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Artículo original

Increase of the H reflex amplitude and absence of long latency reflexes in the intrinsic hand muscles in patients with spasticity

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AUMENTO DE LA AMPLITUD DEL REFLEJO H Y AUSENCIA DE LOS REFLEJOS DE LARGA LATENCIA EN LOS MÚSCULOS INTRÍNSECOS DE LA MANO EN PACIENTES CON ESPASTICIDAD

RESUMEN

Objetivo: investigamos los reflejos H y los reflejos miotáticos de larga latencia (LLRs) de los músculos tenar e hipotenar de 15 sujetos sanos y de 20 pacientes con espasticidad. Los objetivos del estudio fueron: **1.** dilucidar si los reflejos H de los músculos intrínsecos de las manos podían ser inducidos en pacientes espástico en reposo muscular; **2.** evaluar si la amplitud del reflejo H podía ser incrementada con la contracción muscular, y **3.** determinar si la ausencia de los reflejos de larga latencia estaba relacionada al tamaño del reflejo H que lo precedía o a falta de contracción muscular. *Material y métodos:* el estímulo eléctrico fue un pulso cuadrado de 1 ms con una tasa de repetición de 3 Hz, submáximo para la onda M. Las respuestas fueron promediadas 100 veces e inducidas con el músculo en reposo y con una contracción muscular isométrica sin resistencia. *Resultados:* la respuesta H fue obtenida en reposo en 100% de los pacientes espásticos y sólo en 30% de los sujetos sanos. En los pacientes espásticos el reflejo H en reposo muscular fue de mayor amplitud que en los sujetos normales y durante la contracción muscular aumentó un promedio de seis veces su amplitud durante el reposo. No se obtuvieron potenciales de larga latencia en reposo en el sujeto normal pero fueron fácilmente observados durante la contracción muscular. Los reflejos de larga latencia no estuvieron presentes en los pacientes espásticos ya sea en reposo o con la contracción muscular. La ausencia de reflejos de larga latencia no estuvo relacionada con el tamaño del reflejo H que la precedía y estuvo ausente a pesar de una contracción muscular efectiva. *Conclusiones:* los hallazgos son indicativos de un aumento en la excitabilidad del *pool* de motoneuronas alfa en reposo y un aumento en la sincronicidad y reclutamiento durante la contracción muscular. La ausencia de reflejos de larga latencia no están relacionados al tamaño del reflejo H que los precedía y no estuvo relacionado a una ausencia de contracción muscular efectiva.

Palabras clave: músculos tenar e hipotenar, espasticidad, reflejo H, reflejos de larga latencia.

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ABSTRACT

Objective: we investigated the H reflexes and the long latency reflexes (LLRs) of the thenar and hypothenar muscles in fifteen normal subjects and in twenty patients with spasticity. The objective were: **1.** to elucidate if the H reflexes could be elicited in the intrinsic hand muscles in patients with spasticity under a relaxed condition; **2.** evaluate if the amplitude of the H reflex could be increased with muscle contraction and **3.** to determine if the absence of the long latency reflexes was related to the size of the preceding H reflex or to a lack of

muscle contraction. *Methods:* the stimulus was a constant current square pulse of 1 ms with a repetition rate of 3 Hz with a threshold intensity for the M wave. The responses were averaged 100 times. The response was induced with the muscle at rest and with sustained muscle isometric contraction without resistance. *Results:* the H response was obtained at rest in 100 percent of the spastic patients and only in 30 percent of the normal subjects. In the spastic patients the H response at rest was of higher amplitude than in normal subjects and during muscle contraction the H response increased a mean of 6 times the amplitude at rest. The LLRs were not obtained at rest in the normal subject but they readily appeared during muscle contraction. The LLRs were not present in the spastic subjects either with the muscle at rest or with muscle contraction. The absence of the LLR was unrelated to the size of the H reflex and did not appear despite the presence of an effective muscle contraction. *Conclusions:* the findings are indicative of an increase in excitability of the alpha motoneuron pool at rest and increased recruitment and synchronicity of firing of alpha motoneurons during muscle contraction in patients with spasticity. The absence of the long latency reflexes appears to be unrelated to the size of the preceding H reflex and also unrelated to the lack of muscle contraction.

