*Journal of Neurology, Neurosurgery, and Psychiatry* 1987;**50**:71–77

# Long latency EMG responses in hand and leg muscles: cerebellar disorders

H H FRIEDEMANN,* J NOTH,† H C DIENER,‡ M BACHER‡

*From the Departments of Neurology, University of Düsseldorf* and University of Tübingen,‡ Department of Neurology with Clinical Neurophysiology, Alfried Krupp Hospital, Essen,† Federal Republic of Germany*

SUMMARY Electromyographic responses to stretches of hand muscles (first dorsal interosseus) and leg muscles (triceps surae, tibialis anterior) were investigated in patients with cerebellar disorders of different locations. Stimuli consisted of short dorsiflexions of the index finger during background force and in tilting (toe up) of a movable platform on which the subject stood. The most important findings were increased long latency responses in upper and lower extremities. For hand muscles it was the late part of the long latency complex, which was increased. For leg muscles it was the long latency response in the anterior tibialis muscle, the antagonist of the stretched triceps surae. The medium latency response in the triceps surae was unaffected. Latencies of the early segmental reflexes and the long latency responses were normal except for cases with peripheral neuropathy (moderate increase in latency of all EMG responses) and diseases affecting both the peripheral nerves and the dorsal columns (for example Friedreich's ataxia). The latter leads to a pronounced delay of the short latency response and a massive delay of the long latency complex in the first dorsal interosseus and of the long latency response in the anterior tibialis muscle.

The occurrence of long latency electromyographic (EMG) responses after limb perturbations is well established in man,[1-3] but the pathway responsible for these responses is still debated.[4 5] Based on clinical evidence there are several reasons to assume a supraspinal origin, at least for responses recorded from distal hand muscles.[3 6] If so, the question arises, which supraspinal structures are engaged in the transmission of long latency responses. In monkeys precentral corticospinal neurons respond to limb perturbations with short latencies, which suggests a direct loop via the sensorimotor cortex.[7 8] Nevertheless, an additional cerebellar contribution to the later part of the long latency complex was considered by Lee and Tatton.[9] This assumption is supported by two results obtained in monkeys: (1) Sudden load changes can influence the activity of neurons in the dentate nucleus with a latency of only 30 ms.[10] (2) Cooling of the dentate nucleus depresses only the late responses of precentral neurons in area 4 to perturbations of the elbow. This late response may possibly be related to the M3 response in the EMG.[11] In patients with cerebellar lesions there are only single case reports about long latency reflexes in arm muscles[12 13] and in leg muscles.[14] The aim of this study was to investigate the influence of the cerebellum on long latency reflexes in man and to compare the responses in hand and leg muscles. Diener *et al*[15] described short and medium latency responses in the stretched triceps surae and a long latency response in the released anterior tibialis muscle to sudden tilts (toe up) of a movable platform. These responses were related to the M1, M2 and M3 responses observed in arm muscles,[2] but whether a strong analogy exists between arm and leg muscles in this respect is still an open question. This analogy is supported if congruent pathological alterations are detected in the corresponding EMG components of arm and leg muscles. This was tested in the present investigation. Some aspects of the results have been published earlier.[14 16]

Address for reprint requests: Prof Dr J Noth, Neurologische Klinik mit klinischer Neurophysiologie, Alfried Krupp Krankenhaus, Alfried-Krupp-Strasse 21, D-4300 Essen 1, FDR.

Received 5 September 1985 and in final revised form 13 February 1986.
Accepted 15 February 1986

## Material and methods

EMG responses were evoked in the first dorsal interosseus muscle (FDI) using short imposed stretches under otherwise isometric conditions. The general procedure has been described before[3] and will only be briefly mentioned here. Short triangular stretches (rise time 6 ms, transient time 20 ms) were applied to the index finger with the help of a lever, on which the subject exerted a constant force of 10% maximum. The lever was coupled to an electromagnetic vibrator

(Ling Dynamic System), which was controlled by a position and velocity feedback system. The extension of the index finger amounted to 1° dorsiflexion measured at the proximal joint. The low pass filtered force was displayed on an oscilloscope for visual control. The task was "not to react". The EMG activity of the first dorsal interosseus muscle was recorded by surface electrodes, filtered (corner frequencies 5 Hz and 1000 Hz), full wave rectified and averaged 64–128 times. Analysis time was 200 ms, the bin-width 0·2 ms.

