EMG responses in leg muscles to postural perturbations in Huntington’s disease

J Huttunen, V Hömberg

Abstract
This paper compares leg muscle electromyogram (EMG) responses to sudden toe-up tilts of a moveable platform in patients with Huntington’s disease (HD), clinically normal offspring at risk of developing HD (HD risks) and healthy controls. The EMG pattern in standing subjects and patients consisted of short- and middle-latency responses (SL and ML) in the stretched triceps surae muscles and long-latency responses (LL) in the shortened tibialis anterior muscles. The SL response could be further divided into two distinct sub-components termed SL1 and SL2. An ML response was identified in only 50% of normal subjects and patients. HD patients differed from normal subjects by showing delayed onset latencies and prolonged durations for the LL response, and smaller amplitudes for the ML response. The subjects at risk also showed diminished ML amplitudes and prolonged LL durations, but normal LL onset latencies. In the sitting condition, the EMG responses of the HD patients and of the HD risks did not differ from those of controls; in all groups SL1 was reduced, and delayed, SL2 slightly enhanced, while ML and LL were absent. Because both afferent and efferent conduction times are normal in HD, the delayed LL onset reflects abnormal supraspinal organisation of postural control in HD, and indicates that basal ganglia may have a modulatory effect on the LL responses. The normal EMG responses in the sitting patients suggest appropriate regulation of these responses according to postural set in HD.

In subjects standing on a moveable platform, a sudden toe-up tilt evokes short- and middle-latency reflexes (SL and ML, respectively) in the stretched triceps surae (TS), and a longer latency response (LL) in the shortened tibialis anterior (TA) muscle. SL and ML have onset latencies at about 40ms and 90ms, respectively, and LL usually starts at about 130ms in healthy subjects. While SL and ML tend to further destabilise the subject’s posture, the LL response prevents the subject from falling backwards.

The latency of the SL response suggests that it corresponds to a monosynaptic myotatic or an oligosynaptic segmental reflex. The longer latency ML and LL components are assumed to be mediated via longer pathways. The ML response is enhanced in Parkinson’s disease (PD), suggesting that its amplitude is modulated by basal ganglia activity. The LL response is delayed in upper motor neuron disease of spinal or hemispheric origin, which has led to the hypothesis of a transcortical loop mediating this response. However, duration and amplitude of the LL response are also changed in patients with lesions of the cerebellum, which therefore appears to modulate the LL responses, as well. Since the LL responses are of normal latency and amplitude in Parkinson’s disease (PD), there is as yet no evidence for involvement of the basal ganglia in the control of these late postural adjustments.

The pathology of Parkinson’s disease is characterised by degeneration of the dopaminergic input to the neostriatum. By contrast, in HD the intrinsic neurons of caudate, putamen and pallidum undergo degeneration. The most conspicuous “positive” sign of HD, is choreatic hyperkinesia, consisting of fast and irregular movements, in contrast to the regular tremor of PD. Detailed quantitative studies of the “negative” symptoms have shown, however, that similar slowing of voluntary movement is present in both degenerative diseases of the basal ganglia. In the upper extremities long-latency reflexes may be enhanced in PD, but are absent in HD. Although postural abnormalities are common in the clinical picture of HD, no information is available about stance regulating reflexes in the lower extremities of HD patients.

The aim of this study was to analyse whether abnormalities in postural EMG responses are present in HD. The EMG responses were also recorded with the patients sitting, to discover whether the responses are appropriately regulated according to different postural sets in HD.

Methods
Twenty seven patients with definite HD (13 females, 14 males, mean age 41, range 27 to 63) were studied. Demographic data of the patients are shown in table 1. The extent of choreatic movements was assessed with a clinical score ranging from 0–3, and the overall intellectual, social and motor impairment was evaluated with a disability score ranging from 0–3. The latter is a modification of the score used by Shoulson and Fahn. Fourteen of the patients received sulpiride or phenothiazines, two were
Table 1  Demographic data of HD patients

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on tiapride and two on the reserpine agonist tetrabenazine alone. Nine received no medication.

A control group of 26 subjects was recruited from the hospital staff. These subjects had no previous experience as subjects in neurophysiological studies and no history of neurological disease. The group consisted of 13 males and females with a mean age of 38 years (range 20 to 54).