Key words: thenar and hypothenar muscles, spasticity, H reflex, long-latency reflexes.

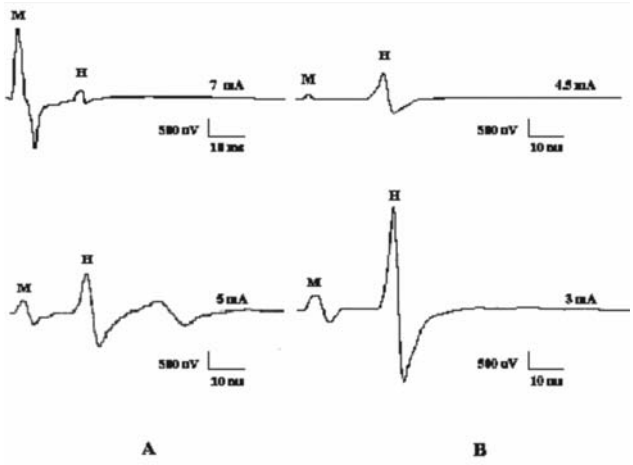
H reflexes can be evoked in the thenar and hypothenar muscles of the upper extremities with electrical stimulation, at the wrist, of the median and ulnar nerve respectively [1-6]. In normal subjects these reflexes can only be elicited if these muscles are subjected to some degree of contraction [1,4,7-9]. Under relaxed conditions the H reflexes of these muscles are usually not attainable[1,5,7,9]. It has been found that in spastic patients the H reflexes are of high amplitude[10-12] but Sica *et al,* indicated that they do not increase in amplitude with muscle contraction [13]. Adam *et al,* found that the long latency reflexes induced by mechanical stretch of the human long thumb flexor are attenuated or absent in spastic patients with lesions of the internal capsule or sensorimotor cortex[14]. Conrad and Aschoff demonstrated absence of the long latency reflexes of the abductor pollicis brevis (APB) muscle with electrical stimulation of the median nerve at the wrist in hemiparetic patients[8]. This has been confirmed by others[4,5,9,15-17]. It has also been observed that in spastic subjects the H reflexes can be elicited in muscles other than the soleus with the muscle relaxed[10-12,18]. However some of these observations have not been systematically studied. For example it is not known if in normal subjects the H reflexes in the intrinsic hand muscles are sometimes elicited at rest. Although it has been found that some spastic subjects are unable to increase the size of the H response with muscle contraction it is not clear if this lack of potentiation is due to an inability to contract the muscle due to paralysis or severe paresis. The mechanism of the absence of the long latency reflexes (LLRs) in spastic subjects has also been a matter of controversy. It might be related to the high amplitude of the preceding H reflex with a subsequently prolonged refractory period, to damage of the descending excitatory supraspinal influences upon the motoneurons that subserve the long latency reflexes, or simply to a lack of muscle contraction due to the paralysis[4,5,9]. Hence we decided to investigate if the amplitude of the H reflex was correlated with the absence or presence of the LLRs in spastic subjects that were paretic but not paralyzed. We undertook a systematic study to assess the presence or absence of these reflexes in the APB muscle and measured the amplitude of the H reflex at rest and with muscle contraction in patients with spasticity of the upper extremity and we also investigated if in the presence of an effective muscle contraction the long latency responses remained absent.

