Physiological analysis of simple rapid movements in patients with cerebellar deficits

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Abstract

Patients with cerebellar deficits made elbow flexion movements as rapidly as possible for three different angular distances. Electromyographic activity of biceps and triceps and the kinematics of the movements were analysed. Results were compared with those of normal subjects making both rapid and slow movements. In the patients, the first agonist burst of the biceps was frequently prolonged regardless of the distance or speed of the movement. The most striking kinematic abnormality was prolonged acceleration time. The pattern of acceleration time exceeding deceleration time was common in patients but uncommon in normal subjects. The best kinematic correlate of the duration of the first agonist burst was acceleration time. Altered production of appropriate acceleration may therefore be an important abnormality in cerebellar dysfunction for attempted rapid voluntary movements.

Although it is clear that the cerebellum contributes to the coordination of voluntary movement, the nature of the contribution is not well understood. One prevalent idea is that the cerebellum acts like a clock to control the timing of movement.[1-4] The analysis of simple rapid, or ballistic, movements appears to be a good approach to probe the timing of movement. Such movements are accomplished in normal persons in a stereotypical fashion with predictable timing.[5-7] The pattern, sometimes called the "triphasic pattern," is characterised on the electromyogram (EMG) by an initial burst of muscle activity in the agonist of the movement that lasts 50–100 ms. The agonist then falls silent and the antagonist muscle produces a burst that also lasts for 50–100 ms. Then the agonist again resumes activity, often in the form of a burst. For angular movements of less than 30 or 40 degrees, the timing of the movement changes only a little; larger movements are accomplished with higher velocities. For movements of larger amplitude, the duration of the movement may increase and the EMG bursts would be prolonged.[8-9] Slow movements are characterised by long EMG bursts that parallel the duration of the movement.[5]

A previous study[10] of attempted ballistic movements in patients with cerebellar deficits

showed the triphasic pattern of EMG activity, but the duration of the first agonist burst was prolonged. The relationship between the EMG features and the kinematics of the movements was not analysed, but it was speculated that this prolonged burst might predispose the patients to hypermetric movements. The prolonged activity in the agonist muscle would move the limb for a longer time than was expected. This study examines the kinematic consequences of the prolonged EMG activity. Early in the investigation, we found that patients with cerebellar deficits often make movements that are either hypermetric or slower than normal, or both. Both large amplitude movements and slow movements in normal subject are characterised by prolongation of the first agonist EMG burst. Therefore we found it necessary to reconsider the question of whether patients with cerebellar deficits actually produce long first agonist bursts.

Patients and methods

We studied 13 patients, nine men and four women, aged 16 to 80 years, with cerebellar deficits in the arm, but no other neurological signs. Cerebellar signs in the arm were dysmetria, dysdiadochokinesia, and excessive rebound. Tremor was not prominent in any patient. Patients such as these are not common, as most who have clinical cerebellar signs also have corticospinal tract signs, sensory disturbance, weakness, or peripheral neuropathy. The dominant arm was studied unless the cerebellar deficit was restricted to or significantly worse in the nondominant arm. The patients had a variety of aetiological diagnoses (table 1). Sixteen normal subjects, all men, aged 20 to 75 years, were also studied. All but one of these subjects was right handed, and the dominant arm was studied.

Patients and normal subjects were asked to make flexion movements of their elbows as rapidly as possible. They sat in a chair facing an oscilloscope screen with their arms strapped into a lightweight splint composed of an upper arm and forearm portion connected at the level of the elbow joint with a low-friction hinge. In the hinge was a potentiometer that allowed measurement of the elbow angle by a voltage. The beam on the oscilloscope screen was made into a line, the height of which indicated the elbow angle. Subjects were asked to make movements of 10, 20, and 30 degrees by specifying the beginning and end-

Table 1   Clinical description of patients with cerebellar deficits

Patient	Age/sex	Diagnosis	Degree of ataxia	Handed/ hand studied
1	80/F	Familial cerebellar atrophy; severe cerebellar atrophy on CT scan	Severe	R/R
2	68/M	Carcinomatous cerebellar degeneration; CT scan normal	Severe	R/R
3	21/M	S/P surgical excision of astrocytoma of cerebellum	Severe	R/R
4	38/M	OPCA; cerebellar and pontine atrophy on CT scan	Severe	R/R
5	60/M	OPCA; small pons, medulla, and cerebellum on CT scan	Severe	R/R
6	40/F	Cerebellar degeneration; cerebellar atrophy on CT scan	Moderate	R/R
7	55/F	OPCA; cerebellar atrophy on CT scan	Moderate	R/R
8	58/M	Bilateral cerebellar infarctions	Moderate	R/R
9	30/M	Multiple sclerosis	Moderate	R/R
10	43/M	Multiple sclerosis; CT scan normal	Mild	R/R
11	35/M	Left cerebellar haemorrhage, demonstrated by CT scan	Mild	R/L
12	16/M	"Ramsay Hunt"; cerebellar atrophy on CT scan	Mild	R/L
13	40/F	Multiple sclerosis	Mild	L/R

OPCA = olivopontocerebellar atrophy.

ing heights of the line. The command to initiate movement was a verbal request to make the movement itself as rapidly as possible. Subjects were told that accuracy was less important than speed. The EMG was recorded with pairs of surface electrodes placed longitudinally over the bellies of the biceps brachii and triceps brachii. Subjects performed some practice trials until they were comfortable, and then data from a minimum of ten single consecutive trials were collected for each movement distance. The agonist and antagonist EMG and the elbow position voltage were recorded by computer. Most normal subjects were also asked to make a set of ten movements at each distance with variable slow speeds. Five normal subjects were also asked to make a set of ten movements of 60 degrees, both rapidly and more slowly.

