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Soleus H-Reflex Tests in Spasticity and Dystonia: A Computerized Analysis

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Summary. In 54 healthy individuals, and in 25 spastic and 7 dystonic subjects, soleus H-reflex vibratory inhibition, H/M ratio, and homonymous recovery curves obtained at two stimulus intensity levels were investigated in the same subject. In spasticity, the most prominent changes consist of a diminution of the vibratory inhibition at stimulus intensities lower than needed for a maximum H-reflex and an increase in the H/M ratio. These results suggest that presynaptic inhibition is reduced mainly at low-intensity levels and that excitability of motoneurons is increased. Recovery curves in spasticity do not show such significant changes as found for the recruitment curves. In dystonia, prominent changes occur in the late facilitatory phase of the recovery curve obtained at 0.5 H_{max} stimulus intensity, suggesting increased interneuronal activity. Vibratory suppression may be diminished, but H/M ratio is unaltered. A multivariate analysis was used to identify variables that discriminated between control, spastic, and dystonic subjects. The analysis yielded two canonical variables that are a linear combination of four H-reflex variables that contribute significantly to the group classification. Based on these two canonical variables, each group can be properly differentiated quantitatively. **Key Words:** Soleus H-reflex—Spasticity—Dystonia—Recruitment—Recovery.

Spasticity, hypertonia with a velocity-dependent increase in tonic stretch reflexes, is often associated with exaggerated tendon jerks, extensor plantar response, weakness, and slowing of movement (14). Some of its underlying spinal mechanisms can be elucidated electrophysiologically by means of the Hoffman (H)-reflex of the soleus muscle, by testing presynaptic inhibition of Ia afferents acting on Ia terminals (8,10,16), excitability of the soleus motoneurons (1,17), and the activity of some interneurons (9,21,23).

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In most spastic patients, reduced inhibition of the soleus H-reflex during vibration of the Achilles tendon appears to be caused predominantly by reduced presynaptic inhibition, although other mechanisms such as transmitter depletion may contribute to the vibration-induced H-reflex suppression (13). The total inhibitory action as a function of stimulus intensity can be shown by the soleus H-reflex recruitment curve recorded before and during vibration. Before a maximal H-reflex response is achieved, a direct soleus muscle (M) potential occurs and continues to increase along a sigmoid curve until it reaches a plateau at supramaximal stimulation. Recently, a cumulative vibratory index (CVI), which incorporates all vibratory effects up to a certain intensity, was reported to distinguish control subjects

from spastic patients better than the classic vibratory index that compares at only one intensity the maximum soleus H-reflex amplitude during vibration with the maximum amplitude lacking tendon vibration (19).

Many spastic patients may also have an increase in the so-called H/M ratio, i.e., the ratio between the peak–peak value of the maximum H-reflex response and the maximum soleus M potential. Although the H-reflex is not purely monosynaptic (5), the H/M ratio expresses, to a certain extent, an increased excitability of soleus motoneurons and can at least be used as an adequate parameter if it is corrected for the effect of age (10,19) and if it is combined with findings of the CVI (19).

Less commonly observed in spasticity are altered late facilitatory and inhibitory phases in the homonymous recovery curve of the soleus H-reflex, which may reflect an altered reactivity of some spinal interneuronal circuits (9,15,20,27,28). This curve is constructed by a plot of the H-reflex response to a test stimulus as a percentage of the H-reflex response to a conditioning stimulus of equal intensity versus the time interval separating both stimuli. At short time intervals $\leq$50 ms, an early facilitation followed by an early inhibition of the reflex to the test stimulus occurs in the curve, followed by a late facilitation at time intervals $\leq$350 ms, a late inhibitory phase up to a time interval of ~1,000 ms, and gradually by complete recovery.

In dystonia, clinically characterized as an involuntary movement disorder with slow and twisting movements, enhancement of the late facilitation in the homonymous recovery curve of the soleus H-reflex was reported by Sax et al. (24). Recently (22), physiologic recovery of the H-reflex in the upper limb at a 200-ms delay in patients with spasmodic torticollis and generalized dystonia was shown to be increased. In addition, a marked reduction of reciprocal inhibition was demonstrated in recovery curves derived from test H-reflexes of the median nerve conditioned by radial nerve stimulation in patients with writer's cramp, blepharospasm, spasmodic torticollis, and generalized dystonia; in the last two patient groups, a facilitation during the third period of inhibition was also noted (21,22). The investigators suggested that the derangement is secondary to alterations of supraspinal control upon postsynaptic, interneuronal activity. To our knowledge, no data are available on H/M ratio and vibration-induced suppression in dystonia.

We wished to investigate which changes in presynaptic inhibition, motoneuron excitability, and interneuronal activity alone or in combination are specific for spasticity and which changes are specific for dystonia as compared with normal controls.

METHODS

Subjects

The control group consisted of 54 healthy individuals aged 20–70 years (mean 37 years). Reflex studies were performed in the left leg in 17 and in the right leg in 37 controls. The patient group with spasticity consisted of 25 selected patients, aged 18–73 years (mean 47 years). Eighteen subjects had myelopathy, 3 had multiple sclerosis, 1 had a vascular lesion, 3 had a traumatic lesion, 7 had a familial spastic paresis, and 4 had myelopathy of unknown origin. The remaining 7 spastic patients had cerebrovascular disease. Twelve patients with myelopathy and 6 patients with cerebrovascular disease had mild characteristic velocity-dependent hypertonia [Ashworth (3) scale 1 and 2]. The remaining patients had severe hypertonia (Ashworth scale 3 and 4). All patients except 6 (4 with cerebral disease and 2 with myelopathy) had ankle subclonus or clonus. All spastic patients except two cerebral patients had an extensor plantar response. In 23 spastic patients, the duration of the neurologic deficit was >6 months; in 2 patients, it was ~6 weeks.

