellar patients, *F* female, *L* left, *M* male, *N1, N2, N3,* control subjects, *R* right, (+ light, ++ mild, +++ moderate, severity of dysmetria)

Group	Controls			Cerebellar patients		
Subject	N1	N2	N3	C1	C2	C3
Gender	F	M	M	F	M	M
Age	34	55	38	42	48	53
Handedness	R	R	L	R	R	R
Dysmetria	–	–	–	+	+++	+++
Duration (years)	–	–	–	2	8	10

has been well demonstrated already by some authors (Miall et al. 1987; Brown et al. 1993; Cody et al. 1993; Van Donkelaar and Lee 1994). We wanted to determine first of all whether cerebellar patients were capable of producing any modification of the motor program once planning had commenced following the initial stimulus. If modifications do occur, is the timing appropriate and similar to that which occurs in the controls? Recognizing that errors in movement trajectory would already be present during the initial part of the movement, does the increased complexity posed by the introduction of a second target exaggerate the abnormalities resulting from the cerebellar dysfunction? We anticipated that the results of these experiments might provide further insights into the role that the cerebellum plays in incorporating visual information into the motor programs controlling pointing movements.

Materials and methods

Subjects

The subjects consisted of three patients with late-onset cerebellar ataxia (Table 1). The basis of the selection was the existence of a bilateral dysmetria in the clinical examination. The neurological examination show isolated cerebellar signs in all cases. The patients exhibited neither intention tremor nor any head tremor. We did not find clinically any evident, abnormal voluntary eye movement. All patients had magnetic resonance imaging (MRI) scans that showed diffuse cerebellar atrophy. Three healthy volunteers served as controls. The study was approved by the appropriate ethics committee and performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki. All subjects were told of the purpose and procedures of the investigation and gave their informed consent.

Apparatus

The subject was seated at a constant distance from a vertical square screen (30 × 30 cm) with two light-emitting diodes (LEDs) 5 mm in diameter. A platform was placed 16 cm in front of the bottom of the screen to mark the starting position of the hand. The screen consisted of a printed circuit of a 5-mm grid, which recorded the spatial and temporal coordinates of the first contact of the finger (Fig. 1A). The two LEDs were situated on the same horizontal axis 10 cm to either side of the centre of the screen and at 30 cm height from the platform. The programmed sequences were controlled on-line by a micro-processor system, which also recorded the data and carried out the statistical processing.

The arm movement was analysed by the ELITE system, which is based on real-time processing of TV images. A marker attached at the extremity of the index finger of the dominant hand was detected by a digital dedicated image processor. Two TV cameras placed above the subject in a frontal plan at a distance of 3 m allowed a three-dimensional (3D), 100-Hz analysis of kinematics by a computer. The software performed 3D reconstruction, derivative variables computing (linear velocities and accelerations) and graphic representations (Fig. 1B).