We analysed the EMG for the duration and "amount" of activity within bursts. The beginning of the first agonist burst was the onset of EMG activity. The end of the first agonist burst was determined on the basis of two criteria applied in order. According to the first criterion, the end was indicated by any substantial decline in the amplitude of agonist EMG activity relating to the pause between the two agonist bursts. The pause was often accompanied by the peak of activity in the antagonist muscle. We looked for the end of the first agonist burst from 20 ms before to 20 ms after the start of the first antagonist burst, which could be recognised not only by initiation of activity but also by restart of activity after a pause or a sudden increase in activity. The second criterion specified that there should be a simultaneous decrease in the magnitude of the EMG to less than 10% of peak magnitude lasting for more than 10 ms. It was expected that this decrease would occur before the peak velocity of the movement (the peak velocity was defined as the highest velocity at or before the time the acceleration first went to zero). If there was no end to the first agonist activity before 50 ms after the

Figure 1   Individual elbow flexion movements from a normal subject moving as fast as possible. The four traces in each panel are, from top to bottom, rectified biceps EMG, rectified triceps EMG, angular position, and velocity. The line under the EMG of the biceps record indicates the measurement of the first agonist burst duration in that record. A shows a nicely formed triphasic pattern. B–D show co-contraction of the antagonist during the first agonist burst. In B, it is not clear whether the antagonist activity is only the first antagonist burst with an early start or whether there is some co-contraction activity also. In C, there is a clear separation of the first agonist burst and co-contraction activity; in D, the separation is not clear.
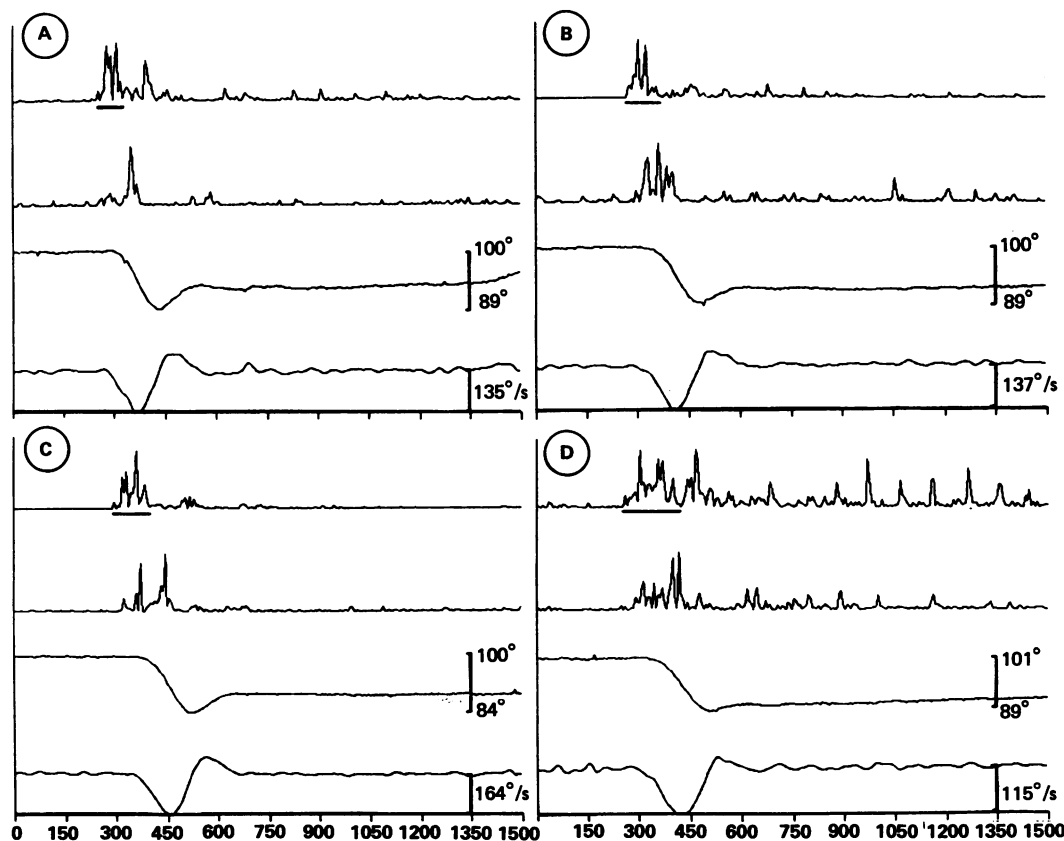
Figure 2   Individual
elbow flexion movements
from normal subjects
moving slowly. The four
traces in each panel are
in the same order as in
fig 1. The line under the
EMG of the biceps
record indicates the
measurement of the first
agonist burst duration in
that record. Note the
prolongation of the first
agonist burst compared
with the records in fig 1
and the fragmentation of
some of the bursts. B
shows co-contraction
activity of the agonist
with the latter part of
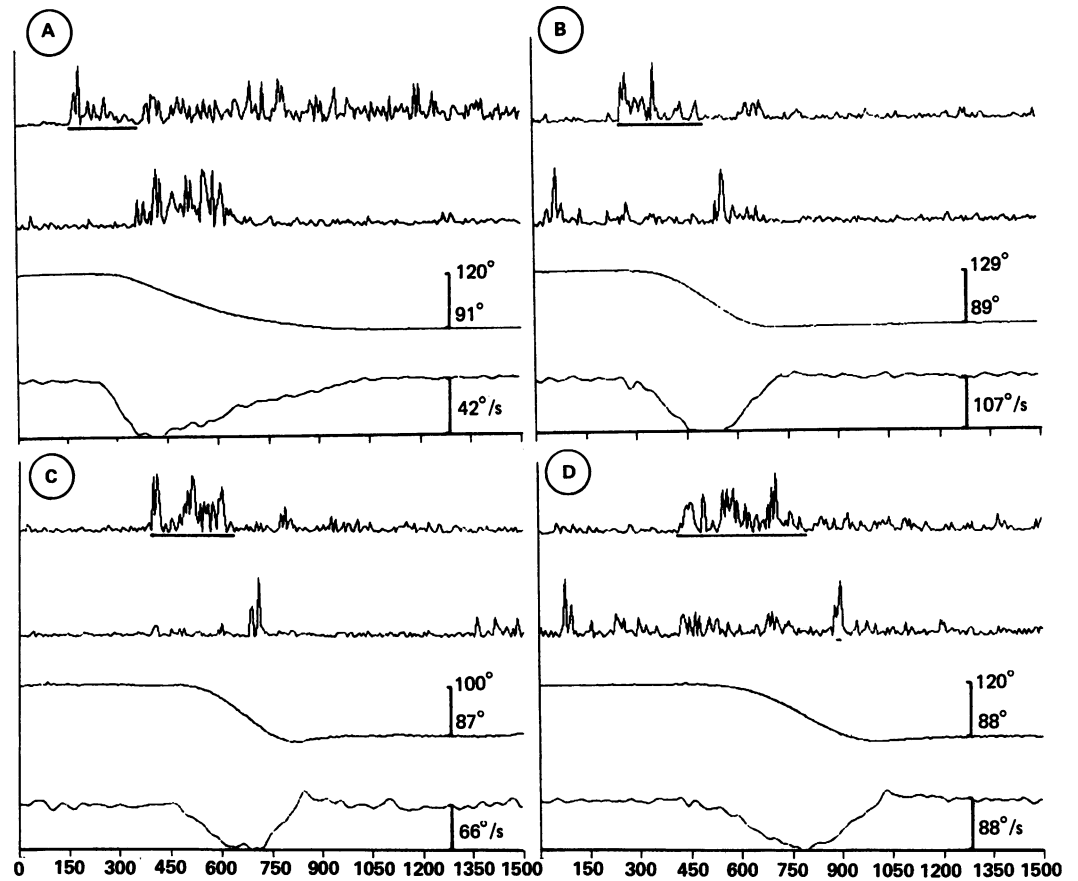the first antagonist burst.