Seven subjects, aged between 15 and 48 years (mean 35 years) with predominant dystonic features were selected. Four subjects had cerebral palsy with generalized dystonia and an intermittent degree of normal tone or moderate rigidity. One patient underwent stereotactic surgery for tremor that caused a moderate lower limb dystonia and hypotonia. The other two patients had mild leg dystonia with normal muscle tone owing to a thalamic hemorrhage and encephalitis, respectively.

All normal volunteers and patients gave informed consent. At the time of the neurophysiologic investigation, none of the patients had received medical therapy. Patients were assessed clinically shortly before they were tested neurophysiologically. In hemiplegic patients, only the most affected limb was investigated; in patients with myelopathy or dystonia, the limb most affected or, when there was no difference between the legs, the right leg only was investigated. When spasms or clonus occurred H-reflexes were elicited only in the absence of so-

leus electromyographic (EMG) activity. In the dystonic patients, we noted the absence of EMG activity on the oscilloscope at the beginning and end of each reflex study in order to be certain of the relaxation of the legs. In addition, soleus EMG activity was also monitored aurally during the investigation.

Experimental Procedure

Recording and stimulation techniques for the soleus H-reflex have been described previously (12,19). During all tests, the subject was positioned in a reclining chair. Reflex responses, elicited by 1-ms square current pulses to the posterior tibial nerve in the popliteal fossa were amplified with a band pass filter of −3 dB at 2 Hz and 10 kHz, digitally stored with a sample frequency of 10 kHz in the EMG apparatus (Medelec, MS91) and subsequently digitally transferred from the EMG apparatus to the minicomputer (PDP 11/73).

To reduce the variability of the H-reflex responses, the time interval between successive trials during determination of the recruitment curves was at least 30 s (17,26). Because a strong gradient exists in the recruitment curve at low-intensity levels, the intensity increment of successive stimuli chosen was small at low-intensity levels and was gradually enlarged at higher intensity. The highest intensity was always supramaximal to obtain the maximum soleus M potential. Each recruitment curve consisted of 12–20 H-reflexes at different intensities. Vibration of the Achilles tendon with a frequency of 100 Hz and an undamped amplitude of 1 mm was applied by a Brüell and Kjær 4809 vibrator.

H-reflex recovery curves were constructed by application of 10–14 paired square-wave pulses of equal intensity. The time interval between successive trials was at least 30 s. Time intervals between conditioning and test stimulus were chosen at 50, 100, 250, 300, 400, and 500 ms and at 1, 3, 10, and 30 s. If necessary, extra paired stimuli with time intervals within the range of 50 to 320 ms, covering the facilitatory phase of the recovery curve, were added. Early facilitation and inhibition were not examined. The reflex responses to both the conditioning stimulus and the test stimulus were stored. Two intensity levels of the paired pulses were chosen; the intensity (S1) at which the H-reflex has its maximum value (H_{max}) and the intensity (S2) at which the H-reflex reaches half its maximum value (0.5 H_{max}) between threshold and H_{max} intensity level. Thus, for every patient, two H/M recruitment curves (without and during vibration) and two recovery curves (at H_{max} and at 0.5 H_{max} intensity) were recorded.

Quantitative and Statistical Analysis

Analysis of digitally stored waveforms was performed automatically in an off-line procedure. Peak–peak (PP) as well as area (AR) values of H-reflex responses and M potentials were calculated. A fixed window ranging from 5 to 25 ms and a window ranging from 25 to 60 ms were chosen for determination of the AR values of the M potential and H-reflex response, respectively. To reduce the effect of an artificial extra AR value, a correction was made for the baseline shift within this window by subtraction of a straight line that connected the signal values at the start and the end of the window.

The computerized method to plot H/M recruitment curves was described earlier (19). PP and AR values of the H/M ratio were measured. In addition, PP and AR values of the H/M ratio corrected for age were calculated because earlier the H/M ratio was shown to be inversely related to age. The CVI was used as a quantitative measure for the vibratory effects on the H-reflex (19). This CVI is defined as:

$$CVI(I_x) = \frac{\sum_{I=I_0}^{I_x} (V(I + dI) + V(I)) \times dI}{\sum_{I=I_0}^{I_x} (H(I + dI) + H(I)) \times dI} \times 100\%, \quad (1)$$

where V = H-reflex value (PP or AR) during vibration, H = H-reflex value (PP or AR) unvibrated, I_0 = stimulus intensity for H-threshold, I = actual stimulus intensity, and dI = stimulus intensity interval between two successive stimulus intensities.

The formula expresses the ratio between the surface under the recruitment curves obtained during and without vibration at the stimulus intensity (I_x) up to which integration is carried out. Only at two intensities were the CVI(S1) and CVI(S2) used for further statistical analysis: the intensity level yielding H_{max} (S1) and 0.5 H_{max} (S2), respectively. For PP and AR values, this resulted in four variables. Together with the age-corrected and uncorrected H/M ratio for PP and AR values, this yields a total of eight variables. The recovery curves were plotted as the ratio in percentage between the test H-