Recordings were also made in 18 first-order offspring of HD patients without clinical signs or symptoms, having a 50% risk of being gene carriers of HD (HD risks). This group consisted of 10 females and 8 males with a mean age of 32 years (range 20 to 50).

The subjects were standing on a movable platform with their heels separated by 4 cm and the axes of their feet positioned at an angle of 30 degrees. The amplitude of the toe-up tilt was 4° and the velocity 50°/s. Electromyographic (EMG) responses were recorded with bipolar surface electrodes placed over the lateral gastrocnemius (triceps surae) and tibialis anterior muscles bilaterally. The EMG signals were filtered with a bandpass flat from 1-6 Hz to 1 kHz, digitised at 2 kHz, and full-wave rectified. Eight artefact free runs were averaged for each subject.

During the recordings the subjects were instructed to stand as still as possible and to fixate on a point 1-5 m in front of them. The procedure was explained to the patients to minimise anxiety and choreatic movements. None of the patients showed any clear choreatic movements on the lower extremities during the reflex recordings. As EMG responses could be analysed after each trial all the trials which included bursts of involuntary EMG activity were rejected from the analysis and additional artefact free trials were added. This rarely occurred and hardly ever more than two trials out of a series of 10 had to be rejected for this reason. After two or three familiarising trials, measurement runs were performed at about 15 to 45 s intervals without warning the subject beforehand. Data collection was performed until eight artefact free trials had been recorded. The procedure was then repeated with the subject sitting on a chair, instructed to relax and not to respond in any way to the platform tilts. The feet were positioned as in the standing condition.

Latencies, peak amplitudes and areas of rectified EMG responses were measured with an interactive cursor display on a microcomputer. The onset of each reflex component was defined as the first rising face of EMG activity leading to a clearly defined reflex EMG activity peak. In cases where the definition of a particular reflex component appeared to be too ambiguous due to background noise the recording procedure was repeated over another eight artefact free trials. The area value of a given component was defined as the integral of amplitudes over time for the whole duration of the deflection before EMG activity reached baseline level again. This measure was found to be highly correlated with peak amplitude and duration. Stimulus onset was defined as the moment of first observable deviation of the platform position from horizontal, as recorded with a potentiometer.

As no consistent differences were observed between left and right legs, average values from the two sides were used in statistical comparisons. Significance levels between groups were computed using two-tailed Student's t test for uncorrelated means, while for within-group comparisons between standing and sitting conditions the t test for correlated means was used. Pearson product-moment correlation for variable pairs was used to examine the relations between reflex parameters, demographic and clinical data of the patients.

Static posturography was performed in every patient and control subject before collecting reflex data with the platform. The platform system for static recordings is equipped with four strain-gauge force transducers positioned in every corner of the platform. From the readout of these four transducers the sway path of centre of foot pressure was worked out. To
EMG responses in leg muscles to postural perturbations in Huntington’s disease

Figure 1  Rectified and averaged EMG responses in left tibialis anterior (TA) and triceps surae (TS) muscles to sudden dorsiflexion displacements of ankle in two normal subjects. The tick marks on the axes are separated by 100ms. Bottom trace shows the stimulus, which begins at 100ms after sweep onset. Note different y-axis scales for different curves.

obtain sway path measurements patients and subjects were instructed to stand as still as possible fixating a point 1-5m in front of them similar to the reflex recording condition. Sway path data were collected over 20s and the total length of the sway path centre of foot pressure was determined. The dimension of this measure is cm/sec after dividing the entire sway path length through the recording period of 20s.

In 25 of the HD patients magnetic cortical stimulation of motor cortex was performed to estimate the conduction time from cortex to leg muscles. Using a Cadwell MES 10 stimulator magnetic pulses (duration 70 us, intensity 2 T) were delivered to the vertex region. Surface EMG responses were recorded from the abductor hallucis muscles bilaterally.

Results
Sway path measurements
The mean (SE) length of sway path for 20s for standing as still as possible in normals was 1.32 (0.85) cm/s. In HD risk patients it was slightly lower (0.82 (0.19) cm/s). In HD patients sway path length was significantly longer with 3.40 (2.87) cm/s than in normals and HD risks indicating more postural instability in the HD patients. T test comparisons for uncorrelated means with unequal variances showed a significant difference only between normals and HD patients (t = 3.59, df = 34; p < 0.01).