Figure 3   Individual
elbow flexion movements
from three patients
moving as fast as possible
(A and D, patient 9; B,
patient 3; C, patient 5).
The four traces in each
panel are in the same
order as in fig 1. The line
under the EMG of the
biceps record indicates
the measurement of the
first agonist burst
duration in that record.
The first agonist burst is
mildly prolonged in A
and markedly prolonged
in B and C. In C, the
first agonist burst is
fragmented. D shows the
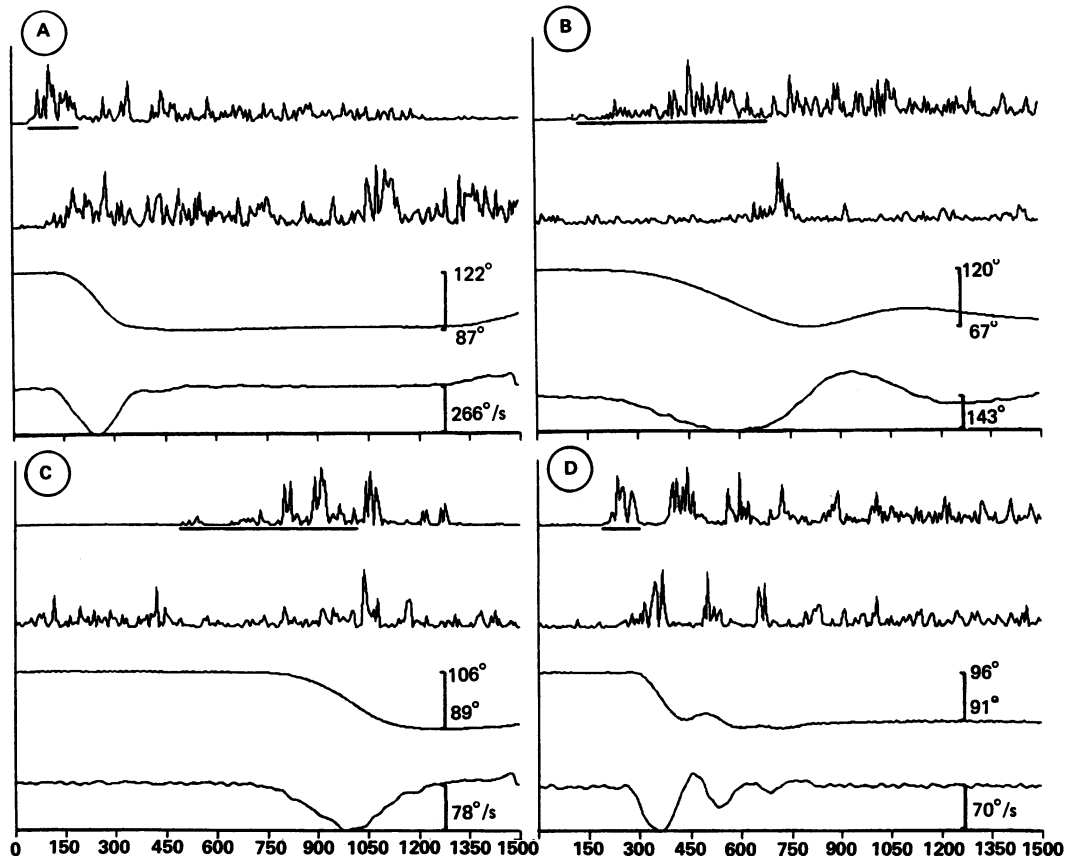relatively rare event of a
tremorous movement.

Table 2   Duration of first agonist burst (ms) and coefficient of variation (CV)

Subject	10°			20°			30°		
	Mean	CV	N[a]	Mean	CV	N[a]	Mean	CV	N[a]
				Normal subjects[b]					
N1	70·0	32·9		135·0	12·2		130·0	19·2	
N2	90·0	22·9		94·5	18·0		93·6	10·6	
N3	88·0	22·9		91·8	26·9		115·0	28·0	
N5	78·9	38·0		89·0	43·8		115·0	9·1	
N6	75·0	14·7		77·0	10·4		75·0	18·0	
N7	117·0	17·7		117·0	16·2		118·0	19·5	
N8	94·0	27·1		106·0	23·2		113·0	20·5	
N9	70·0	24·3		86·3	12·3		92·5	11·6	
N10	70·0	10·6		72·5	16·6		83·8	9·0	
N11	66·3	16·9		65·0	24·6		88·8	10·1	
N12	72·5	27·6		88·8	7·3		92·5	17·3	
N13	72·5	21·4		81·3	13·5		81·3	12·7	
N14	82·0	14·9		93·2	19·3		85·6	17·1	
N15	86·4	14·7		90·4	10·5		103·2	14·4	
N16	84·7	12·1		97·2	10·8		101·6	14·5	
Mean	81·2	21·2		92·3	17·7		99·3	15·4	
SD	13·1	7·8		17·3	9·2		16·0	5·2	
				Patients[c]					
1	235·0*	31·5	4/6	218·9*	68·5*	9/11	165·7*	64·0*	7/11
2	166·7*	29·4	6/7	218·0*	31·7	5/5	252·5*	8·7	4/7
3	ND			ND			461·1*	13·8	9/10
4	ND			119·0	16·8	10/10	ND		
5	380·0*	34·2	10/10	386·0*	34·2	10/10	262·0*	38·2*	15/15
6	188·4*	35·3	10/10	242·0*	30·1	10/10	273·6*	29·5*	10/10
7	210·0*		2/11	172·5*	30·3	4/4	170·0*	41·8*	4/9
9	100·0	31·0	10/10	119·0	16·0	10/10	136·0	13·2	10/10
10	116·3*	10·3	8/8	125·0	10·4	8/8	123·7	7·3	8/8
11	97·5	33·8	4/4	156·7*	42·1*	9/10	129·0	13·2	10/10
12	89·2	21·2	10/10	105·6	26·2	10/10	139·6*	14·0	10/10
13	101·6	24·0	10/10	129·2	28·2	10/10	151·6*	44·1*	10/10
Mean	168·5	27·9		181·1	30·4		205·9	26·1	
SD	91·0	8·1		82·7	15·6		101·4	18·6	