reflex response and the conditioning response against the time interval between the two stimuli of equal intensity. Two characteristic values of the recovery curves were used for the statistical analysis: (a) the value of the local maximum (LMA) of the test H-reflex occurring in the facilitatory phase with a time interval of the stimulus ranging from 50 to 320 ms, and (b) the value of the local minimum (LMI) of the test H-reflex noted in the inhibitory phase of the recovery curve with a time interval ranging from 320 to 1,000 ms. For PP and AR values at the two intensity levels S1 and S2, this resulted in eight variables. In addition, two other important recovery features were examined: the difference between the PP value and the AR value (P-A) of LMA for intensities S1 and S2 and LMA(S1)P-A and LMA(S2)P-A, as well as the difference between the PP value and the AR value of LMI for the two intensities LMI(S1)P-A and LMI(S2)P-A. This yielded four variables. All these variables together resulted in a total of 12 recovery curve variables; LMA(S1)PP, LMA(S1)AR, LMA(S2)PP, LMA(S2)AR, LMI(S1)PP, LMI(S1)AR, LMI(S2)PP, LMI(S2)AR, LMA(S1)P-A, LMA(S2)P-A, LMI(S1)P-A, and LMI(S2)P-A.

To assess the collective value of the various features of the total of 20 variables yielded by the H-reflex recruitment curves, H/M ratios, and recovery curves, a stepwise discriminant analysis (BMDP statistical software package, 7M) was performed. *F*-Statistics were used to determine whether a feature was entered ($F \geq 5.50$) into the discrimination. U-statistics (Wilk's λ) were used to test the equality of the three groups. This resulted in two canonical variables for each individual case that are a linear combination of variables that contribute most significantly to the discrimination. The two canonical variables are defined, respectively, as:

$$CAN1_k = \sum_{i=1}^{1} c1_i \times (v_{ik} - \bar{v}_i)/100\% \qquad (2)$$

$$CAN2_k = \sum_{i=1}^{1} c2_i \times (v_{ik} - \bar{v}_i)/100\%, \qquad (3)$$

where $CAN1_k$ and $CAN2_k$ = canonical variable 1 and 2 for the kth case, respectively; $c1_i$ and $c2_i$ = coefficient for the canonical variables 1 and 2 derived from the classification functions, respectively, v_{ik} = measured value of the most significant variables that contribute to the classification for the kth case, and $\bar{v}_i$ = mean value of v_{ik} of all groups used. From these canonical variables, a posterior probability score was derived by which each individual case was classified in one of the three groups, i.e., the control group, the group with spasticity, and the group with dystonia.

RESULTS

In the control group, H/M ratios decreased with increasing age. The linear regressions for H/M were (age) PP = H/M PP + 0.0080 × age − 30 ($r = -0.61$, $p < 0.001$) and for H/M (age) were AR = H/M AR + 0.0089 × age − 33 ($r = -0.59$, $p < 0.001$). Table 1 shows the mean values and SD of H/M ratios without and with age correction for the three different groups. The mean of H/M ratio corrected for age increased significantly ($p < 0.01$) in the group with spasticity as compared with that of the control group and that of the group with dystonia. Conversely, no significant difference of mean values of H/M ratios was noted between the control group and the group with dystonia.

Figure 1 shows recruitment curves without vibration and during vibration in a control subject and a patient with spasticity. These curves demonstrate that suppression of the H-reflex is maximal at lower stimulus intensities and that the overall suppression in the patient is markedly lower. In the control group, correlation coefficients between each of the four CVIs and age were <0.5. Age correction in this case did not reduce variability of CVI values significantly and was not included. Table 2 shows that all

TABLE 1. *H/M ratio (%)*

Variables	Controls (n = 54) Mean (SD)	Spasticity (n = 25) Mean (SD)	Dystonia (n = 7) Mean (SD)
H/M PP	49(18)	73[a](25)	49(23)
H/M AR	46(20)	74[a](27)	47(20)
H/M(age)PP	49(14)	81[b](24)	47[c](17)
H/M(age)AR	46(16)	83[b](27)	45[c](19)

H/M, H/M ratio; PP, peak–peak value H-reflex response; AR, area value H-reflex response.

[a] Significant difference ($p < 0.01$) with respect to control subjects (*t* test).

[b] Significant difference ($p < 0.01$) with respect to control group and other patient group (*t* test).

[c] Significant difference ($p < 0.01$) with respect to the other patient group (*t* test).