EMG responses in leg muscles were evoked by tilting a movable platform, on which the patients stood, toe up around the ankle joint (ramp stimulus 50°/s, 4°). The EMG of the anterior tibialis (TA) and triceps surae (TS) muscles was recorded using bipolar surface electrodes and processed as above (for details see reference 15), analysis time being 400 ms. The results from eight platform tilts were averaged.

Latencies and durations of the EMG responses were measured by visual inspection with the help of an electronical cursor on the interactive display of a mini-computer. For quantification, the integrals of the EMG activity were evaluated in arbitrary units during the periods of interest. Means and standard deviations were calculated for the control population (24 age-matched normals for the hand muscles and 30 normals for the EMG responses in leg muscles). Upper limits of normality were defined as exceeding the mean $\pm 2$ times standard deviations for latencies and integrals.

Thirty nine patients with different disorders of the cerebellum were included in the study.

(1) Eight patients had lesions confined to the *cerebellar hemispheres* confirmed by computed tomography or by surgery. Three patients had a tumour, five ischaemic lesions restricted to the lateral cerebellum. In seven patients the lesion was unilateral, in one patient bilateral. Clinically, these patients suffered mainly from ataxia of the upper extremities, that is, dysmetria, intentional tremor and adiadochokinesis, mostly restricted to the affected side. Only mild disturbances of posture and eye movements were present. Three patients formerly exhibited ataxia of the upper extremities, but at the time of the investigation no ataxia was visible. None of the patients had difficulties to hold the force constant during the hand experiment.

(2) Four patients were afflicted with a lesion of the *lower vermis*, three of them with a tumour and one by hemorrhage from an angioblastoma. The vestibulocerebellar portion of the cerebellar cortex is mainly engaged in the modulation of eye movements and in the coordination of stance and gait. Correspondingly, the patients suffered from gait and stance ataxia. The coordination of the upper extremities was mostly undisturbed.

(3) Nine patients exhibited a late alcoholic atrophy predominantly of the *anterior lobe* with ataxia of stance and gait. As in the patients with lesions of the lower vermis almost no ataxia of the upper extremities was visible. In five out of the nine patients there was an additional polyneuropathy.

(4) Eleven patients had *diffuse lesions* of the cerebellum. Four of these patients suffered from an olivo-pontocerebellar atrophy, a disease which affects multiple neural systems including the dorsal columns, the cerebellum, the olivary and pontine nuclei, the substantia nigra, the thalamus and the cortex. Again ataxia of stance and gait was prominent, but also modest signs of ataxia of the lower extremities were present. The coordination of the upper extremities was normal. Four patients had diffuse atrophies of unknown origin and three toxic atrophies. These seven patients were clinically affected with mild ataxia both of stance and gait and of arm.

(5) Seven patients suffered from *Friedreich's ataxia*, but were still able to walk without support. The disorder involves degeneration of the posterior columns, the spinocerebellar and the corticospinal tracts. The tendon jerks were absent in all patients and the deep sensibility was impaired. Most prominent was ataxia of stance and gait.

## Results

### *Normals*

Typical EMG responses of hand and leg muscles are depicted in fig 1 with grand averages in the upper traces and individual examples in the lower traces. Imposed displacements of the index finger often evoke three separable EMG responses in the stretched muscle. According to Lee and Tatton,[2] the responses were labelled M1–M3. In our paradigm, the first (M1) response appeared with a mean latency of $33{\cdot}3 \pm 3{\cdot}7$ ms. The mean duration was $16{\cdot}4 \pm 4{\cdot}4$ ms, the mean integral $4{\cdot}3 \pm 2{\cdot}5$ arbitrary units. In hand muscles it was often impossible to separate clearly M2 from M3, therefore the M2/3 complex was taken as a whole. The latency of this M2/3 complex was $60{\cdot}2 \pm 6{\cdot}9$ ms, the integral $7{\cdot}9 \pm 4{\cdot}0$ arbitrary units.