ND, not done.
[a]Number of movements where EMG burst was measured/number of movements included in analysis (the kinematic measurements in tables 3 and 4 were taken from all movements included in analysis).
[b]EMG artifact in subject N4 prohibited any measurement.
[c]Patient 8 showed only tonic EMG patterns.
*Value exceeds the normal mean plus 2·5 SD.

peak velocity, then the agonist burst was said to be "tonic" and not to have a measurable duration. The amount of activity was calculated by rectifying and continuously integrating the EMG record. The difference in value of the continuously integrated trace between the beginning and end of the burst gave the amount. We attempted a similar analysis for the first antagonist burst, but discontinued it because of considerable difficulties producing reliable data (see Results).

A patient value was considered abnormal if it deviated more than 2·5 standard deviations from the normal mean.

Results

First antagonist burst

Even for the normal subjects, it was not a simple matter to determine the duration of the first antagonist burst. The detail of the EMG pattern of the first antagonist burst was not constant, even for the same person attempting the same movement with similar kinematics (fig 1). Defining the start of the antagonist burst was difficult. The start was clearest when the burst began about the time that the first agonist burst stopped (fig 1A). "Co-contraction" of the antagonist with the agonist often occurred, and while it was usually possible to separate the co-contraction from the antagonist burst itself (fig 1C), it was difficult to decide whether the start of the antagonist contraction was a co-contraction or an early start of the burst (fig 1B). Many times, definite co-con-

traction antagonist activity continued directly into the burst, making it impossible to define the burst onset (fig 1D). Defining the end of the antagonist burst was also a problem; the EMG activity did not clearly stop, but continued in a tonic fashion, sometimes with the appearance of co-contraction with the second agonist burst (figs 2B and 3A).

Finally, we decided that we could not establish objective criteria that would produce consistent data, and hence we will not report on the duration of the first antagonist burst.

First agonist burst

The duration of the first agonist burst was also not easy to determine, but by establishing the criteria described above we were able to measure it with some objectivity. Again, in the normal subjects, the EMG pattern of the first agonist burst was not constant, even for the same person attempting the same movement with similar kinematics (fig 1), but it was less variable than that of the first antagonist burst. The onset of the first agonist burst was nearly always clear, since the background was quiet. However, the end of the first agonist burst was often difficult to determine for several reasons. More often than not, the agonist did not fall completely silent at the "end" of the first burst. The activity would sometimes reduce substantially but continue at a low tonic level, or it would stop very briefly and then resume. At times, it would appear that some of the agonist activity was co-contraction activity with the antagonist. The problem was even more dif-

Figure 4 Three-dimensional plots of duration of the first agonist burst as a function of distance moved and peak velocity. Data from normal subjects (A) are plotted as small round points. Data from patients 5, 2, and 1, respectively (B–D), are plotted as large round points on the background of the normal data.
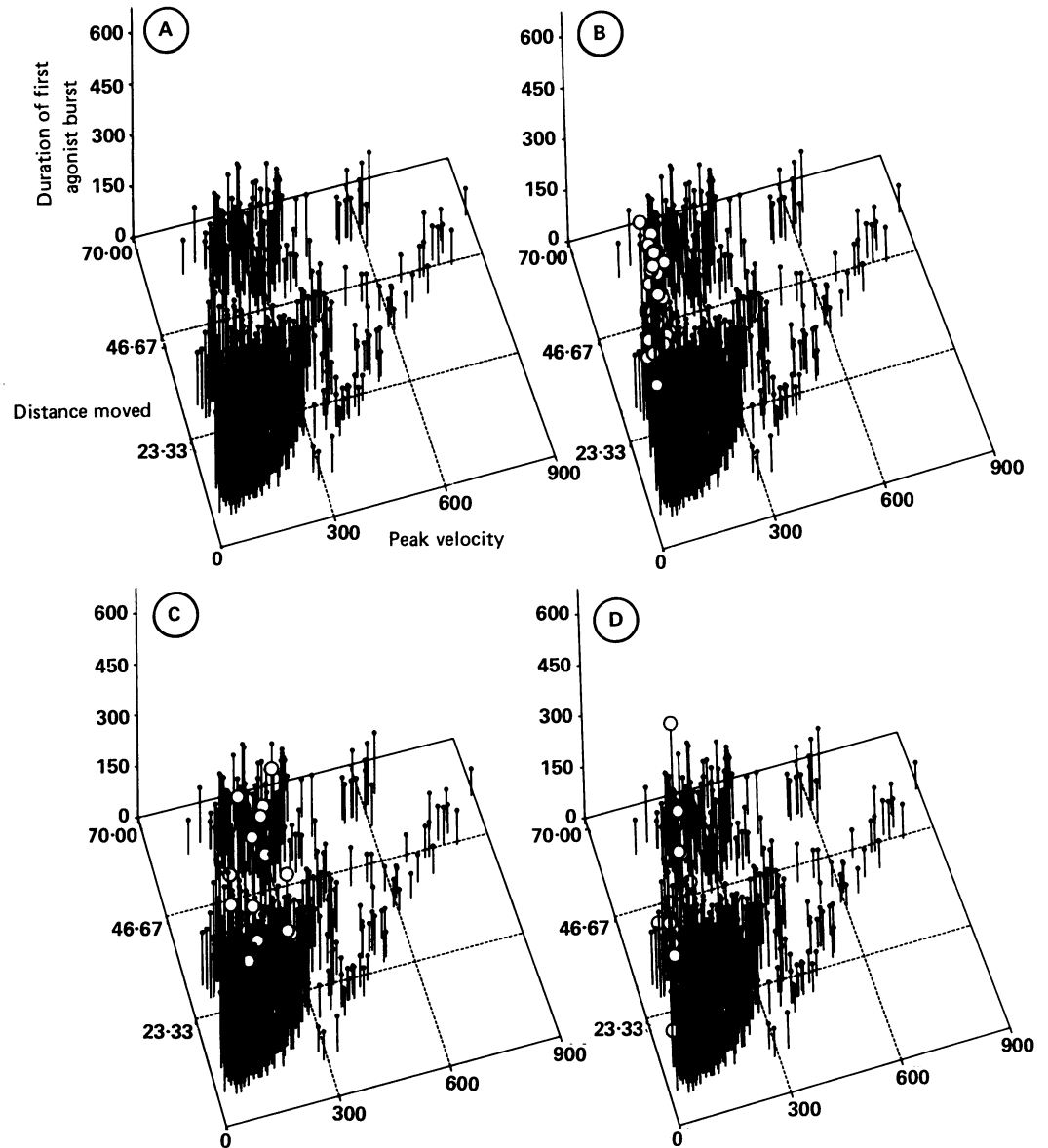
ficult with the normal slow movements (fig 2). Additionally, the first agonist burst fragmented, that is, there were substantial brief drops in EMG amplitude well before the first antagonist burst began or well before the peak velocity, or both (fig 2C, D).