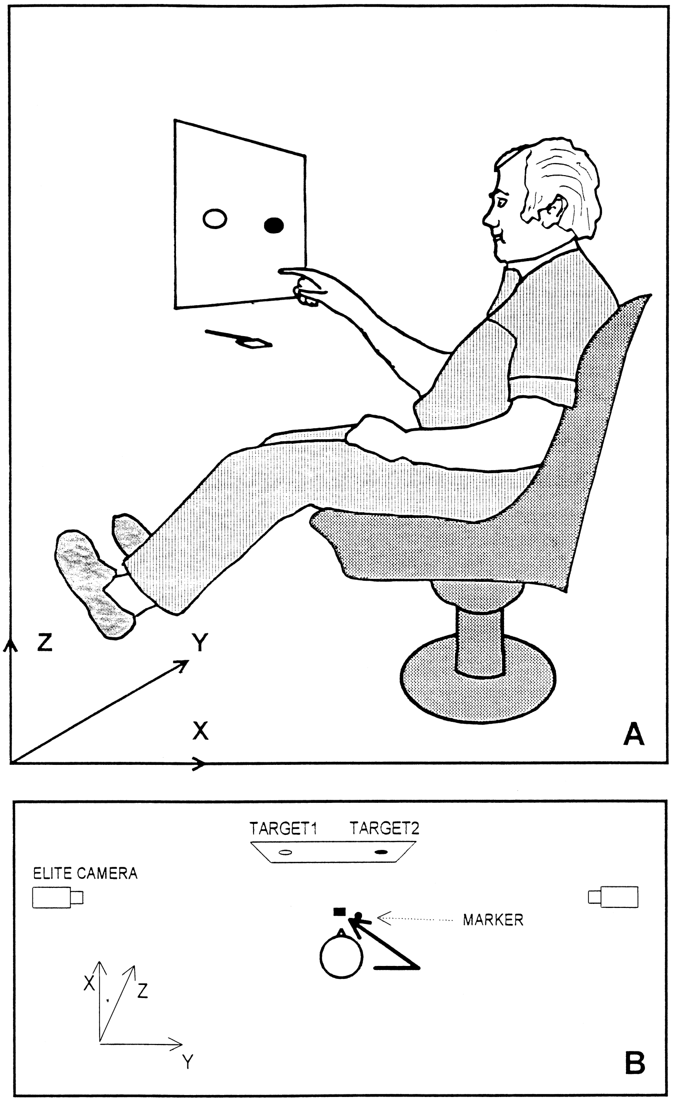

Fig. 1 **A** The apparatus and the two possible locations of the visual stimulus. **B** The ELITE system by which the pointing movements were analysed in a three-dimensional space (*x*, *y*, *z*)

Experimental procedures

The basic configuration presented to the subject was a single-step (ss) or a ds stimulus. In this study, the subjects used only their dominant hand. The contact of the index finger on the platform triggered the appearance of the luminous target in its first position after a preparatory period (PP), defined as the interval between platform pressing and signal onset; this PP could have any one of four durations (500, 1000, 1500 and 2000 ms), which were randomly distributed. The RT was defined as the time interval between the signal onset and release from the platform. The movement time (MT) was defined as the time interval between the release from the platform and the first contact of the index finger on the board. For each pointing movement, the rectangular coordinates were recorded and used to calculate the spatial error (*E*), which was given by the distance between the target and the position of the finger contact. The recordings by the ELITE system started with the appearance of the visual stimulus and reconstructed the trajectories in each plane (sagittal *xz*, frontal *yz* and horizontal *xy*), the displacement velocities and accelerations in the three axes (*x*, front-back direction; *y*, right-left direction and *z*, vertical direction) against time of the positions of the marker. The velocity trajectories were analysed in the *y* direction where the greatest change was seen.

In 25% of trials, ds stimuli were presented to the subject: the visual stimulus jumped from the initial position (S1) to the second position (S2) during the RT after a random time (*T*). These times were 50 ms, 75 ms and 100 ms for control subject N2, 50 ms, 100 ms and 150 ms for N1 and N3, and 100 ms, 200 ms and 300 ms for the patients. The delay values were choosen for each subject so that the control value represented approximatly one-third of the mean value of the RTs calculated during practice trials. After the practice trials, two blocks of 60 stimuli each were presented with a rest of several minutes after each set of 30 trials. In the first block the position of the initial target was always on the same side of the board (left or right). This was reversed in the second block. The 45 ss stimuli (75% of 60) and the 15 ds stimuli (25% of 60) were randomly presented. The whole session took approximately 1.5 h for the controls and 2 h for the patients.

Instructions to subjects

Subjects were instructed to point as rapidly as possible to the perceived position of the target. No instruction was given concerning accuracy of the movement, because the aim of the study was not to focus on the accuracy but rather on the timing of rapid, target-directed movements. The subjects used only their dominant hand.

Data analysis

Four types of trajectories were identified. P1, ss pointing movement towards the visual stimulus S1; P2, ds pointing movement towards S1, that is, the subjects reached directly to the first target, even though the target shifted to a second location; P3, ds straight pointing movement towards S2, that is, the subjects reached directly to the second target with no movement towards the first target; and P4, ds pointing movement towards S2 with an initial direction towards S1, that is, the movement went towards the first target initially, but changed direction en route when the target shifted position (Fig. 2). We calculated the proportion of P2, P3 and P4 in the case of ds pointing movement. RT, MT and *E* were analysed for all types of trajectories. Furthermore, from P4 trajectories, we calculated the following values (Fig. 3): *time of change of trajectory* (CT), measured on the *y* displacement curve defined as the time interval between the appearance of the visual stimulus in S1 and the change in direction of the trajectory; *reaction time to the second stimulus* (RT2), defined as the time interval between the appearance of the visual stimulus in S2 and the change in the direction of trajectory; RT_2b, defined as time interval between the onset of the movement and the change in the direction of the trajectory; the ratio *T/RT*. Student's *t*-test was used for the statistical analysis.