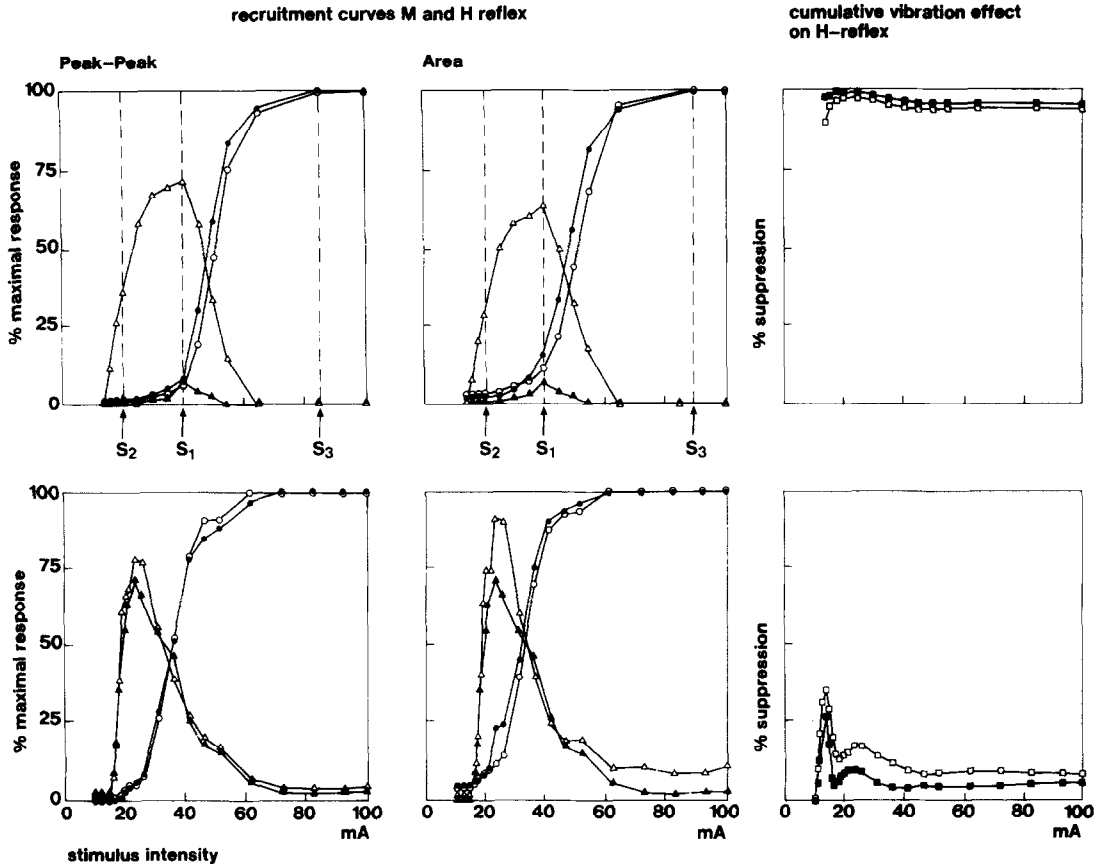

FIG. 1. Recruitment curves of soleus H-reflex response and muscle (M) potential (peak–peak as well as area values) without vibration (open triangle) and during vibration (solid triangle) of the Achilles tendon are shown for a control subject **(top)** and a patient with spasticity **(bottom).** All values are presented as percentages of the maximum M potential. The right-most panels show the cumulative vibration effect on the peak-peak (solid square) as well as the area (open square) values of the H-reflex. The amount of suppression is equal to 100% minus cumulative vibratory index (CVI).

CVI variables in the spasticity group differed significantly ($p < 0.01$) from the variables in the control group. Although the mean values of the CVIs in the group with dystonia were much higher than the ones in the control group, their SD were too large to be significantly different with a criterion of $p < 0.01$ ($p < 0.05$).

TABLE 2. *CVI (%)*

Variables	Controls (n = 54) Mean (SD)	Spasticity (n = 25) Mean (SD)	Dystonia (n = 7) Mean (SD)
CVI(S1)PP	20(17)	69[a](25)	77[a](40)
CVI(S1)AR	21(19)	68[a](22)	66(41)
CVI(S2)PP	12(13)	64[a](47)	78(60)
CVI(S2)AR	13(13)	67[a](46)	90(90)

CVI, cumulative vibratory index; S1, stimulus intensity at H_{max}; S2, stimulus intensity at 0.5 H_{max}; other abbreviations as in Table 1.

[a] Significant difference ($p < 0.01$) with respect to control subjects (*t* test).

Figure 2 shows recovery curves in a control subject (Fig. 2A), a patient with spasticity (Fig. 2B), and a patient with dystonia (Fig. 2C). Complete recovery occurred after ~10 s. At the 0.5 H_{max} stimulus intensity, longer time intervals usually were needed for complete recovery of the H-reflex than at the H_{max} stimulus intensity. Mean values of the recovery curve variables with their SD obtained in the control group and the two patient groups are shown in Table 3. None of the variables depended on age. For the group with spasticity, all mean values of the recovery-curve variables except LMA(S2)P-A and LMI(S2)P-A differed significantly ($p < 0.01$) from means in the control group. All mean values of the recovery curve except LMA(S1)P-A, LMI(S1)P-A, LMA(S2)P-A, and LMI(S2)P-A in the dystonic patients differed signif-