These problems of measurement were even greater in the patients' records, with fragmentation of the first agonist burst being a common finding (fig 3C). Only rarely did patients have movements characterised by tremor, and it was not a significant problem when it occurred (fig 3D). Not infrequently, the first agonist burst in patients was underestimated because of the fragmentation and co-contraction problems.

The number of movements for which the bursts could be measured and the total number of acceptable movements (used for kinematic analyses) are shown in table 2. Usually, all trials were measurable for the normal subjects, but in an occasional set of ten trials, one trial might not be measurable. In some patients, it was possible to measure all ten trials, but in many it was not. Some movements were performed so badly, including for example, movements in the wrong direction, that the trial was discar-

ded. For some movements, the burst was not measurable because of a tonic or highly fragmented pattern.

Normal subjects making rapid movements of 10, 20, and 30 degrees had first agonist bursts averaging between 80 and 100 ms (table 2). Ten of 12 patients had first agonist bursts that were significantly longer than normal for at least one movement distance (one patient had long-duration agonist activity from which bursts could not be identified). Two-way analysis of variance (ANOVA) with burst duration as the dependent variable, and distance aimed and subject group as factors, showed a highly significant difference between the normal subjects and the patients ($p < 0.001$), but no difference with distance aimed and no significant interaction. Regression analysis showed no changes in the first agonist burst duration with age. A Kruskal-Wallis test showed that patients who had shorter bursts had milder ataxia than did those who had prolonged bursts ($p < 0.001$).

The duration of the first agonist burst was prolonged in normal subjects when the movements were slow or greater than 30

Table 3 Accuracy of movement (degrees) and coefficient of variation (CV)

Subject	10°		20°		30°	
	Mean	CV	Mean	CV	Mean	CV
			Normal subjects			
N1	12·8	13·3	23·3	14·6	30·6	9·2
N2	16·0	38·1	29·6	17·9	47·3	12·1
N3	12·4	21·8	21·7	17·1	33·8	9·8
N4	7·7	9·1	17·4	14·4	29·2	5·8
N5	11·6	25·0	20·3	16·3	30·9	8·7
N6	19·0	8·9	29·3	19·1	38·8	9·8
N7	12·1	37·2	27·2	6·6	40·1	5·5
N8	12·1	17·4	21·7	4·1	31·0	11·6
N9	9·9	11·4	19·5	12·9	29·8	5·9
N10	13·9	14·6	19·9	15·3	31·1	5·4
N11	12·1	19·5	21·8	12·2	28·0	12·1
N12	11·8	18·0	19·9	12·2	29·9	8·8
N13	10·6	12·2	18·8	7·4	25·1	8·6
N14	12·8	17·2	20·9	16·9	32·8	11·8
N15	12·0	24·0	20·4	16·9	30·5	8·9
N16	13·1	12·5	22·4	13·0	31·1	4·8
Mean	12·5	18·8	22·1	13·5	32·5	8·7
SD	2·5	8·8	3·6	4·3	5·4	2·5
			Patients			
1	6·8	66·2*	27·5	41·8*	33·3	22·8*
2	26·9*	27·1	38·8*	24·0	36·6	32·5*
3	ND		ND		53·8*	8·9
4	ND		30·7	18·2	ND	
5	17·4	20·1	20·8	10·1	32·3	6·5
6	16·0	23·8	25·1	16·3	37·8	16·4*
7	19·5*	68·7*	24·7	30·0*	42·1	17·6*
8	14·1	18·4	26·8	27·2	42·4	11·6
9	7·3	34·2	19·8	14·6	32·6	19·0*
10	14·6	13·7	25·0	8·8	32·2	10·2
11	10·0	29·0	21·0	9·5	31·5	8·6
12	15·5	25·2	28·2	22·7	46·2*	25·1*
13	17·7	28·2	34·8*	10·6	36·2	19·6*
Mean	15·1	32·2	26·9	19·5	38·1	16·6
SD	5·7	18·3	5·7	10·1	6·9	7·8

ND, not done.
*Value exceeds the normal mean plus 2·5 SD.

degrees. Accordingly, the burst durations in the patients might have been longer than normal because their movements were slow or hypermetric. From this point of view, it is essential to assess burst durations for movements matched for distance and velocity. To do this, we looked at the first agonist burst duration for individual movements as a simultaneous function of angular distance and peak velocity (fig 4). The three-dimensional plot of this relationship is a surface. The surface is flat and parallel to the plane of origin for rapid movements of less than 30 degrees. The edge of the surface, which comes from the most rapid movements that can be done for each distance, runs diagonally across the plane because longer movements can be accomplished at faster velocity. The surface moves upward with longer or slower movements. (The normal distribution surface has some "holes" that originate from not collecting data on any movements between 30 and 60 degrees and not collecting data on medium speed movements at 60 degrees.) In nine of 12 patients (all but patients 4, 7, and 11), at least some of the first agonist burst durations exceeded the normal distribution.

Kinematics of the movements

We defined the end point of the movement as the point at which the velocity first became zero. By this definition, normal subjects attempting to move rapidly made movements that were slightly hypermetric. The movement typically first overshot the desired endpoint and then quickly backtracked (fig 1). This type

of trajectory was not present in normal subjects making slow movements (fig 2) or in any of the patients (fig 3), Even with this difference in trajectory, two-way ANOVA showed that the patients were more hypermetric than the normal subjects (p < 0·001) (table 3). The variability of the distance moved was assessed with the coefficient of variation of the distance moved for each distance aimed. Two-way ANOVA showed that the variability was greater in the patients than in the normal subjects (p < 0·001) and significantly different for the different movements (more variability with shorter movements).