EMG responses in normals
Examples of EMG responses in two normal subjects are illustrated in fig 1. In the standing condition, slight EMG activity was always seen in the triceps surae before stimulus onset, reflecting the tonic contraction of this muscle in standing subjects.16 In tibialis anterior, no such background activity was seen. The SL reflex in triceps surae started on the average at 34ms and showed only small interindividual variability (SD) 3.4ms. In the majority of the subjects SL consisted of two distinct peaks, which are clearly visible in the two subjects illustrated in fig 1. Because of this consistent double-peak appearance, SL was divided into two subcomponents labelled as SL1 and SL2. The onset latency for SL2 was defined as the relative minimum of rectified EMG between the 2 peaks. Its mean value was 55ms, also with small interindividual variability (SD) 3.6ms. The peak amplitudes for SL1 and SL2 were highly variable from one subject to another, with mean values (SD) of 117 (79) μV and 81

Figure 2  EMG responses to ankle displacements in two HD patients. Details as in fig 1.
levels significance standing

Figure 3 Mean SL1 and SL2 latencies, amplitudes and areas in normals, HD risks and HD patients in standing and sitting conditions. The bars denote SEM. Statistical significance levels refer to comparison between sitting and standing conditions: ** = p < 0.01, *** = p < 0.001.

EMG responses in HD patients and persons at risk

In the standing condition, HD patients and persons at risk showed background EMG patterns similar to those of controls, that is, tonic activation of triceps surae but no activation of tibialis anterior. Also, SL1 and SL2 latencies and amplitudes were comparable to those of the normal subjects (fig 2), there being no statistically significant differences in any of the SL1 or SL2 parameters between the three groups (fig 3). Neither did we observe any individual patients, whose SL1 or SL2 amplitudes would have been outside the normal range of variability.

In both HD patient and HD risk groups, the frequency of occurrence of a clearly discernible ML response was similar to that found in normal subjects: an ML response in either leg was identified in nine HD risks (50%) and 13 HD patients (48%). However, statistical comparison of those patients, risks and normals with identifiable ML responses showed significantly reduced ML amplitudes for HD patients and HD risks (fig 4). The mean (SD) ML amplitudes were 209 (51) μV and 94 (11) μV in the HD risk and HD patient groups, respectively, did not differ from the mean latency for normal subjects.

Both of the HD patients in fig 2 show clearly delayed LL onset latencies when compared with the controls in fig 1. The mean (SD) LL onset latency in the HD patient group was 139 (26) ms, being significantly higher than the value for the normals (fig 5). In contrast, the mean LL latency for the HD risk group, 115 (18) ms, did not differ from that of normals. On an individual basis, nine HD patients (30%) showed LL latency values exceeding the mean of the normal group exceeding the confidence limit, that is, 142 ms. Only one person at risk had a clearly delayed LL onset at 165 ms.

The mean (SD) LL duration in HD patients was 209 (51) ms, being significantly higher than the corresponding value for normals (fig 5). In 13 (48%) individual patients LL duration exceeded the confidence interval (217 ms) for the normal group. The mean (SD) LL duration

Figure 4 Mean ML latencies, amplitudes and areas in the normal group, HD risks and HD patients in the standing condition. p-values refer to comparisons with the normal group. NS = non significant. Bars denote SEM.
Figure 5 Means of latencies, amplitudes, areas and durations of LL responses in normals, HD risks and HD patients in the standing condition. Details as in fig 4.

Table 2 Correlation coefficients between LL parameters and demographic variables in HD patients

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Discussion

The principal finding of this study shows that LL responses in tibialis anterior, stabilising the upright posture after a sudden platform perturbation, start later and last longer in Huntington’s disease compared with the normal group. The delayed LL onset is largely a group effect, but may be sufficient to distinguish individual cases from normal subjects when clearly delayed. In first-order offspring of HD patients, being at 50%, risk of being gene carriers for the disease, LL onset latency appeared normal in 17 out of 18 cases, indicating that the LL responses do not provide a useful test to detect presymptomatic HD patients.