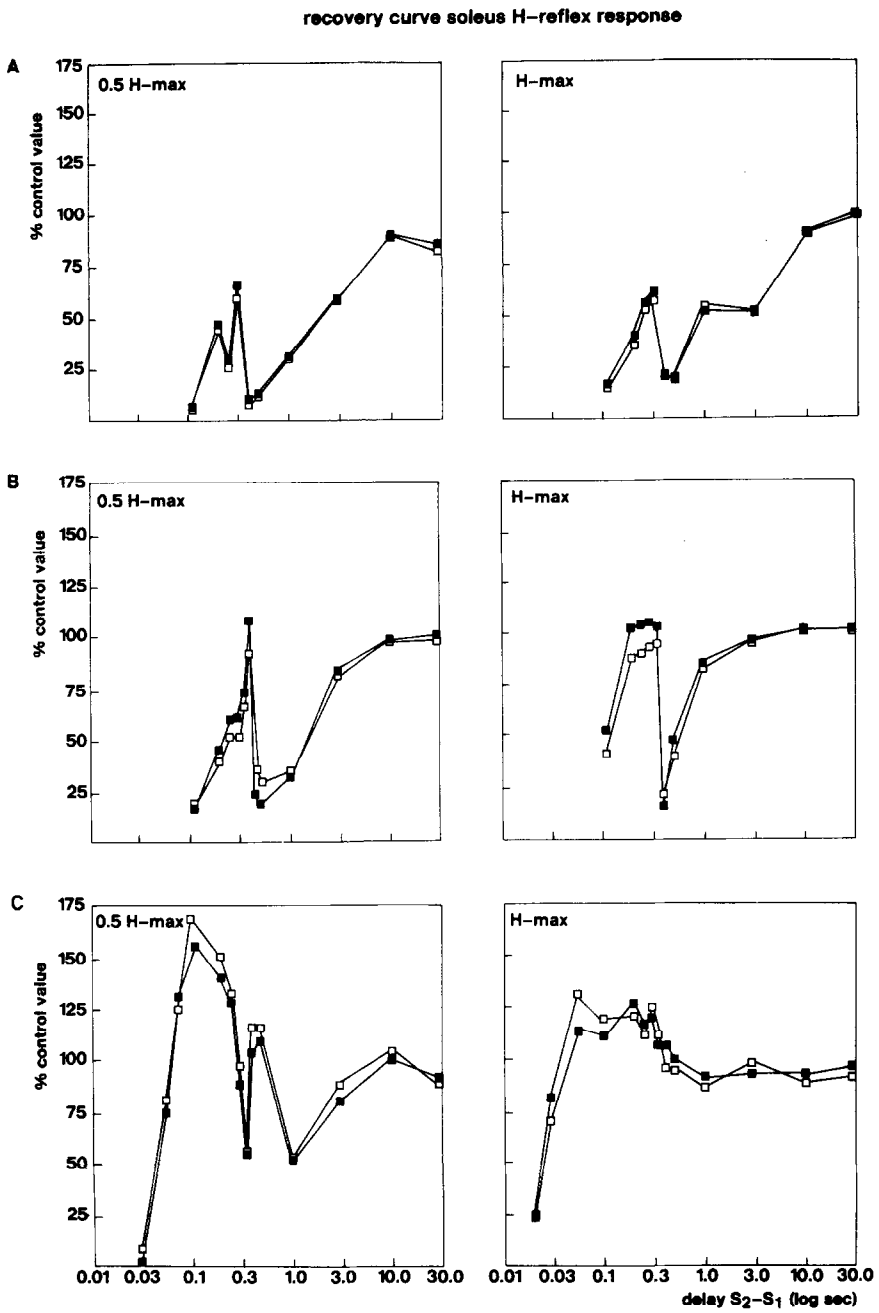

FIG. 2. Recovery curves of soleus H-reflex response [peak–peak (open square) as well as area (solid square) values] at 0.5 H_{max} and H_{max} stimulus intensity are shown for a control subject **(A)**, a patient with spasticity **(B)**, and a patient with dystonia **(C)**. All values of the H-reflex response to the test stimulus are presented as percentages of the preceding H-reflex response to the conditioning stimulus. The time interval between the conditioning and test stimulus is shown on a logarithmic scale.

icantly ($p < 0.01$) from the mean values of the control group. Between both patient groups, however, only the variables LMA(S2)PP, LMA(S2)AR, LMA(S1)P-A, and LMA(S2)P-A differed significantly ($p < 0.01$). The most prominent feature of the recovery-curve variables was the peak of the late facilitatory phase, especially at 0.5 H_{max} stimulus intensity. Its mean PP and AR value were increased in the group with spasticity but were even more increased in the group with dystonia. In the group with dystonia, recovery curves sometimes appeared to be bi- or even triphasic. The first of these peaks in the facilitatory phase occurred at a stimulus time interval shorter (i.e., 100–200 ms) than normal, but in some patients this first peak did not have the largest value. In the dystonic group, PP values of the largest peak in the late facilitatory phase, which was used for the statistical analysis, were often smaller than AR values. This is reflected by the negative mean value of LMA(S1)P-A and LMA(S2)P-A in the dystonic group. Conversely, in the group with spasticity, PP values of the facilita-

TABLE 3. *Recovery curve variables (%)*

Variables	Controls (n = 54) Mean (SD)	Spasticity (n = 25) Mean (SD)	Dystonia (n = 7) Mean (SD)
LMA(S1)PP	66(29)	103[a](23)	123[a](44)
LMA(S1)AR	62(27)	93[a](22)	128[a](52)
LMI(S1)PP	40(21)	66[a](23)	82[a](22)
LMI(S1)AR	39(20)	62[a](23)	79[a](18)
LMA(S2)PP	48(22)	84[b](30)	123[b](18)
LMA(S2)AR	44(20)	77[b](27)	132[b](28)
LMI(S2)PP	20(15)	39[a](19)	46[a](18)
LMI(S2)AR	19(14)	39[a](18)	43[a](16)
LMA(S1)P-A	4(7)	10[b](9)	−5[c](8)
LMI(S1)P-A	1(4)	4[a](4)	3(6)
LMA(S2)P-A	4(4)	7[c](8)	−9[c](12)
LMI(S2)P-A	1(3)	0(5)	3(5)

LMA, local maximum condition/test ratio of the H-reflex response in facilitatory phase; LMI, local minimum condition/test ratio of the H-reflex response in inhibitory phase; P-A, difference between peak–peak (PP) and area (AR) value of the H-reflex response; other abbreviations as in Tables 1 and 2.

[a] Significant difference ($p < 0.01$) with respect to control subjects (*t* test).

[b] Significant difference ($p < 0.01$) with respect to control group and other patient group (*t* test).

[c] Significant difference ($p < 0.01$) with respect to other patient group (*t* test).

tory peak tended to be larger than AR values and one facilitatory peak always was present between 100 and 1,000 ms. Figure 3 shows the raw recordings of both H-reflex responses to the conditioning and test stimulus at the stimulus time interval that relate to the late facilitatory peak in a patient with spasticity and a patient with dystonia. The response to the test stimulus in the patient with spasticity had a slightly shorter duration than the response to the conditioning stimulus, whereas in the patient with dystonia the response to the test stimulus was longer owing to a small after potential. This difference in duration between both H-reflex responses actually explains why the AR value is higher than the PP value in dystonia and smaller in spasticity and in controls.