It was apparent that many of the patients' movements were slow. We studied several parameters to characterise this deficit. The total movement time was the best measure, with eight of the 13 patients having at least one value statistically above normal. We divided the movement time into the acceleration time (beginning of the movement until the peak of the velocity) and the deceleration time (peak of the velocity until zero velocity). (If a corrective extension movement immediately followed the flexion movement, then the movement "decelerated" for some time longer than the deceleration time, but this additional time was not relevant to our end point.) The normal range of acceleration times was very tight, and nine of the 13 patients had abnormal acceleration times (table 4). Two-way ANOVA with acceleration time as the dependent variable, and distance aimed and subject group as factors, showed a highly significant difference between normal subjects and patients (p < 0·001), but no difference with distance aimed and no significant interaction. A Krusk-
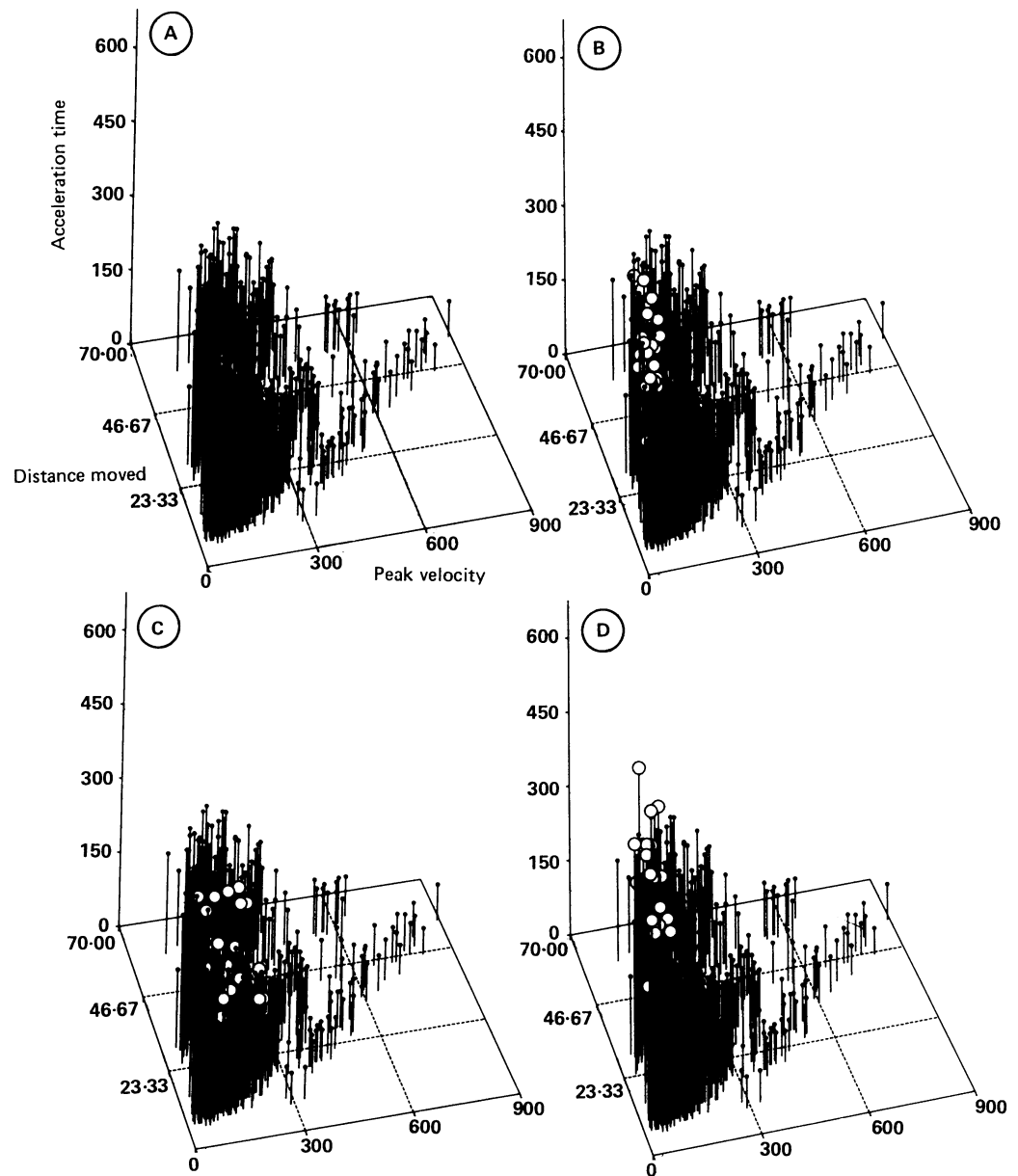
Table 4 Acceleration time (ms)

Subject	10°	20°	30°
		Normal subjects	
N1	76·0	90·0	105·0
N2	65·6	67·5	62·8
N3	72·5	77·5	81·5
N5	58·0	68·5	84·0
N6	65·0	69·0	79·5
N7	81·0	85·5	88·4
N9	65·0	77·5	101·3
N10	71·3	86·3	100·0
N11	62·5	82·5	93·8
N12	62·5	82·5	93·8
N13	60·0	80·0	87·5
N14	89·2	124·0	128·0
N15	57·2	68·8	84·4
N16	85·8	90·4	104·8
Mean	69·4	81·8	91·8
SD	10·3	14·5	15·5
		Patients	
1	441·7*	256·4*	345·5*
2	137·1*	132·0*	178·6*
3	ND	ND	301·0*
4	ND	109·0	ND
5	250·0*	263·0*	270·7*
6	129·2*	134·7*	167·5*
7	155·5*	192·5*	185·6*
8	284·4*	336·2*	407·5*
9	95·0	103·0	121·0
10	80·0	88·7	88·7
11	80·0	83·0	80·0
12	131·5*	171·3*	182·7*
13	136·3*	170·6*	187·2*
Mean	174·6	170·0	209·7
SD	109·8	79·4	101·6

ND, not done.
*Value exceeds the normal mean plus 2·5 SD.

