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EMG analysis of patients with cerebellar deficits[1]

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SYNOPSIS EMGs from biceps and triceps were recorded during stereotyped elbow flexion tasks performed by 20 patients fulfilling clinical criteria for 'cerebellar deficits' and the data were compared with previously established normal standards. In a fast flexion task, 15 of 18 patients showed prolongation of the initial biceps and/or triceps components, and it is suggested that this abnormality might be an elemental feature of dysmetria. Ten of 14 patients showed the normal pattern of smooth flexion indicating that, with cerebellar deficits, smooth movements are better preserved than fast movements. The timing of the cessation of triceps activity before the initiation of biceps activity in an alternating movement was abnormal in 12 of 16 patients; this abnormality might be an elemental feature of dysdiadochokinesia.

Careful behavioural observations of animals and humans with lesions of cerebellum or cerebellar pathways have documented a variety of motor deficits (Holmes, 1939), analysis of which may lead to a better understanding of normal cerebellar physiology. These deficits include hypotonia, mild weakness and fatiguability, disturbances of associated movements, and certain disorders of voluntary movement. This last category of deficits is associated with a special kind of clumsiness, difficult to characterize, which has been referred to by many terms, including cerebellar ataxia, asynergia, dyskinesia, and incoordination. As suggested by Holmes, the disorders of voluntary movement can be divided into (1) abnormalities of rate, range (dysmetria), direction and force of a movement in one direction at a single joint; (2) abnormalities of the timing including regularity of successive movements (dysdiadochokinesia); (3) abnormalities of the organization of a complex movement (decomposition of movement); and (4) intention tremor. It is commonly believed that all of these symptoms occur together (Growdon *et al.*, 1967), and if this be true it would imply that there may be a single or few elemental abnormalities that underlie them all. A detailed study of simple movements might more easily reveal such elemental abnormalities.

We have previously defined several stereotyped simple movements for clinical use and quantified the associated EMG patterns in a group of normal subjects (Hallett *et al.*, 1975). The movements are fast flexion of the elbow (FF), smooth flexion of the elbow (SF), and fast flexion of the elbow after an isometric contraction of the triceps, in which antagonist inhibition occurs before agonist activity (AI). There is now some understanding of the physiology of such movements, and abnormalities of them, both qualitative and quantitative, might have implications about the elemental pathophysiology. We have studied 20 patients with abnormalities during clinical tests of 'cerebellar function' and though, in many, the lesions affect cerebellar pathways rather than the cerebellum itself, we have grouped them together in this study under the term 'patients with cerebellar deficits'. We have identified certain definite EMG abnormalities in this group.

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METHODS

The clinical data are summarized in the Table. The dominant arm was studied whenever the arms were

equally affected. There was no impairment of strength or sensation unless noted.

The experimental methods have been described previously in detail (Hallett *et al.*, 1975). In brief, surface EMG was recorded from biceps and triceps during stereotyped elbow flexions, the subject attempting to match the step displacement of a line on an oscilloscope screen. Three different protocols were utilized. Subjects were asked to flex the elbow as rapidly as possible (FF, fast flexion) and as smoothly as possible (SF, smooth flexion). In the third protocol, a weight was attached to the arm which pulled in the direction of elbow flexion and required a tonic contraction in triceps as the subject attempted to keep his line at the starting position before the step displacement. The subjects were asked to perform a fast flexion and the relation between the inhibition of triceps activity and the initiation of biceps activity was observed (AI, antagonist inhibition). The EMGs, the position of the experimenter's line and the subject's line, and the velocity of the movement were recorded on an oscilloscope. A permanent record was made by photographing the sweep with a Polaroid camera.