Platform movement toe up evokes short and medium latency responses in the stretched triceps surae (TS) and long latency responses in the antagonistic anterior tibialis (TA) muscle.[15] The latency of the short latency response in the triceps surae was $42{\cdot}9 \pm 3{\cdot}4$ ms, the latency of the ML response $92{\cdot}3 \pm 7{\cdot}6$ ms. The durations were $24{\cdot}1 \pm 5{\cdot}1$ ms and $24{\cdot}2 \pm 6{\cdot}5$ ms and the integrals $12{\cdot}0 \pm 4{\cdot}2$ and $12{\cdot}5 \pm 5{\cdot}2$ arbitrary units, respectively. As the medium latency responses are absent in about 25% of normals a missing medium latency response in the triceps surae muscle thus cannot be regarded as pathological. The long latency responses in the antagonistic anterior tibialis muscle appeared with a mean latency of $113{\cdot}6 \pm 18{\cdot}6$ ms, duration of $76{\cdot}0 \pm 19{\cdot}0$ ms and integral of $40{\cdot}2 \pm 10{\cdot}4$ arbitrary units.

### *Lesions of the cerebellar hemispheres*

The general finding was a relatively small M1 component and a pronounced M2/3 complex in hand muscles. Typical examples of EMG responses are depicted in fig 2. In only one patient with an additional lesion of the brainstem the M2/3 complex was smaller than the M1 component on both sides: In another patient the M2/3 complex was missing on one side. The integral of the whole M2/3 complex was enlarged in more than 50% of the patients. The latency of the M2/3 complex was normal in patients with pure cerebellar lesions (55·6 ms, $n = 4$) and was

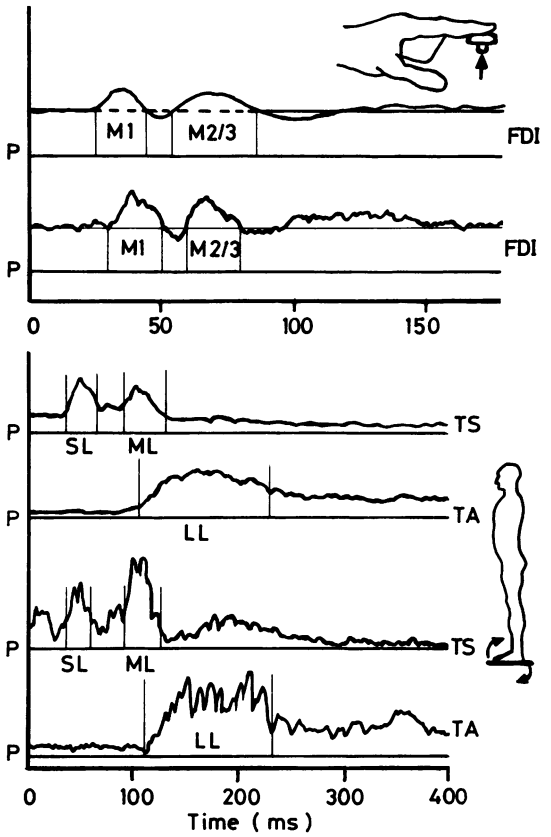


Fig 1 *Averaged rectified EMG responses after stretch of the first dorsal interosseus muscle (FDI) and after platform tilt toe up. Grand average from 20 normals and an original recording in the upper part show the typical pattern with the spinal component (M1) and the long latency response (M2/3). Grand average and original recording in the lower part show the typical EMG pattern with the short (SL) and medium (ML) response in the stretched triceps surae muscle (TS) and the long latency response (LL) in the anterior tibialis muscle (TA). Vertical lines indicate beginning and ending of EMG responses. P, preactivity.*

slightly longer in patients, who showed signs of additional brainstem involvement (63·6 ms, n = 4). Remarkable is the absence of a lateralisation of these pathological differences in the EMG responses in unilateral hemispheric lesions.

The latencies of all three EMG responses in leg muscles were normal except for one patient with an additional polyneuropathy. Integrals of these EMG responses were in general normal. Only two patients showed enlarged long latency responses in the anterior tibial muscle.

*Lesion of the lower vermis*

The spinal (M1) response in hand muscles was normal regarding latency (35·4 ms) and duration (13·7 ms). As in the patients with hemispheric lesions the latency of the M2/3 complex was normal, but the integral was enlarged in three out of the four patients. This was an astonishing result, since clinically no signs of an ataxia of the upper extremities were present.