Figure 5 Three-dimensional plots of acceleration time as a function of distance moved and peak velocity. Data from normal subjects (A) and patients 5, 2, and 1, respectively (B–D), are plotted as in fig 4.
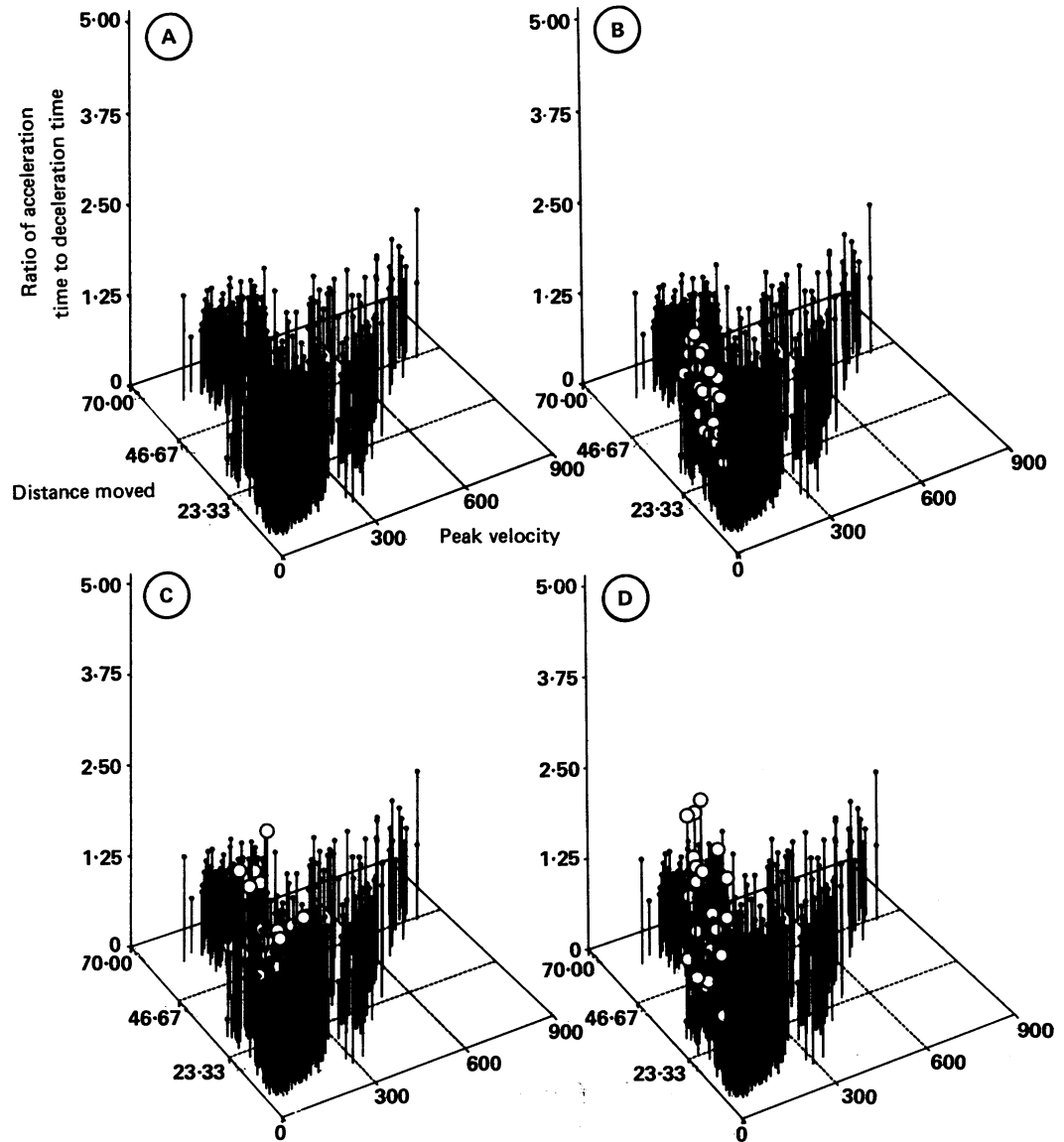

al-Wallis test showed that the severity of ataxia correlated with acceleration time (p = 0·03). Many of the deceleration times were also abnormal, but not as dramatically; the values exceeded the normal range in nine patients.

Rapid movements of normal subjects yielded a symmetric velocity curve, the acceleration time being roughly equal to the deceleration time (fig 1). The attempted rapid movements of the patients, however, often showed a longer acceleration time than deceleration time (fig 3). Normal subjects making slow movements used a variety of strategies, but most commonly showed a longer deceleration time than acceleration time (fig 2A). To quantify these observations, we studied the ratio of acceleration time to deceleration time. The normal subjects had mean ratios of 0·94, 0·99, and 0·96 for the fast 10, 20, and 30 degree movements, respectively. For the slow movements, the ratios dropped to 0·88, 0·83, and 0·83, respectively. The ratios for the patients were 1·17, 1·09, and 1·08, respectively. ANOVA showed no significant difference between the patients' movements and the normal fast movements,

but there was a significant difference (p < 0·05) when the patients' movements were compared with the normal slow movements.

For careful assessment of the apparent abnormalities of acceleration time and the ratio of acceleration time to deceleration time in the patients, it is necessary to look at individual movements. The acceleration time might well be expected to vary in different types of movements. With respect to the issues being considered here, we might expect the acceleration time (like the duration of the first agonist burst) to increase with distance moved and slowness of the movement. To explore this possibility, we studied acceleration times in individual movements as a simultaneous function of the distance moved and the peak velocity (fig 5). The three-dimensional plot of this relationship is a surface with a shape similar to that plotted for the duration of the first agonist burst, that is, flat and parallel to the plane of origin for fast movements of less than 30 degrees and rising for longer or slower movements. Ten patients showed long acceleration times with this type of plot,

Figure 6 Three-dimensional plots of the ratio of acceleration time to deceleration time as a function of distance moved and peak velocity. Data from normal subjects (A) and patients 5, 2, and 1, respectively (B-D), are plotted as in fig 4.

Figure 7 Acceleration time as a function of agonist duration for patients and normal subjects using the mean values for distance aimed.

indicating a kinematic abnormality even when movement distance and peak velocity were considered simultaneously. The three patients (4, 7, and 11) who had normal acceleration times were the same ones who had a normal duration of the first agonist burst.

We used a similar three-dimensional plot to study the ratio of acceleration time to deceleration time for individual movements as a function of the distance moved and the peak velocity

(fig 6). The normal surface was flat (with an approximate value of 1·0) for all fast movements and sloped downward with slower movements. An abnormality of the ratio was found for nine patients.