TABLE

CLINICAL DATA AND RELATED EMG FINDINGS

Patient	*Age (yr)*	*Sex*	*Arm*	*B1* (ms)*	*T1* (ms)*	*B1–B2* (ms)*	*SF**	*AI**	*Diagnosis*	*Exam*
I. *Prolonged B1*										
PW	56	F	L	540	80	50	N	N	9 d after resection of metastatic cancer to R cerebellar hemisphere	Severe AT of both arms, L more than R
HS	15	M	R	260	75	80	N	Fair	SD since age 11 yr	Mild AT, rebound, overshoot, dysdiadochokinesia
AS	50	M	R	170	80	105	A	—	Multiple sclerosis	Moderate AT, brisk reflexes
FM	54	M	R	160	95	80	—	Poor	SD for 4 yr	Mild dysmetria, rebound, but no AT
JB	31	M	L	115	75	80	N	Fair	Mild multiple sclerosis	Mild AT, brisk reflexes
			R	90	95	100	N	N		Normal
DS	51	M	R	90	75	130	A	N	10 d after R-sided crural-paresis-and-homolateral-ataxia lacunar stroke	Moderate AT, slight weakness
			L	55	70	95	—	—		Normal
II. Prolonged B1 and T1										
SB	38	F	R	210	100	115	N	Poor	Familial CD beginning in childhood	Marked dysmetria, rebound, and dysdiadochokinesia, but little AT
RP	15	M	R	170	105	75	N	Fair	Very mild dementia and movement disorder for 2 yr	Moderate AT, overshoot, rebound, dysdiadochokinesia, and probable mild dystonia
JG	52	M	R	155	145	145	A	Fair	SD since age 3 yr	Moderate AT, mild weakness and distal sensory loss
RF	22	F	R	145	120	160	N	Poor	SD similar to Friedreich's ataxia since age 11 yr	Moderate AT, and dysdiadochokinesia with areflexia
PG	36	F	R	220	100	110	A	N	Degenerative disease with cerebellar signs and seizures for many years	Mild dysmetria, no AT
RB	21	M	L	155	110	125	—	N	Degenerative disease with cerebellar signs, slight dystonia, mild dementia	Moderate AT, overshoot, dysdiadochokinesia, but no rebound
RC	7	F	L	145	130	115	—	—	3 w after resection of cystic atrocytoma of L cerebellar hemisphere	Marked AT
			R	140	85	120	—	—		Normal

TABLE (*continued*)

Patient	*Age (yr)*	*Sex*	*Arm*	*B1* (ms)*	*T1* (ms)*	*B1–B2* (ms)*	*SF**	*AI**	*Diagnosis*	*Exam*
III. Prolonged T1										
MP	67	F	R	70	140	160	—	Fair	8 d after probable embolic stroke characterized by AT of all limbs	Marked AT, brisk reflexes
GB	74	M	R	85	135	120	N	Fair	2 m after sudden onset of AT of all limbs	Moderate dysmetria, rebound, dysdiadochokinesia, AT
IV. Not significantly abnormal										
SH	55	M	L	85	45	75	N	Fair	CD for 1½ yr	Mild AT
JGr	66	M	R	95	90	105	—	—	2 y after embolic infarction of R cerebellar hemisphere	Mild AT, moderate dysdiadochokinesia, slight rebound, no dysmetria
LB	10	F	R	115	80	140	N	—	CD for 1½ yr	Mild dysmetria, AT, dysdiadochokinesia
			L	115	90	115	—	—		Same as R arm
V. Unanalysable										
CC	57	M	L	—	—	—	N	Poor	12 d after L cerebellar hemisphere infarction	Moderate AT, rebound, dysmetria
HW	60	M	R	—	—	—	—	Poor	Familial olivopontocerebellar atrophy for 20 yr	Moderate AT, overshoot and dysdiadochokinesia; no rebound
Normal average				80	60	70				
Normal range				60–105	30–85	40–150				

* In columns B, T1 and B1–B2 are listed the average times of these components for the FF task. Qualitative performances on the SF and AI tasks are also noted. See test for definitions.
AT: ataxic tremor. CD: cerebellar degeneration. SD: spinocerebellar degeneration. N: normal. A: abnormal.

RESULTS

1. FAST FLEXION All patients performed the FF task and, in general, the initial part of the EMG activity showed a pattern qualitatively similar to that of previously described normals (Hallett *et al.*, 1975). In the biceps there was an initial burst (B1), followed by a silent period (B1–B2), followed by a second burst (B2). The triceps activity (T1), to a fair approximation, occurred during the biceps silent period producing a 'triphasic' pattern: B1, T1, B2. The exact relationship of the end of B1 and the beginning of T1 is described by the parameter B1–T1, and the relationship between the end of T1 and the beginning of B2 by T1–B2. The normal limits for duration of B1, T1, and B1–B2 were sufficiently well defined to be considered standards against which patients could be judged. Eighteen of the 20 patients produced patterns that were interpretable in this scheme; parameters were measured in five to 15 trials, averaged and recorded in the Table. There were several kinds of abnormal performances on the FF task and the patients are grouped in the Table according to the type of abnormality. A component was considered abnormal if it were more than 10 ms greater than the normal range. No subject had a significant abnormality of B1–B2 or a duration of B1 or T1 less than normal.