The leg muscles latencies and integrals of short latency and medium latency responses were nearly always normal in the triceps surae muscle, but the long latency response was enlarged in two of the four patients.

*Late cortical atrophy of the anterior lobe*

In the nine patients investigated, the M2/3 complex in hand muscles was enlarged in four patients and the long latency response in the anterior tibial muscle was enlarged in six patients (figs 3, 5). In the five patients with an additional polyneuropathy all components were moderately delayed if visible. The size of the medium latency response of the triceps surae muscle was in the normal range. In the remaining four patients without polyneuropathy the latencies of the M1 and M2/3 components were normal, as were the latencies of the short latency, medium latency and long latency responses in leg muscles.

*Diffuse cerebellar lesions*

This group of patients (n = 11) showed no consistent results. This is not surprising since different central systems are involved in the pathological process. The only notable finding was an increase of the M1 integral (first dorsal interosseus muscle) in the four patients with olivo-ponto-cerebellar atrophy, which corresponds to the pyramidal lesion in this degenerative disease.

*Friedreich's ataxia*

The most striking finding in the EMG responses of the hand muscles was a massive delay or absence of the M2/3 complex in nearly all patients (fig 4). With one exception the spinal M1 component was clearly distinguishable in hand muscles, but the latency was delayed in 50% of the patients due to the associated neuropathy. The short latency and medium latency responses were mostly abolished in leg muscles. In the anterior tibial muscle a prolonged and slowly rising increase in the EMG activity was observed (fig 4).

Figure 5 shows the frequency of pathological results regarding the integrals of the different EMG responses. For lesions of the cerebellar hemispheres and of the lower vermis the most frequent pathological result was an enhanced M2/3 response of the first dorsal interosseus muscle. On the other hand, patients with lesions of the anterior lobe showed most often

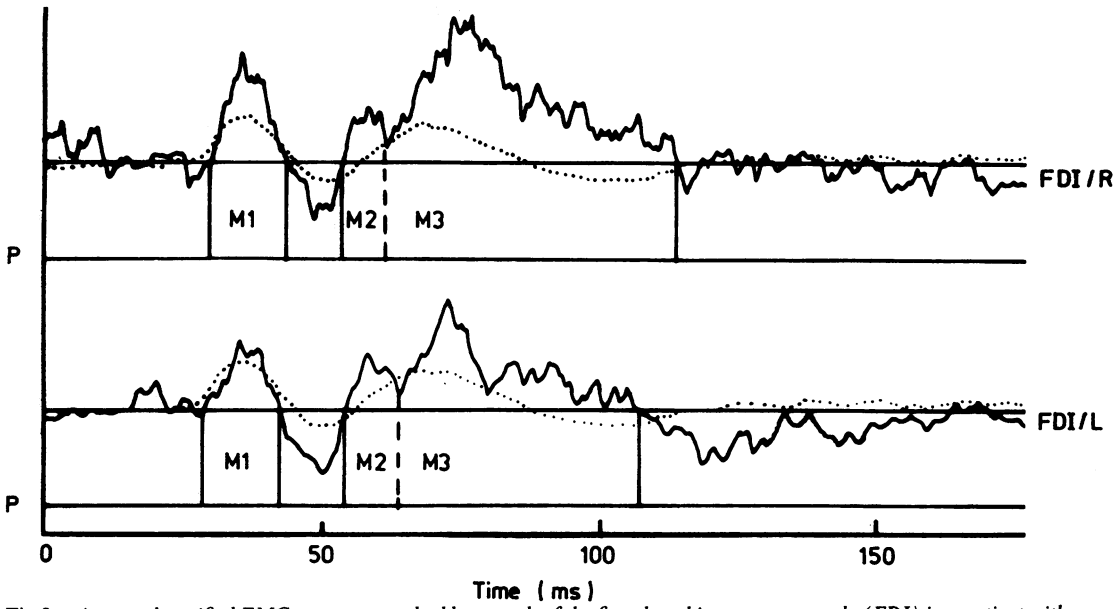


Fig 2 *Averaged rectified EMG responses evoked by stretch of the first dorsal interosseus muscle (FDI) in a patient with an ischaemic lesion of the right cerebellar hemisphere. The dotted line corresponds to the grand average of the normal population and indicates the normal latencies. Note the increased M3 response in the right (R) and left (L) finger muscle.*