PATIENTS AND METHODS

Subjects and patients: twenty spastic subjects were studied, eight had spastic hemiplegia, two had spastic quadriparesis and ten had spastic cerebral palsy. Their ages ranged from 15 to 64 with a mean age of 56 years. The patients signed an informed consent. The procedure was fully explained to them and they had the freedom to withdraw from the study at any time if the test proved too disagreeable. The two patients with spastic quadriparesis had spondylotic myelopathy confirmed by MRI; the eight cases with spastic hemiparesis or hemiplegia had cerebral infarctions demostrated by CT or MRI. Three had capsular infarction and five cortical infarctions. The infarction was present for at least a year. The patients with spondylotic myelopathy had symptoms and signs of spasticity between 6 months and a year. The patients with cerebral palsy had spastic quadriparesis. All had rapid 4+ reflexes in the muscles of the upper extremities and particularly in the finger flexor muscles. They all could abduct the thumb fully without

resistance and could make some movement against resistance. Hoffmann sign was present in the twenty cases with rapid snapping of the middle finger. None of the patients had an inverted supinator reflex. We excluded the patients who could not make a sustained abducting movement of the thumb as we wanted to evaluate the response with muscle contraction. We also excluded those cases who had chronic contracture in flexion of the wrist and the thumb. We evaluated the degree of spasticity using the Ashworth classification [2]. Fifteen patients were considered to be 3 in the scale of Ashworth (the spasticity allows flexion and extension of the fingers) and five were considered to be in 4 in the Ashworth scale (spasticity slightly limits flexion and extension of the fingers). Nineteen patients had a Babinski reflex in the side of the spasticity. We also studied fifteen healthy control subjects (aged 24 to 32 years) who underwent the same protocol and who volunteered for the study.

Electrophysiological procedure: The H reflexes were elicited with constant current stimulation at the wrist of the median or ulnar nerves with the cathode positioned proximally with a constant current stimulus of 1 ms duration at a repetition rate of 3 Hz (Cadwell Sierra II, USA). The electrodes were gold-cup electrodes positioned in the bulk of the APB and abductor digiti minimi (ADM) muscles with the reference in the root of the respective finger. The ground electrode was placed in the dorsum of the hand. The electrodes were securely fastened to the skin with double or triple tape to avoid movement during the contraction. The filters were set at a low frequency pass of 10 Hz and at a high frequency pass of 10 kHz. The sweep speed was set at 10 ms per division. The stimulation was threshold for the M wave and was kept constant throughout the study. The compound muscle potential was averaged 100 times and stored digitally for further analysis. The responses were not rectified. The muscle contraction was isometric and sustained during the total period of stimulation for each trial. The muscle contraction of the APB and the ADM was a sustained abducting movement of the thumb and fifth finger respectively without resistance. Two conditions were studied: **1.** rest; **2.** sustained abduction of the thumb or fifth finger. Two responses, for each hand, were obtained in each patient, four for each patient. The percentage of H reflexes obtained at rest in normal subjects and in spastic patients was calculated for each H reflex. We measured the amplitude of the H reflex and the M wave under two conditions **1.** at rest and **2.** during muscle contraction. We also obtained the index of amplitude of the H at rest divided by the H amplitude with muscle contraction (H_{cont}/H_{rest}) as well as the Hrest/Mrest index in order to assess the degree of motoneuron recruitment with muscle contraction. In general we followed the procedures recommended by the Guidelines of the International Federation of Clinical Neurophysiology for the study of long latency reflexes except that we did no rectify the responses[9]. The latencies could be precisely measured without the rectification. The latencies were measured for subjects and patients off line. We did not investigate the maximal M response or the ratio H_{max}/M_{max} .We used descriptive statistics for the measurement of mean and standard deviation of amplitudes and latencies and the Student T test to compare data.