To assess the collective value of some of the 20 variables shown in Tables 1–3, we performed a stepwise discriminant analysis. Classification functions were derived from the control group, the group with spasticity, and the group with dystonia. In descending order of significance, four variables were entered into the classification; i.e., LMA(S2)AR ($F = 27.7$), CVI(S1)PP ($F = 26.9$), LMA(S2)P-A ($F = 24.6$), and H/M(age)PP ($F = 22.6$). Table 4 shows the calculated coefficients $c1_i$ and $c2_i$ for the two canonical variables and the mean values of v_i for the groups used that must be substituted in expressions 2 and 3 (described in the Methods section). After substitution of the four most significant variables for each case, two canonical variables, *CAN*1 and *CAN*2, were calculated with expressions 2 and 3. The correlation of each of the canonical variables was 0.87 and 0.74, respectively. Figure 4 shows a plot of the three groups in the canonical plane. The three lines mark the borders between three areas. The upper-right area, the lower area, and the upper-left area refer to the control, spastic, and dystonic areas, respectively. A subject who falls into one of these three areas is classified in the group to which the area is assigned. A subject who comes to one of the borderlines, has an equal chance of being classified in one of the two groups on either side of the line. There is a great separation between the control group and the group with spasticity and an optimal separation between the group with dystonia and the two other groups. The asterisk in each area indicates the mean value of all cases of a particular group.

Four of 54 normal subjects fell into the area assigned to spasticity, and 2 patients with spasticity fell into the area assigned to the control group. Four of 7 (57%) patients with cerebrovascular disease were classified nearby or just across the borderline between the spasticity and control area, whereas this was true of only 6 of 18 (33%) patients with myelopathy. Furthermore, 3 of 7 (43%) patients with cerebrovascular disease but only one (6%) of the patients with myelopathy was located toward the dystonic area. Thus, the patients with myelopathy were better assigned to the area of spasticity than the group with cerebrovascular diseases. Table 5 shows the classification matrix (jackknifed) of the data shown in Fig. 4 and summarizes the number of subjects correctly assigned to the three clinically divided groups by four soleus H-reflex variables. Specificity and sensitivity between the control group and the group with spasticity as well as between the control group and the group with dystonia is 92.6 and 100.0%, respectively.

To determine for an individual spastic patient whether recruitment curve variables or recovery curve variables are more important for classification in the control group or the group with spasticity, multivariate analysis was performed twice with exclusion of the patients with dystonia. The first analysis was performed only with recruitment curve variables (Tables 1 and 2); the second was performed with both recruitment and recovery curve variables (Tables 1–3). With the first analysis, 52 (96.3%) of 54 controls and 22 (88%) of 25 spastic