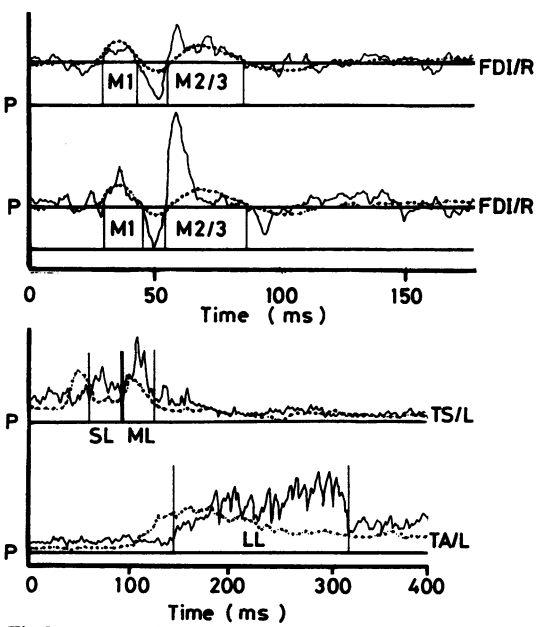


Fig 3 *Averaged rectified EMG responses evoked by stretch of the first dorsal interosseus muscle (FDI) and by platform tilt toe up in leg muscles (TS, TA) in a patient with anterior lobe atrophy due to chronic alcoholism. Note the normal M1 and M2/3 responses on the right (R), the increased M2 response on the left (L). SL and ML are normal, but the integral and duration of the LL response is markedly increased.*

enhanced long latency responses of the anterior tibialis muscle. This result corresponds to the different functional role of different parts of the cerebellum.[17]

## Discussion

*Comparison between EMG responses of hand and leg muscles.*

The early spinal components (M1 of the first dorsal interosseus muscle, short latency of the triceps surae muscle) showed no major alterations in patients with cerebellar lesions regarding size and latency, as long as the peripheral nerves were intact. This finding indicates the regular excitability of spinal alpha-motoneurons in these patients. This result corresponds to the normal tendon jerks in subacute and chronic cerebellar diseases. Patients with recent cerebellar lesions may exhibit hypotonia and reduced tendon jerks.[17] Cases of this kind were not included in the study.

The question arises whether M2 responses in hand and finger muscles and medium latency responses in leg muscles are analogous. At least for muscles controlling the fingers, a supraspinal pathway can be assumed,[3 6] (for review see reference 4). The evidence for a supraspinal pathway being involved in the generation of the medium latency response in the triceps surae muscle is much weaker.[18] The lack of an increase in latency of the medium latency response in patients with multiple sclerosis,[19] who exhibit a clear

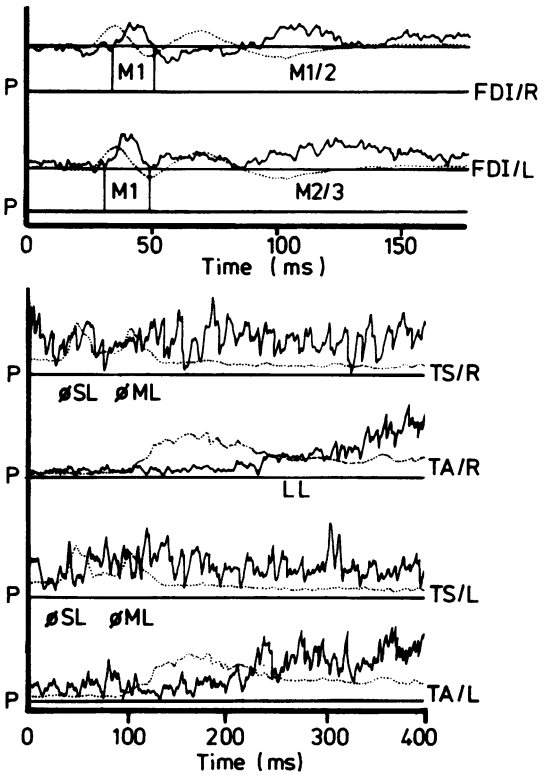


Fig 4 *Averaged rectified EMG responses in a patient with Friedreich's ataxia and abolished tendon jerks. The recording from the first dorsal interosseus muscle (FDI) shows a small delay of the M1 response, and no consistent M2/3 complex. SL and ML in the triceps surae muscle (TS) are missing. In the anterior tibial muscle (TA) only a slow increase of the muscle activity is detectable.*

increase in latency of the long latency component in the anterior tibialis muscle, makes a supraspinal origin of medium latency rather unlikely.