The ML response in TS was present in only about 50% of normals, risk persons and HD patients. Therefore, although showing reduced amplitudes in HD patients and in HD risks, the ML response does not provide useful diagnostic information in individual cases.

Normal sequence of EMG responses in standing and sitting conditions

For the control group, our results are in general agreement with the data reported by Diener et al., using a similar set-up. Minor differences include shorter latencies found in the present study: The SL1 onset latency (34ms) and the LL onset latency (114ms) are lower than those reported by Diener et al., while the ML onset latency of 86ms falls within the range of variability found in the literature. These differences are likely to be due to...
different definitions of stimulus onset: Diener et al, used the signal triggering the platform tilt whereas we defined the onset as the first observable movement of the platform as recorded by a potentiometer. It therefore appears that the present latency values provide more accurate estimates for the actual time between receptor activation and muscle responses.

In the reports by Diener et al, an ML component was identified in 50–93% of healthy subjects. Our results agree with the lower figure; in this study identifying an ML response was often difficult because of its low amplitude and slow onset compared with the preceding SL1 and SL2 components. Diener et al1 reported that ML is velocity dependent: stimulus velocities exceeding the 50's used in this study may be necessary to elicit this response in a larger proportion of subjects.

Another apparent difference between the present data and the data obtained by Diener et al is the dissociation of the SL response into two distinct components, SL1 and SL2. This subdivision was based on two observations: (1) SL1 and SL2 showed two distinct peaks in almost all normal subjects and HD patients, and (2) in the sitting condition SL1 and SL2 were differentially modified. It is unlikely that the SL2 component actually corresponds to an ML response, which in our recording conditions might have occurred earlier than previously reported, because a separate SL2 component could be identified also in those subjects showing a well-defined ML response. Moreover, the ML response was abolished in the sitting condition, agreeing with previous studies, while the SL2 component remained essentially unchanged.

Further support for the distinction between different SL1 and SL2 components can be derived from comparison with observations in the literature. In the tibialis anterior muscle Iles19 found two reflex components at 28 and 50ms in response to electrical stimulation of the peroneal nerve or to mechanical tendon taps. These two responses could be functionally dissociated by their different behaviour with increasing electrical stimulus intensity. The latencies of these responses agree with the latencies of SL1 and SL2 in this study, and are both well below the value for ML. A similar occurrence of two distinct short latency reflexes was observed by Gottlieb and Agarwal20 in the triceps surae muscle following mechanically induced dorsiflexion pulses in sitting subjects. These responses occurred at latencies of 40 and 60ms comparable to our SL1 and SL2 latencies. The first of the components of Gottlieb and Agarwal20 showed a gain proportional to voluntary activation in the triceps surae whereas the second did not. In our study the triceps surae was tonically active in standing but not in sitting subjects. Therefore, our finding that only SL1 was larger in the standing than sitting condition, while SL2 remained unchanged, is in keeping with the gain characteristics described by Gottlieb and Agarwal for their short-latency components.

While the SL1 component probably corresponds to the myotatic reflex mediated via the Ia afferent system, an interpretation of the SL2 component is necessarily speculative. It might be mediated via afferents other than Ia fibres, such as group II spindle afferents,21 or it might be due to an oligosynaptically transmitted spinal Ia volley.22 As a human correlate for a group II mediated reflex is not available,23 it would be interesting to study whether SL2 fulfills criteria for group II activation, for example, by means of vibration.24 The idea that spindle II afferents become slightly less sensitive to small amplitude stretches in the presence of background fusimotor drive25 would be consistent with our findings that SL2 was slightly smaller in the standing condition, associated with background activation of the triceps surae.

At present, there is little information about the receptors being involved in the generation of ML and L1. Persistence of these responses after an ischaemic block at the ankle level causing anaesthesia of the foot and ankle, indicates that cutaneous receptors from the foot or ankle joint receptors are not involved.24 The reduction of SL1 in the sitting condition may be explained as a consequence of reduced alpha-motor neuron excitability in this condition, with the tibialis anterior muscle being generally active during background activation. It is not known whether the same explanation applies for the abolished ML response, as well. Because the LL response was recorded in the tibialis anterior, which was not tonically activated either when standing or sitting, similar changes in the tonic background activation are unlikely to have caused the disappearance of LL in the sitting condition. It is therefore more appropriate to attribute the observed LL changes to different modes of central processing ofafferent information according to different postural sets in the sitting and standing conditions.