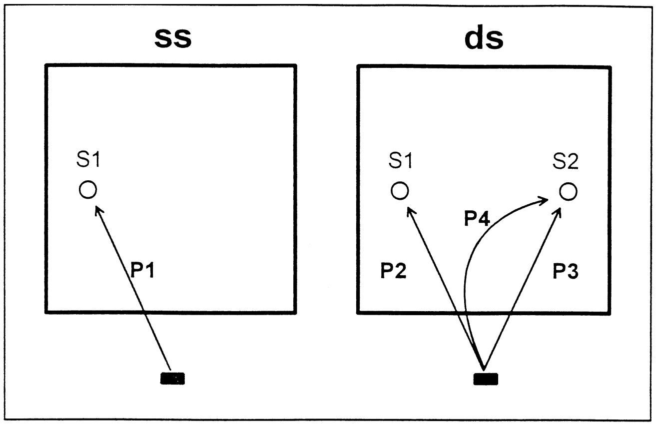

Fig. 2 The *two squares* represent the vertical board and the *small black rectangles* represent the platform from which the movement starts. On the *left*, the single-step (*ss*) paradigm with only the first position of the visual stimulus (*S1*) and one type of trajectory (*P1*); on the *right*, the double-step stimulus (*ds*) with the first position (*S1*) and the second position (*S2*) of the visual stimulus and the three types of trajectories (*P2*, *P3*, *P4*)

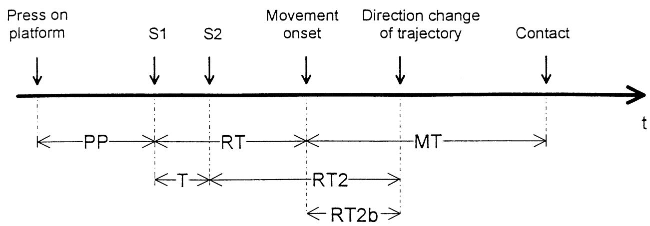

Fig. 3 The different data calculated on the P4 trajectory (*MT* movement time, *PP* preparatory period, *RT* reaction time, *RT2* reaction time to the second stimulus, *RT2b* time interval between the onset of the movement and the direction change of the trajectory, *S1* initial position of the visual stimulus, *S2* second position of the visual stimulus, *t* time, *T* random time)

Table 2 Mean reaction time values (milliseconds) in controls and patients for the four types of trajectory P1, P2, P3 and P4

		Count	Mean	Standard error
Controls	P1	270	278	3
	P2	15	250	12.1
	P3	16	340	9.7
	P4	56	238	6.1
	Total	357	273	2.8
Patients	P1	268	600	10.2
	P2	21	645	31.1
	P3	32	775	34.5
	P4	33	548	28.7
	Total	354	614	9.3

Results

Spatio-temporal variables

We did not find any difference in the means for RT, MT and *E* for the movements towards targets on the same side or opposite side of the screen from the pointing hand.

Table 3 Comparison of reaction time (RT) between the 2 groups for each type of trajectory (P1, P2, P3, P4) given in the *squares shaded grey*; and comparison of RT in each group of the different types of trajectory (the results for control are given in the *lower left* of the table and the results for patients are given in the *upper right* of the table)

		P1	P2	P3	P4	P2+P3+P4	Patients
P1	*t*	30.46	1.18	5.5	1.7	2.5	P1
	p	0.01		0.01		0.05	
P2	*t*	1.34	10.26	2.6	2.2		P2
	p		0.01	0.05	0.05		
P3	*t*	4.8	6.51	8.78	5		P3
	p	0.01	0.01	0.01	0.01		
P4	*t*	2.59	0.06	8.3	12.71		P4
	p	0.01		0.01	0.01		
P2+P3+P4	*t*	0.57				17.59	P2+P3+P4
	p					0.01	
Controls		P1	P2	P3	P4	P2+P3+P4	

Table 4 Mean movement time values (milliseconds) in controls and patients for the four types of trajectory P1, P2, P3 and P4