A. H reflexes and long latency reflexes in the APB of a normal subject. Upper figure H reflex in a normal subject obtained with the muscle at rest. Lower figure H reflex with muscle contraction. Notice the low amplitude H reflex at rest and the increase of amplitude with muscle contraction. There is a clear long latency reflex after the H reflex with the muscle contraction. **B.** H reflex in the APB muscle in a patient with spasticity. Upper figure H reflex in the spastic patient at rest. Lower figure H reflex with muscle contraction. Notice the higher amplitude of the H reflex in relation to the M response at rest and the large increase of the H response with muscle contraction. In both cases the H response is much higher than the M response. The LLRs are absent at rest and with muscle contraction (the responses were averaged 100 times). The stimulus intensity (at the right of each figure in mA) was slightly lower with muscle contraction. The variable size of the M response is probably related to minimal changes of the position of the stimulating electrodes in relationship to the nerve due to the muscle contraction probably due to movement of the wrist tendons.

RESULTS

The H response was clearly different in the spastic side as compared to the normal non spastic side and the normal subjects.

Normal subjects or normal side in spastic patients: at rest we found the H reflex present in 30 percent of the normal subjects. The H response was usually of small amplitude; about 10 to 15 percent of the M wave. Contraction against resistance elicited the response in the sound side in all patients and in all normal subjects; the muscle contraction also increased its amplitude. The latency of the H response did not change from rest to muscle contraction. The amplitude of the H response with muscle contraction was about the same size of the M response although in some subjects was as much as three times greater. The LLRs were present only with contraction (figure 1A).

Spastic patients in the spastic side. Presence of the H reflex: in the spastic subjects the H response was present at rest under relaxed condition in all trials (100 percent). The amplitude of the H response at rest in patients with spasticity was clearly greater than the amplitude seen in normal subjects or in the normal side in the same patient; being frequently of the same size of the M response and ranging from 50 to 100 percent of the M response amplitude (ratio H_{rest}/M_{rest} 0.5 ± 0.2 in spastics as compared to the ratio in normal subjects of 0.1 ± 0.01) (figures 1B, 2 and 3). The amplitude with muscle contraction was also significantly greater in spastic subjects (figures 1,2 and 3). When we obtained the ratio H amplitude with muscle contraction/ H amplitude at rest ($H_{cont}H_{rest,}$ index) we found that the H response increased with muscle contraction up to 6 times compared to the amplitude observed at rest (6 ± 2 in spastics compared to 2 ± 0.5 in normal subjects). The LLRs were consistently absent regardless of the size of the preceding H reflex. The LLRs were not observed despite the clear presence of an effective muscle contraction as assessed by surface EMG recordings.

Latencies of the H reflex: the latency of the H response was not different when we compared the normal (26 ms ± 2.03 ms with a standard error of 0.28, range 22-28.2ms) to the spastic side (27 ms ± 1.89 ms with a standard error of 0.32, range 23-28.8ms). In patients with quadriparesis the H response latencies were the same in both sides and did not differ from the normal subjects. LLRs were not observed in any trial following the H response in the spastics. In normal subjects or in the sound side LLRs were seen following the H wave in 71 percent of the trials during muscle contraction.