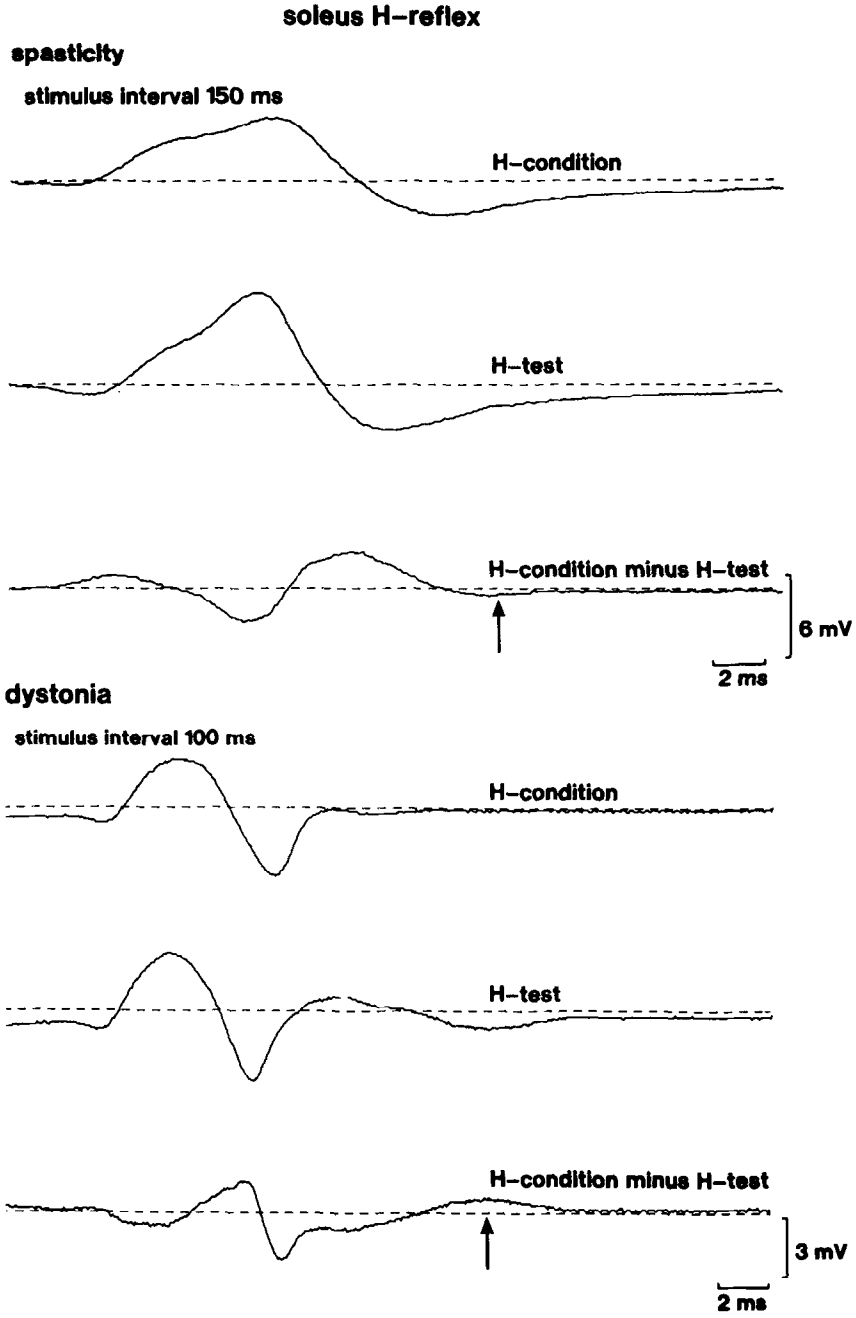

FIG. 3. Raw recordings of the soleus H-reflex response to the conditioning stimulus, the H-reflex response to the test stimulus, and the difference between both responses are shown in a patient with spasticity **(top three traces)** and a patient with dystonia **(bottom three traces).** The time interval of both stimulus pulses corresponds with the maximum of the late facilitatory peak. Arrow indicates the moment when a small after potential could be observed in the response to the test stimulus for the patient with dystonia but not for the patient with spasticity.

patients were correctly assigned. With the second analysis, a correct classification was achieved for 51 (94.4%) of 54 controls and 24 (96.0%) of 25 patients. Although most of the recovery curve variables of Table 3 differ significantly between the two groups, these analyses indicate that the contribution of these variables can almost be neglected. The recovery curve variables LMA(S2)AR and LMA(S2)P-A are essential for identifying the dystonic group, however.

DISCUSSION

Analyses of variables of soleus H-reflex recruitment and recovery curves within the same subject have demonstrated that in spasticity the most prominent changes occur in the amount of vibratory suppression at stimulus intensities lower than needed for the maximum H-reflex and in the height of the H/M ratio. These results confirm earlier findings (8,10,17,19). Recently, a computer study of Segev

TABLE 4. *Calculated coefficients for canonical variables and mean* v_i

Variables	Coefficients c1	Coefficients c2	Means groups used $\bar{v}$ (%)
1 LMA(S2)AR	−3.24	0.76	64.2
2 CVI(S1)PP	−3.58	−0.92	38.9
3 LMA(S2)P-A	10.02	−8.95	3.5
4 H/M(age)PP	1.40	−4.70	58.0

c1, c2, Coefficients for calculation of canonical variable *CAN*1, *CAN*2, respectively; $\bar{v}$, variable means of three groups used; other abbreviations as in Tables 1–3.

(25) demonstrated that a high density of excitable channels along uniform terminals may lead to a decrease in presynaptic inhibition, but whether in spasticity Ia afferent terminals actually are "hot" is not known. The computer model does not incorporate the excitability of the motoneuron itself and its effect on presynaptic inhibition. Values of the cumulative vibratory index and the regression coeffi-

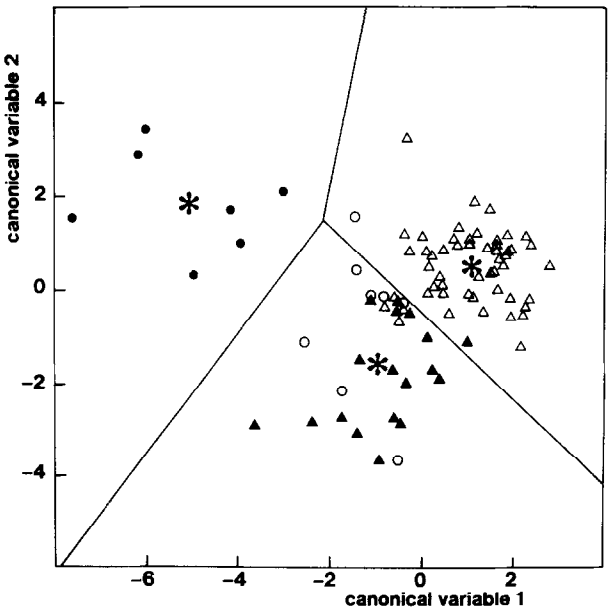

FIG. 4. A plot of the control group (open triangle), the group with spasticity, and the group with dystonia (solid circle) in the canonical plane. A subdivision of the group with spasticity is made between patients with myelopathy (solid triangle) and patients with cerebrovascular disease (open circle). Along the abscissa the first (*CAN*1) and along the ordinate the second (*CAN*2) canonical variable is plotted. The three lines mark the borders between three areas. The upper right area, the lower area, and the upper left area refer to the control, spastic, and dystonic areas, respectively. A subject who falls into one of these three areas is classified in the group to which the area is assigned. A subject who comes to one of the borderlines has an equal chance of being classified in one of the two groups on either side of the line. *Mean value of all cases in a particular group.

TABLE 5. *Classification matrix (jackknifed classification)*

Group	Correct (%)	No. of cases classified into: Controls	Spasticity	Dystonia	Group total
Controls	92.6	50	4	0	54
Spasticity	92.6	2	23	0	25
Dystonia	100.0	0	0	7	7
Total	93.0	52	27	7	96

cients for age correction of the H/M ratio do not differ significantly from previous findings (19), but now they have been obtained from a larger control and patient group. Although recovery curves may be altered in spasticity, they give no clear additional quantitative information to that already provided by the recruitment curves. This holds true for both 0.5 H_{max} and H_{max} intensity levels. Zander Olsen and Diamantopoulos (28) described a clear difference in soleus H-reflexes recovery curves between controls and patients with spasticity only at near-threshold levels.