		Count	Mean	Standard error
Controls	P1	270	201	2
	P2	15	205	8.9
	P3	16	188	6.7
	P4	56	226	7.2
	Total	357	205	2
Patients	P1	268	505	8.5
	P2	21	464	13.2
	P3	32	544	26.8
	P4	33	658	35.6
	Total	354	520	8

Table 6 Mean spatial error values (millimeters) in controls and patients for the four types of trajectory P1, P2, P3 and P4

		Count	Mean	Standard error
Controls	P1	270	3.2	0.1
	P2	15	21.5	4.6
	P3	16	40.1	1.2
	P4	56	36.2	1
	Total	357	10.8	0.2
Patients	P1	268	4.9	0.1
	P2	21	11.1	2.9
	P3	32	39.9	0.6
	P4	33	41.2	0.9
	Total	354	11.8	0.2

Table 5 Comparison of movement time (MT) between the two groups for each type of trajectory (P1, P2, P3, P4) *given in the squares shaded grey*; and comparison of MT in each group of the different types of trajectory (the results for controls are given in the *lower left* of the table and the results for patients are given in the *upper right* of the table)

		P1	P2	P3	P4	P2+P3+P4	Patients
P1	*t*	34.8	1.33	1.48	5.61	3.42	P1
	p	0.01			0.01	0.01	
P2	*t*	0.4	14.88	2.28	4.2		P2
	p		0.01	0.05	0.01		
P3	*t*	1.57	1.6	9.26	2.54		P3
	p			0.01	0.05		
P4	*t*	4.47	1.4	2.7	14.95		P4
	p	0.01		0.01	0.01		
P2+P3+P4	*t*	3.02				17.96	P2+P3+P4
	p	0.01				0.01	
Controls		P1	P2	P3	P4	P2+P3+P4	

Therefore, we will present the combined results for movements towards targets on either side of the screen.

Reaction time

Table 2 shows the mean values for RT in controls and patients. First, we compared the mean RTs in controls and patients for the four types of trajectory, then we compared the mean RTs between controls and patients for each of the four types of trajectory. RTs were significantly prolonged in the patients for all types of trajectory in comparison with controls. RTs for trials in which the direction of the trajectory changed (P4) were significantly longer in both controls and patients (Table 3).

Movement time

Table 4 shows the mean MT values for controls and patients. MT were significantly prolonged in the patients for all types of trajectory in comparison with controls.

Table 7 Comparison of spatial error (*E*) between the two groups for each type of trajectory (P1, P2, P3, P4) given in the *squares shaded grey*; comparison of *E* in each group of the different types of trajectory (the results for controls are given in the *lower left* of the table and the results for patients are given in the *upper right* of the table)

		P1	P2	P3	P4	P2+P3+P4	Patients
P1	*t*	10	6.7	78	74.8	30.6	P1
	p	0.01	0.01	0.01	0.01	0.01	
P2	*t*	15.9	1.99	11.7	11.9		P2
	p	0.01		0.01	0.01		
P3	*t*	74.7	4	0.15	1.2		P3
	p	0.01	0.01		NS		
P4	*t*	68.2	4.9	2	3.53		P4
	p	0.01	0.01	0.05	0.01		
P2+P3+P4	*t*	44.1				0.5	P2+P3+P4
	p	0.01					
Controls		P1	P2	P3	P4	P2+P3+P4	

Table 8 Different times (milliseconds) recorded from the P4 trajectories (*CT* time of change of trajectory, *RT* reaction time to the first stimulus, *RT2* reaction time to the second stimulus, *RT2b* time interval between the onset of the movement and the direction change of the trajectory, *T* random time)

	Count	CT	RT2	RT2b	T/RT
Controls	56	376±37	287±27	112±32	36±14
Patients	33	821±240	563±214	273±119	48±14