The first abnormal group showed a prolongation of B1 without prolongation of T1 (Fig. 1). Four patients showed an absolute prolongation of B1 (compared with normal) and two showed a prolongation only by comparison with the non-ataxic opposite arm. Most of these patients had a tendency to overshoot both on clinical testing and during experimental trials. There was no clear correlation between the duration of B1 and the angular displacement of the elbow, and B1 was prolonged even in those trials when the line was matched correctly or undershot.

A second abnormal group showed prolonga-

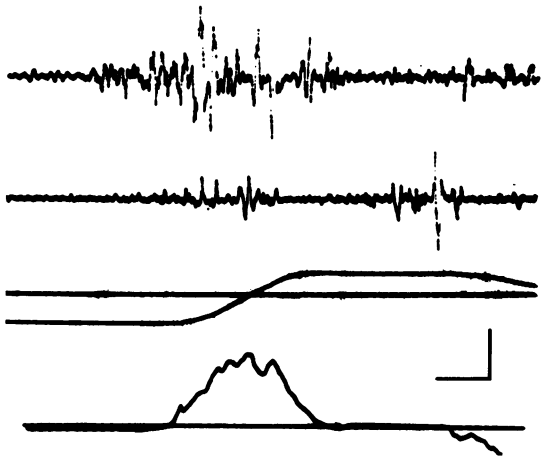

FIG. 1 *Patient H.S. EMG pattern during FF task demonstrating a prolonged B1. Traces are, from above downward, EMG of biceps, EMG of triceps, the experimenter's line with the step displacement which has to be matched, the position of the patient's arm, the velocity of the movement, and a marker line. The step displacement occurred 100 ms before the beginning of the photograph. Note overshoot of subject's line. Calibration: 100 ms; 200 μV.*

tion of both B1 and T1 (Fig. 2). There were seven patients in this group and, like the first group, they had a variety of types of 'cerebellar deficits'.

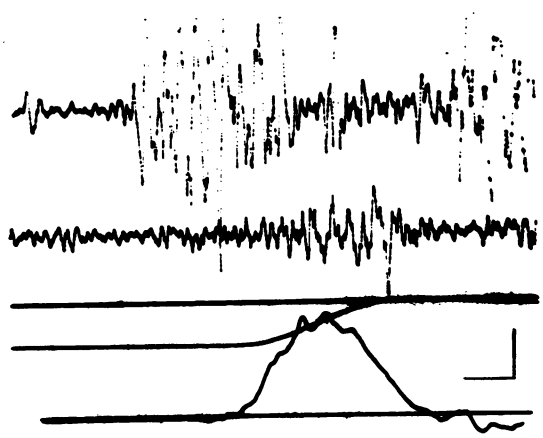

FIG. 2 *Patient J.G. EMG pattern during FF task demonstrating prolonged B1 and T1. Traces are the same as Fig. 1. The step displacement occurred 150 ms before the beginning of the photograph. Calibration: 50 ms; 200 μV.*

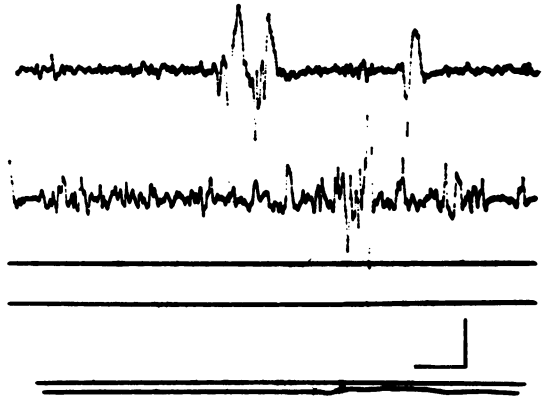

FIG. 3 *Patient G.B. EMG pattern during FF task demonstrating prolonged T1. Traces are the same as Fig. 1. The step displacement occurred 100 ms before the beginning of the photograph. Note the virtual absence of movement during the recorded 500 ms. Calibration: 50 ms; 200 μV.*

A third kind of abnormal performance is characterized by a prolongation of T1 without prolongation of B1 (Fig. 3). In this group, the initial effort for almost all moves was characterized by extreme hypometria—a failure to accelerate the limb. There were two patients with this abnormality and clinically they were similar. Both experienced the sudden onset of severe ataxia of all four limbs; it is presumed that they suffered from similar strokes, but the clinical-pathological correlation is uncertain.