In contrast, the late part of the long latency response (M3) of the first dorsal interosseus muscle and the long latency response of leg muscles were enlarged in patients with cerebellar lesions. In healthy subjects, this long latency response appears at a constant latency in the anterior tibialis muscle which is shortened during a toe up tilt of the platform. It is therefore neither a voluntary response nor a supraspinal stretch reflex.

Nashner[20] who investigated these reflexes in leg muscles under varying functional conditions (linear displacement of the platform versus angular displacement) found that normal subjects were able to alter quickly their motor strategy following new demands. Thereby, the posture-stabilising or destabilising response at about 120 ms (functional stretch reflex), was facilitated or adapted during the test sequence, according to the specific functional requirements. The relatively short and stable latency of this response excluded that "voluntary" reactions were responsible. The observation that patients with cerebellar lesions lost their ability to adapt "long latency reflex gain following changes in the stance task" indicates that the cerebellum is engaged in the control of these EMG responses.[20]

However, the latency of Nashner's functional stretch reflex in the stretched triceps surae muscle was 100–120 ms and that of the posture-stabilising response in the anterior tibialis muscle was 180 ms.[21] The latency of Nashner's functional stretch reflex compares well with the mean latency of 113 ms observed in our paradigm for the posture-stabilising TA response. The reason for the discrepancy of latencies of the stabilising tibialis anterior response remains unclear. We found normal responses in our patients with cerebellar lesions, which argues against an analogy of our medium latency response with the functional stretch reflex response observed by Nashner.[20] The fact that Nashner did not observe the medium latency response in the stretched muscle may be due to the low rotational velocity of 8°/s imposed in his experiments.[20] A positive correlation between platform acceleration and integrated activity of the response in the triceps surae supports this assumption.[18 22]

In hand muscles, medium and long latency responses were usually not separable in our motor paradigm. Some subjects, however, exhibited reproducible M3 responses following the M2 response with a delay of about 12 to 15 ms,[3] the steep increase of which excludes a "voluntary" reaction. Patients with lesions of the cerebellar hemispheres showed enhanced responses just at these latencies. Thus, in analogy to the leg muscles, it is probably a pre-existing response, which is enhanced in patients with hemispheric lesions. The absence of a distinct long latency response in the first dorsal interosseus muscle of most of the healthy subjects may be due to the particular motor task selected in our study. In contrast to the platform tilt, there is no functional demand to respond to the short finger displacement with an increased force output. Longer lasting torque pulses, on the other hand, evoked reproducible EMG responses with long latencies in wrist flexors[9] and in the long flexor of the thumb.[23]

*Consideration of pathways being involved*

Our result indicates that the cerebellum modulates the M3 long latency motor response which can be triggered by peripheral receptors and which, according to functional demands, can appear in muscles which are stretched or released by the initial disturbance. For