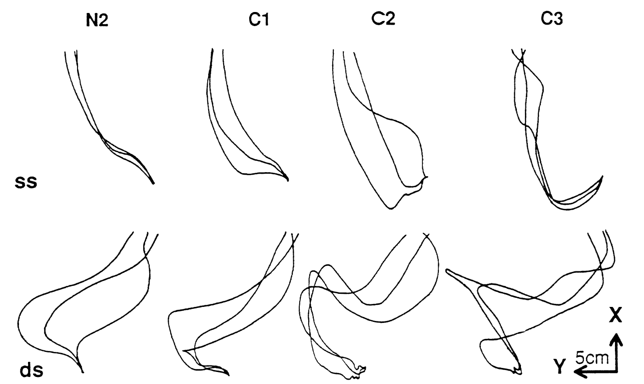

Fig. 4 Three examples of trajectories in one control (*N2*) and the cerebellar patients (*C1*, *C2*, *C3*) for the ss and ds paradigms. The trajectories were analysed on the *x*-*y* plane

MT for trials in which the direction of the trajectory changed (P4) were significantly longer in both controls and patients. Table 5 shows the comparison of MT between the two groups for each type of trajectory.

Spatial error

Table 6 shows the *E* values in controls and patients. *E* was significantly prolonged in the patients in comparison with controls for the trajectories P1 and P4. Table 7 shows the comparison of *E* between the two groups for each type of trajectory.

Trajectories

Table 8 shows the different times (in milliseconds) recorded from the P4 trajectories in the two groups. In the control subjects, movements directed to the single stimulus, target followed a slightly curved trajectory. The initial path for the movement was very consistent from trial to trial and the full trajectories were fairly similar for different trials (Fig. 4).

In the cerebellar patients, there was considerable variability in trajectories for the ss-stimulus trials. In subjects C2 and C3, the initial part of the movement was in a lateral direction, or in some cases even in a direction away from the screen.

The lower traces in Fig. 4 show sample trajectories for the ds-stimulus trials in which the subjects produced a mid-course change in direction to point towards the second target (P4 trajectories). In the normal subject (N2), the position and timing of the direction change varied from trial to trial (depending on the RT to the second stimulus), but the finger followed a smooth curve until the new trajectory was established and then proceeded more or less directly to the second target.

The cerebellar patients were able to change the direction of movement in response to the second stimulus, although this was accomplished in a smaller proportion of the ds-stimulus trials than in the control subjects. The trajectories both before and after the change in direction were highly irregular. In some trials, particularly for subjects C2 and C3, there appeared to be an over-correction, so that the initial part of the revised movement took the hand away from the screen, requiring further corrections.

Velocities

Further information concerning the timing and other characteristics of the movement, particularly for the trials with a mid-course change in direction, can be obtained by examining the velocity profiles in the *y*-axis (across the screen). Examples of these velocity traces are shown in Fig. 5. For the ss-stimulus trials, the velocity profiles for

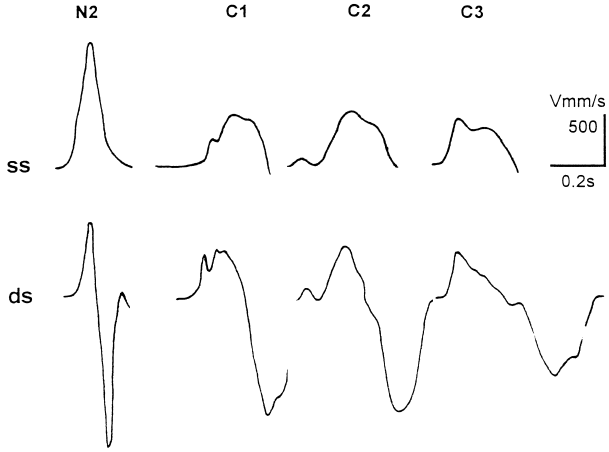

Fig. 5 Examples of velocity trajectories in one control (*N2*) and the cerebellar patients (*C1*, *C2*, *C3*) are shown for the ss and ds paradigms. The velocity trajectories were analysed on the *y*-direction (*V* velocity)

the control subjects were symmetrical, with fairly identical acceleration and deceleration phases. In the patients, the velocity peaks were considerably lower than in the controls, and the deceleration phases were prolonged.

In the ds-stimulus trials, the point at which velocity drops to zero after the initial peak indicates the change in movement direction. In the normal subjects, the two components of the velocity profiles (before and after the direction change) showed approximately symmetrical acceleration and deceleration phases. The examples shown for cerebellar patients C1 and C2 show approximately symmetrical velocity profiles, althougth peak velocities are lower and the timing of the change in direction is delayed. In subject C3, the deceleration phase after the initial velocity peak was considerably prolonged.