In dystonia, on the other hand, the recovery curve variables obtained at 0.5 H_{max} stimulus intensity are predominantly distinctive, while vibratory suppression is diminished in some instances and H/M ratio is not significantly increased. This suggests that reactivity of interneurons that connect the tibial nerve sensory projections with the soleus motoneurons evidently is enhanced in dystonia; excitability of soleus motoneurons and, in some instances, presynaptic inhibition appear to be less affected. The physiologic findings we describe in dystonia with stimulation of the same nerve in the lower limb confirm the findings reported by Sax et al. (24) in the lower limb and recent findings of Panizza et al. (22) in the upper limb. They may have their counterpart in a decreased reciprocal inhibition that has been described in upper limb dystonia (18,21,22) and also indicate a defect in the central control of certain interneuronal circuits.

A new finding is that in the same subject late facilitation of the recovery curve obtained at 0.5 H_{max} intensities distinguishes controls from patients with dystonia to a greater extent than variables obtained at H_{max} intensity. This can be explained by the fact that at H_{max} intensity the conditioning stimulus recruits the maximum amount of motoneurons that can be activated reflexly. Therefore, the test stimulus appears incapable of recruiting more motoneurons and late facilitations may not exceed the

100% value. At 0.5 H_{max} intensities, late facilitations may be increased more strikingly because the reflex response to the test stimulus theoretically can be twice as large as the response to the conditioning stimulus. Another interesting feature of dystonia not reported previously is the dissimilarity between the waveform of the H-reflex response after the conditioning and the H-reflex waveform after the test stimulus. This dissimilarity can be traced to a difference in duration of both responses. In comparison with the conditioning response, the duration of the test response is prolonged owing to the occurrence of an after potential that probably originates from an abnormally high-gained interneuronal loop with a short delay of ~10 ms. This explains why PP values are usually smaller than AR values in dystonia. The observation that in dystonia, in a number of cases during the late facilitatory phase, even polyphasic highly peaked local maxima occur (Fig. 2c), may be due to exaggerated activity transmitted through polysynaptic segmental arcs with considerable long delays lasting up to several hundreds of milliseconds (11).

A further remarkable finding is that in controls, especially at 0.5 H_{max} intensities, a recovery takes place at time intervals >10–30 s. These long complete recovery periods probably are not associated with neurophysiologic processes based on electrical transmission but instead are more related to processes of neurotransmitter release. A practical consequence is that for an adequate judgment of soleus H-reflexes time intervals of successive stimulation must be >30 s, as was recommended by previous investigators but not thoroughly investigated (17,26).

The combination of many different types of soleus H-reflex tests in each subject and later application of multivariate analysis to the various neurophysiologic variables showed that only four variables are necessary for classification of a healthy subject or of a patient with spasticity or dystonia involving the lower limbs; i.e., in practice, only CVI(S1)PP and H/M(age)PP provided by the soleus H-reflex recruitment curves during and without tendon vibration as well as LMA(S2)AR and LMA(S2)P-A provided by the recovery curve at 0.5 H_{max} intensity are necessary for classification. Discriminant analysis provides the opportunity to combine the outcome of these four variables and reduce them to two canonical variables.

In contrast to earlier studies (8,10,17), use of a completely computerized method in our study is advantageous in that the time required for data acquisition as well as for data analyses to obtain the abovementioned four variables is shortened to half an hour for one leg and three quarters of an hour for two legs. Thus, the test procedure now can be used for routine investigations. Cost effectiveness can be further improved because the required hardware and software can be used on a personal computer (XT or AT).

This study has shown that a classification of patients with spasticity or dystonia based on four H-reflex variables is possible; this is a necessary condition for further study of the ways in which the outcome of the proposed neurophysiologic tests correlate with duration of neurological deficit (2) and severity of various clinical symptoms such as tonus, hyperreflexia, and extensor plantar response. These symptoms are a reflection of an underlying disease that actually consists of neurophysiologic and neurochemical alterations in the spinal cord. Quantitative neurophysiologic parameters also may be related more easily to working mechanisms of drugs, since experimental studies (4,6,7,9) showed a clear relationship between neurotransmitters and mechanisms of presynaptic and postsynaptic inhibition. The correlation between the proposed H-reflex tests and the effect of medical therapy is an interesting subject for future investigations.

CONCLUSIONS

Statistical analyses of soleus H-reflex recruitment and recovery curves in each subject showed that in comparison with control subjects patients with spasticity have a strongly decreased vibratory-induced suppression and an increased H/M ratio, if it is corrected for age. In spastic patients, the second facilitatory peak of the homonymous recovery curve was slightly increased. On the other hand, patients with lower limb dystonia demonstrated a tremendous increase of this facilitatory peak and usually a polyphasic shape of the recovery curve, especially at 0.5 H_{max} intensity. In the dystonic patient group, vibratory-induced suppression and H/M ratio were not significantly altered.

Application of multivariate analysis to the various tests showed that in practice only four variables are necessary for classification of a healthy subject, a patient with spasticity, or a patient with dystonia involving the lower limb: i.e., the CVI at H_{max} intensity, the H/M ratio corrected for age, the area value as well as the difference between the area,

and PP value of the second facilitatory peak of the recovery curve measured at 0.5 H_{max} intensity. Introduction of a completely computerized method and the finding that both data acquisition and data analysis now can be restricted to the abovementioned four variables make the proposed neurophysiologic test procedure applicable for routine investigations and follow-up studies.

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