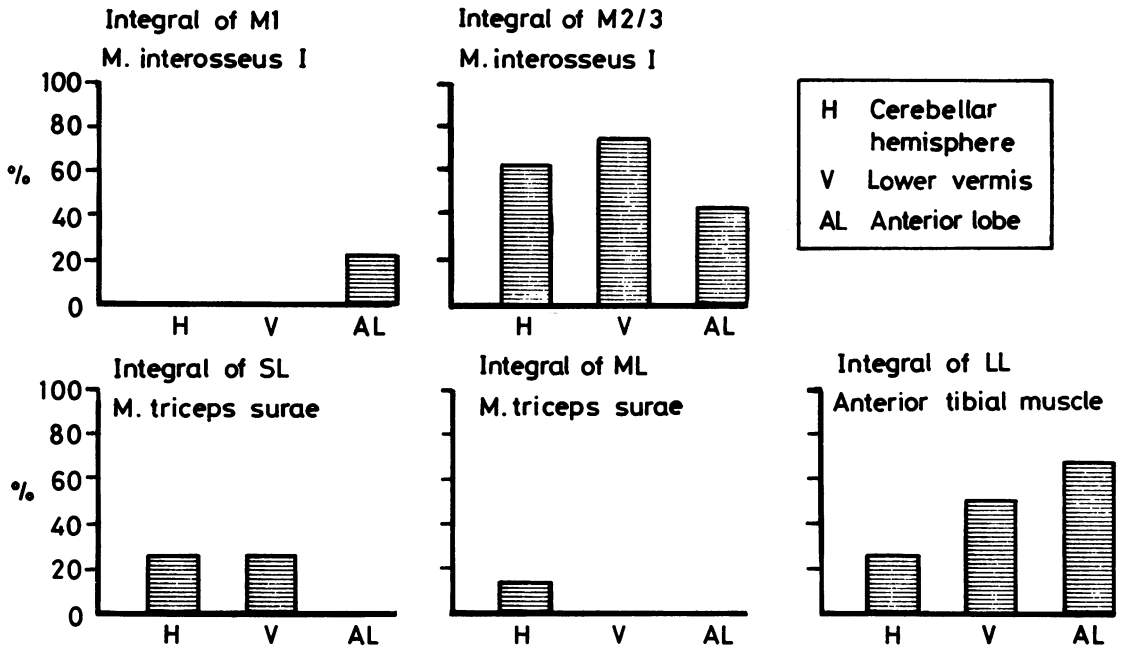


Fig 5 *Frequency of pathological results (exceeding the upper limit of normality greater than mean ±2 SD) in the four groups of cerebellar patients. Note the high frequency of pathological M2/3 responses (FDI) in vermal and hemispheric lesions and the increased LL response (TA) in anterior lobe atrophy.*

hand muscles the latency of this response exceeds that of the presumed transcortical reflex by only 12–15 ms for hand muscles, which provides enough time for a transcerebellar loop. A transcerebellar loop, however, does not necessarily implicate a transmission via the cerebellar cortex in patients with cerebellar disorders. The most vulnerable cells of the cerebellum are the Purkinje cells,[24] and these cells are the only output cells. Any response exclusively transmitted via the cortex should thus be diminished in case of a predominant Purkinje cell damage. The cerebellar nuclei, on the other hand, could be responsible for the exaggerated responses if deprived of the inhibitory influence from the cerebellar cortex. The observations in our patients that large cerebellar lesions, extending to midline structures, were accompanied by a loss of the M2/M3 responses supports this idea.

*Clinical implications*

From a clinical point of view, the recording of long latency reflexes may help to detect subclinical disturbances of the motor system as it has already shown for persons at risk of Huntington's disease.[3] In patients with late alcoholic atrophy the investigation of these reflexes is already a useful tool for therapeutic control, since the long latency responses of leg muscles become smaller, if the alcohol abuse is stopped, and become greater, if the abuse is carried on.[25]

The authors are grateful to Professor Dr J Dichgans for critical reading of the manuscript.

This work was supported by the Deutsche Forschungsgemeinschaft SFB 200, Di 278/1–2.

**References**

1 Marsden CD, Merton PA, Morton HB. The sensory mechanism of servo action in human muscle. *J Physiol (Lond)* 1977;**265**:521–35.
2 Lee RG, Tatton WG. Long loop reflexes in man: clinical applications. In: Desmedt JE, ed. *Progress in Clinical Neurophysiology, vol 4*. Basel: Karger, 1978:320–34.
3 Noth J, Podoll K, Friedemann H-H. Long loop reflexes in small hand muscles studied in normal subjects and in patients with Huntington's disease. *Brain* 1985; **108**:65–80.
4 Wiesendanger M, Miles TS. Ascending pathway of low-threshold muscle afferents to the cerebral cortex and its possible role in motor control. *Physiol Rev* 1982;**62**:1234–70.
5 Matthews PBC. Evidence from the use of vibration that