Three patients, in whom the cerebellar deficit was mild, did not show an abnormality during the FF task. The minimal clinical deficit is not sufficient to explain the lack of abnormality, since others with a similar deficit did show abnormal patterns. Though the patterns were not significantly abnormal, one or more components may be prolonged compared with the unrecorded premorbid state. Such a notion is reinforced by those patients who do not show an absolute abnormality, but can be shown to be abnormal by virtue of comparison with the normal opposite limb.

Two patients were not analysable because they did not demonstrate a triphasic pattern. They had moderate ataxic tremors and, in both, a very long continuous burst of activity in biceps was

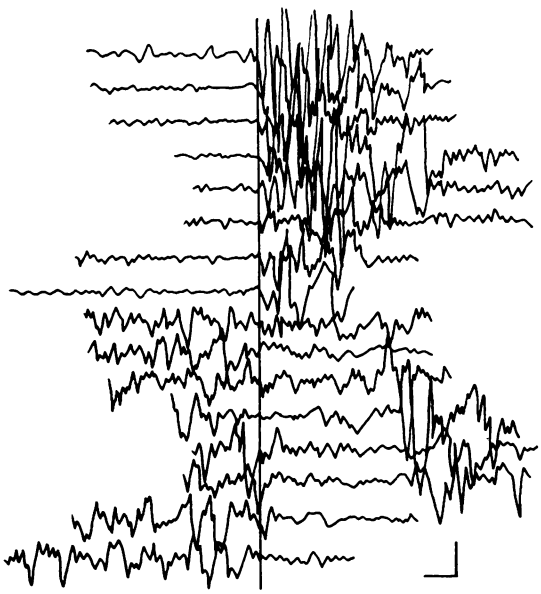

FIG. 4 *Patient S.H. Montage of eight successive trials during the AI task. Traces are aligned at the onset of biceps activity. Biceps EMG's are the first eight lines and the triceps EMGs are the second eight. The first biceps tracing and the first triceps tracing are from the same trial, and subsequent biceps and triceps tracings are related similarly. Note that triceps activity usually ceases the moment biceps begins or shortly thereafter, but never significantly before as it should normally do. Calibration: 20 ms; 100 μV.*

seen with less frequent irregular activity in triceps.

It should be observed that this analysis does not capture the full range of the abnormal movement produced by the patients attempting to do the FF task. Several patients, particularly during the practice phase, would extend rather than flex the arm in which case the 'antagonist' rather than the 'agonist' would be activated first. In the patients with severe ataxic tremor, not only would the arm move in the opposite direction, but movement might also occur at shoulder or other joints. This implies that muscles that would ordinarily fix but not move the limb were activated inappropriately.

2. SMOOTH FLEXION During the SF task 10 of 14 patients performed normally—that is, there was continuous EMG activity only in the agonist. The patients tested and their performances are noted in the Table. In the other four, abnormal EMG patterns were characterized by alternating bursts of activity in biceps and triceps.

3. ANTAGONIST INHIBITION Of 16 patients tested on the AI task (Table), only four performed normally insofar as triceps activity *always* stopped before the initiation of biceps activity. In performances graded 'fair', the activity in triceps would sometimes cease before biceps activity began, but at other times activity in the two muscles would overlap. In some of these patients it was clear (in a statistical sense) that activity in triceps was diminishing as B1 was beginning, but the cessation of triceps activity was significantly delayed (Fig. 4). In 'poor' performances, the triceps activity continued unabated during B1.

DISCUSSION

Electromyographic correlates of the disordered voluntary movement in patients with lesions of the cerebellum or cerebellar pathways might help define one or more elemental abnormalities and elucidate the normal physiology of the cerebellum. Altenburger (1930) had studied fast and smooth movements in much the same way as we have. For his patients with cerebellar symptoms, in an FF task, T1 was foreshortened or absent. We could not confirm his conclusions that patients with cerebellar deficits utilized the antagonist less than the normal subjects did. In this study, we have recorded two distinct abnormalities that may be elemental and may help explain some of the clinical phenomena.

During the FF task, 15 of the 18 analysable patients showed a prolongation of B1, T1, or both. We have previously summarized the evidence that the triphasic pattern during fast flexion is centrally programmed and that B1 and T1 are relatively uninfluenced by segmental input *via* the servo loop (Hallett *et al.*, 1975). Thus, with cerebellar lesions, one common abnormality during FF movements is a distortion of the initial part of the pre-programmed pattern, which implies that the deficit is in the suprasegmental signal, even before the programme gets to the spinal cord.