**EMG responses in HD**

On clinical examination HD patients often show brisk tendon jerks,25 26 Paulson25 even stated that hyperreflexia is the rule in HD. However, Noth et al22 reported normal short-latency reflexes in the upper extremities of HD patients. This study confirms this finding for the triceps surae muscle. These quantitative findings indicate that mono- and oligosynaptic segmental reflexes are unchanged in HD, and that alterations in these reflexes do not contribute to the classically described hypotonia in HD,6 or to the increased muscle tone found in some patients.13 Since in the HD group the SL1 reflex was reduced to a similar degree in the sitting condition as in the normal group, it appears that the myotatic reflex gain is also normally regulated in HD according to varying degrees of background activation.

The tendency for the ML response to be smaller in HD patients than in normals is comparable to the absence of long-latency reflex components in the upper extremities of HD patients.12 Similar parallel features for the postural ML response and the M2/3 reflex in forearm muscles have been reported for
patients with Parkinson’s disease, in whom both types of responses are enhanced. The similarity of these findings indicates that the ML response may depend on mechanisms similar to the M2/3 response in the upper extremities. The M2/3 response has been shown to correlate with rigidity in PD. Hence, ML as its possible counterpart in lower extremities might be related to muscle tone regulation mechanisms, as discussed by Scholz et al. Although we did not quantitatively correlate the EMG responses with the clinical appearance of muscle tone, it would be expected that some of the 27 patients were hypertonic. None of the patients, however, showed an increased ML. Thus it is possible that the occasional hypertonia in HD may differ from classical rigidity of PD.

The finding of delayed LL onset may be considered as a neurophysiological correlate for the clinical notion of impaired postural stability in HD. Interestingly, PD, which is clinically even more characterised by impaired ability to correct postural perturbations, presents with normal LL responses. In PD patients, it is the exaggerated ML tending to assist the perturbation, which seems to correspond to postural instability. These differential findings in HD and PD suggest that different physiological mechanisms are involved in the postural impairments in the two conditions.

Both afferent and efferent conduction times in HD can be assessed with clinical neurophysiological tools: somatosensory evoked potentials after leg nerve stimulation are of normal latencies, although reduced in amplitude. Likewise, the afferent conduction time from the motor cortex to foot muscles have been found normal in HD, as assessed by magnetic cortical stimulation. Hence, a delay in a possible transcerebral reflex route in HD as documented by the increased LL latencies cannot be attributed to slowing of afferent or efferent conduction. This is in contrast to findings of delayed LL responses in cerebrovascular disease or multiple sclerosis, in which afferent or efferent conduction can be slowed down. The findings in this study indicate, however, that also in the absence of any delay in the afferent or efferent pathways a clearly delayed LL response may occur. Fig 6 shows an alignment of the fastest possible afferent and efferent conduction times in an HD patient and a normal subject, as estimated from somatosensory evoked responses to tibial nerve stimulation and muscle responses evoked by magnetic motor cortex stimulation. In the normal subject and the patient the fastest possible transmission from sensory receptors in the leg via motor cortex to leg muscles appears identical. The LL response in this HD patient, however, is considerably delayed, suggesting that in HD the LL delay depends on prolongation of intracerebral processing time. This is corroborated by the lack of correlation between LL latencies and efferent conduction times in the entire HD population, as shown for 25 patients in this study.

Promoted by the finding of normal LL responses in Parkinson’s disease, Diener et al reported persistence of ML and LL responses in the sitting condition, which was interpreted as an exaggerated shortening reaction in PD, as shown earlier for upper extremity muscles, as well as for the tibialis anterior. Diener et al concluded that in PD the regulation of postural response according to different functional demands is impaired. Our finding demonstrates a lack of a
similar appropriate regulation of the leg EMG responses according to postural set in HD.

This study was supported by a scholarship from the Alexander von Humboldt Foundation and by grants from the Deutsche Forschungsgemeinschaft (SFB 200, B9). We thank Dr H W Lange for referring his HD patients to us.

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