Relationship of EMG to kinematic parameters
We studied the correlations in individual subjects between the first agonist burst duration and various kinematic parameters, including distance moved, peak velocity, movement time, acceleration time, deceleration time, and mismatch (the difference between the aimed distance and the actual distance moved). The best correlate to the first agonist burst duration was the acceleration time. The correlation ranged from 0·11 to 0·86 (mean, 0·53) for normal subjects and 0·06 to 0·75 (mean, 0·26) for patients. Using the mean values for pooled data on distance aimed for normal subjects and patients, ANOVA showed a highly significant relationship (p < 0·001) between acceleration time and agonist duration (fig 7).

Discussion
Current theories of motor control suggest that movements are first planned, then executed,

and finally corrected during the execution if the goal of the movement is not being achieved. There is evidence that the lateral part of the cerebellum is involved in the initial planning and that the intermediate part of the cerebellum is involved in the correction phase. Theoretically, there would be no time for correction if a movement was made quickly enough, and such a movement would be called ballistic. Simple monophasic movements made as rapidly as possible, at least in normal subjects, are often thought to be ballistic. Certainly, the triphasic EMG pattern that characterises these movements is generated centrally as part of the original motor plan, because the pattern is present even in the absence of peripheral feedback, as in deafferented patients. The details of the first agonist burst have provided some information on the planning of movement and the function of the lateral cerebellum.

Obtaining exact measurements of burst duration in individual movements is difficult, and the method has often been merely intuitive. By adopting stringent criteria, we attempted to make the method more objective. One difficulty in the measurements, which indicates an interesting feature of the physiology, is the co-contraction bursts, including the antagonist with the first agonist burst and the agonist with the first antagonist burst. The presence or absence of the co-contraction activity has no immediately apparent effect on the kinematics. The effect of the co-contraction may largely relate to stiffness of the joint during the movement. Although it was not specifically studied, we have the impression that co-contraction activity is lessened when the subject is relaxed.

This study confirms and extends the earlier finding[10] that the duration of the first agonist burst is prolonged in patients with cerebellar deficits. Difficulty in measurements, including tonic patterns, indicates that the abnormality is even more severe than the measurements indicate. This feature is manifest for many of the patients even when slowness of movement and hypermetria are considered. The degree of the abnormality correlates with the extent of ataxia. The abnormality has been seen also with movements of the top joint of the thumb[11] (Hallett et al, unpublished observations). An extensively studied primate model of patients with cerebellar deficits is reversible dysfunction of the dentate nucleus (or other deep nuclei) created with a cooling probe.[12] The animals show movements that resemble those of patients, with long first agonist bursts in simple movements[13 14] or prolonged EMG bursts with alternating movements.[15] Additionally, cooling the dentate nucleus produces prolongation of the phasic discharge of neurons in motor cortex that fire before movement.[16]

The most dramatic kinematic abnormality is the prolonged acceleration time. The abnormality holds even in relation to the deceleration time and in relation to slow movements made by normal subjects. The movement trajectory rises slowly to a peak velocity, followed by abrupt deceleration. An abnormally long acceleration time correlates with the degree of ataxia and with the duration of the first agonist burst. A similar kinematic abnormality is found in the primate model,[14 17] indicating that movements made by animals with cerebellar lesions are not merely "inappropriate selection or triggering of an otherwise normal motor program."[14] We would echo this view.

An abnormality of velocity in patients with cerebellar disease is in accord with studies of single-cell behaviour in cerebellum recorded from performing primates. In several different circumstances, cellular activity has correlated with the velocity of movement as the best kinematic variable.[18-20]

It seems reasonable to find that the primary EMG abnormality, prolonged duration of the first agonist burst, and the most dramatic kinematic abnormality, prolonged acceleration time, correlate with each other. The implication is that the limb is accelerated for approximately the same time as the agonist EMG is active. The error of producing a long acceleration time would seem the most fundamental problem of simple movements for patients with cerebellar deficits. Hypermetria should be a common resultant movement error unless there is compensatory reduction in the magnitude of the acceleration or compensatory increase in the magnitude of the deceleration. Acceleration is reduced in the patients, but this may be part of the problem. Deceleration magnitude may be increased, and this may be compensatory. Kinematic features are more easily measured than EMG parameters and may be preferred in future studies.

There is no clear explanation of the prolonged first agonist burst and the prolonged acceleration time. The kinematics indicates a reduction in the peak force exerted by the agonist muscle. In one respect, this is a paradoxical observation, since none of these patients was clinically weak as tested with tonic muscle contraction. This reduction in force was not due to the antagonist, since neither gross co-contraction nor inappropriately early activation of the antagonist was a notable feature. There are two possible reasons for the discrepancy in peak force and the prolongation of the first agonist burst and acceleration time. One of these may be that the primary abnormality is an inability to turn off the agonist muscle activity. We and others have previously speculated that the cerebellum, as an "inhibitory machine," has a role in terminating the first agonist burst properly. If the burst is obligatorily long, then to make movements of the correct distance patients must compensate by reducing the magnitude of the activity.

The other possible reason for the prolonged agonist burst is that the patients have difficulty in producing phasic muscle force. This explanation is consistent with the frequent observation that the beginning of the burst grows slowly rather than abruptly as in normal performance. In the most rapid voluntary contractions, spinal motor neurons reach discharge rates as high as 200 Hz. Presumably, the motor units in cerebellar patients do not achieve such

high rates. If the cerebellum contributes to the onset of a voluntary motor command, in cerebellar patients, the descending phasic motor output would be smaller than normal. This has been seen in monkeys trained to make self-terminated reaction time movements where cooling the dentate nucleus led to a decrease in peak velocity of movement.[16] The decreased phasic descending command was reflected in a reduction in the phasic discharge of precentral cells before the onset of movement.

Prolonged duration of EMG bursts during rapid arm movements has been observed in other pathological conditions such as the upper motor neuron syndrome,[21 22] athetosis,[23] Huntington's disease,[24] and dystonia.[25] The physiological dysfunction is not fully understood in any of these conditions, but may well be different from that occurring in cerebellar disturbance. For example, in the upper motor neuron syndrome, there is significant disturbance of the descending command with abnormalities of spatial and temporal recruitment of motor units, and in athetosis there is significant co-contraction of the antagonist muscle. Slowness of movement is not necessarily linked to prolongation of EMG bursts, however, since in Parkinson's disease the burst amplitude is low and the duration is normal.[26 27]

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