Discussion

This study dealt with spatio-temporal and kinematic analysis of pointing movements in 3D space in cerebellar patients with limb ataxia, using a ds-stimulus paradigm. On the ss trials, we found significant abnormalities in patients, including prolonged RT, MT and increased *E*. These results confirm previous studies (Nakamura and Taniguchi 1980; Inhoff et al. 1989; Diener and Dichgans 1992; Jahanshahi et al. 1993; Bonnefoi-Kyriacou et al. 1995). Kinematic features of movement trajectories and velocity in ss as well as in ds gives an excellent tool with which to analyse the limb ataxia. Our study showed that, in the ss trials, the trajectories were obviously less regular than the ones of the controls and that the trajectories were even more irregular in term of direction and range in the ds trials. This inability to produce pointing movements with consistent directions from trial to trial have been described in cerebellar patients performing 2D or 3D throwing movements (Becker et al. 1990) and in a cerebellar patient performing multi-joint reaching movements (Becker et al. 1991). The velocity curves in cerebellar patients were also very different from the ones of the controls. In cerebellar patients, the delayed velocity peak with smaller amplitude, the shorter acceleration part and the much longer deceleration duration when compared with controls reflected a severe perturbation in the time course of the pointing movements. These results correlate with the study of Brown et al. (1990) which showed the disturbances of temporal structures of voluntary movements in cerebellar patients performing visually guided, step tracking movements about the elbow. In a kinematic and EMG study of cerebellar patients with limb dysmetria, Hore et al. (1991) found during movement of the elbow, wrist and finger that, compared with normal movements of the same peak velocity, cerebellar movements had decreased peak accelerations and increased peak decelerations, with a characteristic asymetry of the movement. For Hallett et al. (1991), prolonged acceleration time was the most dramatic kinematic abnormality and correlated with the degree of ataxia. Normal subjects adjust their trajectory according to change in target location (van Sonderen et al. 1988). Van Sonderen et al. (1989) observed an ongoing adjustment, which occurs once the generation of motor programme has started, and they hypothesized that: "The generation of the motor programme starts after the target presentation and that the activation levels for the appropriate muscles are continuously adjusted to move the hand in the direction of the current internal representation of the target". When a second target appears in a different location during the RT to S1, there are three possible responses. If S2 occurs early during the RT there is sufficient time for the subject to reprogramme the movement so that the trajectory is directed towards S2. If S2 appears relatively late the motor commands have progressed to the point where it is too late to introduce any modification and the finger moves to the point where S1 was located. Between these extremes is the situation where the initial part of the movement is directed towards S1 but a mid-course change in trajectory occurs in response to the appearance of S2. In our control subjects this happened when S2 occurred about one-third to one-half of the way through the RT to S1. Given all the other abnormalities of programming pointing movements in cerebellar patients, the first question was whether they were capable of producing any modification of trajectory in response to a change in target location. Our results clearly showed that the patients were able to do this on some trials, providing that S2 was delayed to occur in a range of 25–65% of the way through the prolonged RT. However the proportion of ds trials in which the patients were able to modify the trajectory was considerablely smaller than in the control subjects. Furthermore, the variability and irregularities in trajectory that were apparent in the ss trials became even more marked when a change in trajectory in response to S2 occurred. In some cases the appearance of S2 resulted in a marked deviation from the optimal trajectory so that the hand initially moved back towards the subject rather than continuing towards the new location on the screen. This might suggest that, with-out a normally functioning cerrebellum, there is an inappropriately large correction for a visually detected change in target location. The inappropriate correction for a visually detected change in target location as seen in this study in the irregularities in movement trajectories seems directly connected to the inability of the cerebellar patients to scale muscle activity and to oppose or assist the interaction torques that are caused by other moving linked segments (Bastian et al. 1996). The cerebellar patients probably begin with an adequate spatial perception of a target; this is supported by studies in monkeys (Liu and Chambers 1971). Inappropriate trajectories have been considered as the consequence of improper formulation or execution of a motor plan (Massaquoi and Hallet 1996). We have shown in this study that cerebellar patients are capable of producing modification of the motor program once planning has commenced following the initial stimulus, but with inappropriate timing and exaggeration of the abnormalities in movement trajectories when a second target is introduced.

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