the human long-latency stretch reflex depends upon spindle secondary afferents. *J Physiol (Lond)* 1984;**348**:383–415.
6 Marsden CD, Merton PA, Morton HB, Adam J. The effect of lesions of the sensorimotor cortex and the capsular pathways on servo responses from the human long thumb flexor. *Brain* 1977;**100**:503–26.
7 Evarts EV, Fromm C. Sensory responses in motor cortex neurons during precise motor control. *Neurosci Letr* 1977;**5**:26–72.
8 Cheney PD, Fetz EE. Corticomotoneuronal cells contribute to long-latency stretch reflexes in the rhesus monkey. *J Physiol (Lond)* 1984;**349**:249–72.
9 Lee RG, Tatton WG. Motor responses to sudden limb displacements in primates with specific CNS lesions and in human patients with motor system disorders. *Can J Neurol Sci* 1975;**2**:285–93.
10 Strick PL. Cerebellar involvement in 'volotional' muscle responses to load changes. In: Desmedt JE, ed. *Progress in Clinical Neurophysiology, vol. 4*. Basel: Karger, 1978:85–93.
11 Meyer-Lohmann J, Conrad B, Matsunami K, Brooks VB. Effects of dentate cooling on precentral unit activity following torque pulse injections into elbow movements. *Brain Res* 1975;**94**:237–51.
12 Marsden CD, Merton PA, Morton HB, Hallet M, Adam J, Rushton DN. Disorders of movement in cerebellar disease in man. In: Rose FC, ed. *Physiological Aspects of Clinical Neurology*. Oxford, Blackwell, 1977:179–99.
13 MacKay WA, Murphy JT. Cerebellar influence on proprioceptive control loops. In: Massion J, Sasaki K, eds. *Cerebro-cerebellar Interactions*. Amsterdam, Elsevier, 1979:141–62.
14 Diener HC, Dichgans J, Bacher M, Guschlbauer B. Characteristic alterations of long loop "reflexes" in patients with Friedreich's ataxia and late atrophy of the anterior cerebellar lobe. *J Neurol Neurosurg Psychiatry* 1984;**47**:679–85.
15 Diener HC, Bootz F, Dichgans J, Bruzek W. Variability of postural "reflexes" in humans. *Exp Brain Res* 1983;**52**:423–8.
16 Friedemann H-H, Diener HC, Matthews HR, Noth J. Enhanced long latency reflexes in patients with lesions of the cerebellar hemispheres. *Electroencephalogr Clin Neurophysiol* 1983;**56**:81.
17 Dichgans J. Clinical symptoms of cerebellar dysfunction and their topodiagnostical significance. *Hum Neurobiol* 1984;**2**:269–79.
18 Berardelli A, Hallett M, Kaufmann C, Fine E, Berenberg W, Simon SR. Stretch reflexes of triceps surae in normal man. *J Neurol Neurosurg Psychiatry* 1982; **45**:513–25.
19 Diener HC, Dichgans J, Hülser P-J, Buettner U-W, Bacher M, Guschlbauer B. The significance of long loop "reflexes" for the diagnosis of multiple sclerosis. *Electroencephalogr Clin Neurophysiol* 1984;**57**:336–42.
20 Nashner LM. Adapting reflexes controlling the human posture. *Exp Brain Res* 1976;**26**:59–72.
21 Diener HC, Dichgans J, Bootz F, Bacher M. Early stabilization of human posture after a sudden disturbance: Influence of rate and amplitude of displacement. *Exp Brain Res* 1984;**56**:126–34.
22 Nashner LM, Grimm RJ. Analysis of multiloop dyscontrols in standing cerebellar patients. In: Desmedt JE, ed. *Progress in Clinical Neurophysiology, vol. 4*. Basel: Karger, 1978:300–19.
23 Marsden CD, Merton PA, Morton HB. Behaviour of short and long latency components of the stretch reflex in human muscle. *J Physiol (Lond)* 1975;**246**: 43P–44P.
24 Adams RD. Nutritional cerebellar degeneration. In: Vinken PJ, Bruyn GW (eds.) *Metabolic and Deficiency Diseases of the Nervous System, part II (Handbook of Clinical Neurology, vol. 28)*, Amsterdam, Elsevier, 1976:271–83.
25 Diener HC, Dichgans J, Bacher M, Guschlbauer B. Improvement of ataxia in late cortical cerebellar atrophy through alcoholic abstinence. *J Neurol* 1984; **231**:258–62.