We would like to propose that this abnormality of B1 and T1 may partially underlie the

phenomenon of dysmetria. A dysmetric performance of the single movement required in the FF task can be characterized by excessive range (overshoot or hypermetria) or premature arrest (undershoot or hypometria). For two patients B1 was normal and T1 prolonged. One could well imagine that the triceps might halt the developing movement and this pattern would correlate with hypometria—this appears to be the case. For 13 patients B1 (and possibly T1) was prolonged. One could imagine that if a patient, in trying to make a movement of a certain length, generates a B1 longer than he normally would, there might be a tendency for hypermetria. Clinically, many movements were hypermetric, but in the performance of the FF task there was a mixture of hypermetric, hypometric, and accurate movements. There was no clear correlation between the duration of B1 and the length of the movement. Moreover, prolongation of B1 does not seem to be the normal physiological mechanism for making a longer movement (Dijkstra and Denier van der Gon, 1973; Hallett *et al.*, 1975). Thus, prolongation of B1 is not easily correlated with hypermetria. Although there might be a tendency for hypermetria with prolonged B1, patients can apparently compensate by adjusting other components of the movement (which may, in fact, be even more important than B1 in determining movement length). These uncertain compensatory mechanisms, however, must also be deranged with cerebellar deficits, since the movements in these patients remain dysmetric. Increasing ability to control these compensatory mechanisms may play a role in the amelioration of cerebellar symptoms. The abnormal centrally programmed 'B1, T1 package' in patients with cerebellar deficits sets up a predisposition to dysmetria which is realized when the compensatory mechanisms fail.

Dysdiadochokinesia is clumsiness in the performance of rapid alternating movements at a single joint. In the AI task an isometric contraction of the elbow extensors is followed by an isotonic contraction of the elbow flexors (B1). Thus an abnormality in the performance of the task—that is, switching activity from one muscle group to its antagonist—might be considered an elemental abnormality underlying dysdiadochokinesia. In the normal performance of this task, activity in triceps begins to decrease during the 50 ms before the beginning of biceps activity; it always ceases before that point (Hallett *et al.*, 1975). Twelve of 16 patients did not perform normally on this task—activity in triceps either ceased too late or not at all. This finding provides evidence for the concept that the cerebellum plays a role in the organization of the relationship between activity in agonists and antagonists in successive movements, a notion that has been presumed by most clinical neurologists writing about cerebellar function. Terzuolo and Viviani (1974), studying a rapid alternating movement, have made a similar qualitative observation.

This failure during the AI task of activity in a muscle to diminish or cease in an appropriate fashion before the beginning of activity in its antagonist may also underlie clinical phenomena other than dysdiadochokinesia. When a limb is flexed strongly against resistance and the resistance is suddenly released, the resultant movement is normally quickly checked. This often does not occur in patients with cerebellar deficits and would be called 'rebound'. Failure to reduce activity in the tonically active muscle might underlie this, a situation which has been demonstrated previously (Struppler and Schenck, 1958; Terzuolo and Viviani, 1974). Holmes had ascribed rebound to hypotonia, but it may come from failure or delay in the appropriate cessation of muscle activity. In fact, both the abnormalities described above (prolonged B1 and/or T1, and failure on A1 task) may be considered failures in termination of muscular activity.

In 10 of 14 patients the pattern of smooth flexion was normal, suggesting that this type of movement is better preserved with cerebellar lesions than is fast flexion. It has been suggested by Kornhuber (1971) that the cerebellum is primarily responsible for the organization and timing of rapid movements and the basal ganglia are responsible for smooth, ramp movements. DeLong and Strick (1974) have provided some evidence for this hypothesis in monkeys, showing that activity of cells in the putamen is particularly associated with smooth movements, whereas activity of cells in the cerebellum changes with both smooth and fast movements. Our findings are in agreement with these general principles.

It remains to be determined if the abnormalities discussed here are specific for cerebellar deficits. This will be important not only for a clear understanding of cerebellar physiology, but also for determining the usefulness of these abnormalities as diagnostic tools for the documentation of cerebellar deficits. In preliminary observations on patients with Parkinson's disease, we were unable to demonstrate either of the abnormalities described here on the FF and AI tasks.

It is not yet clear that all of the symptoms characterizing the disorders of voluntary movement seen with cerebellar deficits must occur together. For the partial 'cerebellar deficits' studied here, dysmetria as defined by an abnormality of FF can be present without dysdiadochokinesia as defined by an abnormality on AI, and conversely.

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