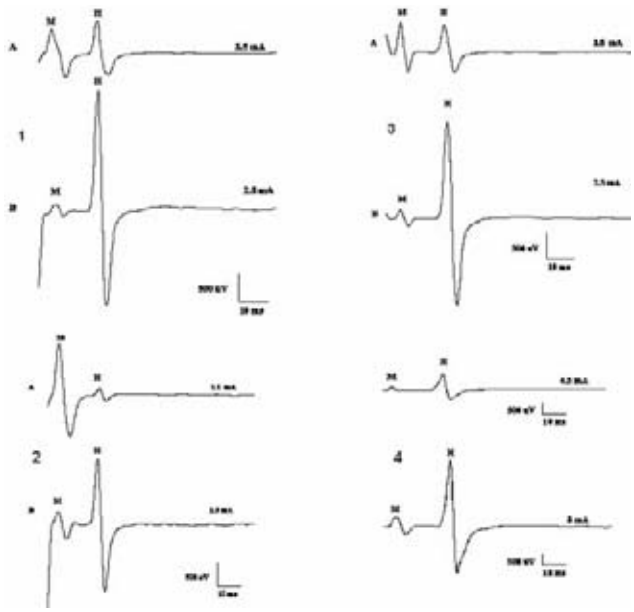


Figura 2. H reflexes obtained in the thenar muscles in four patients with spasticity of the upper extremities in response to a submaximal stimulus to the median nerve. The upper figure depicts the H reflex with the muscle at rest. The lower figure the H reflex with the muscle contracting. The H response is invariably present at rest and in three subjects is greater than the M response. With muscle contraction the H response increases up to eight times the amplitude of the response at rest. The long latency responses are not present neither at rest nor with muscle contraction. The responses were averaged 100 times. (M: M response; H: H reflex,the intensity of the stimulus in mA is at the right side of each trace). The slight variability of the size of the M response is probably related to slight changes of the position of the electrode in relationship to the nerve due to movement of the wrist tendons.

Intensity of the stimulus: the mean constant current intensity of the stimulus that evoked the H reflex of the APB and ADM muscles in normal subjects was 7.5 ± 2.8 mA (standard error 0.3, range 2.5-14mA). The mean intensity of the stimulus that evoked the H reflex in the muscles in spastic patients was 6.5 ± 1.8 mA (standard error 0.4, range 2.5-12mA). Although the intensity of the stimulus required to elicit the M wave was less in the spastic patients it was not statistically significant. We discarded contamination by an F response by the fixed latency, configuration and size of the H reflex and also because the H reflexes and the long latency responses were elicited with a threshold or submaximal stimulus to the M wave and that type of stimulation also made unlikely contamination by an F wave that is usually elicited by a supramaximal stimulus to the M wave. We did not determine the ratio H_{max}/M_{max} because we did not give a supramaximal stimulus to the M wave. However in two spastic patients

we studied the extinction of the H reflex in and found that as the M wave achieved its maximal amplitude the H reflex became rapidly suppressed as it has been found in the soleus H reflex.

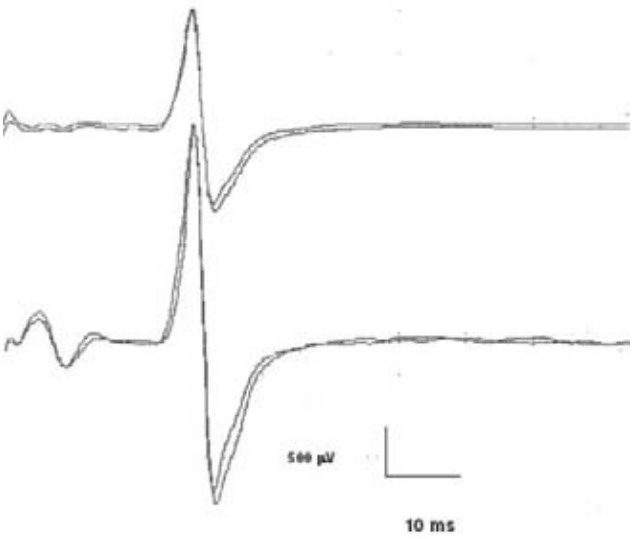


Figura 3. Replicability of the H reflex amplitude of the APB muscle in a patient with spasticity. Upper figure: the M and the H response at rest obtained twice after averaging 100 stimuli and superimposed. Lower figure: the M and the H response with muscle contraction obtained twice after averaging of 100 stimuli and superimposed. Both were obtained averaging 100 responses. The responses were obtained with a submaximal stimulus. The amplitudes of the responses replicate in amplitude and latency.

DISCUSSION

H reflex of the intrinsic hand muscles

The H reflex is a segmental oligosynaptic spinal reflex that in normal subject at rest appears with the muscle relaxed only in the soleus and flexor carpi radialis (FCR)[10,11,18]. The soleus may be more frequently subjected to tonic innervation because is used mainly for antigravity purposes whereas other muscles are primarily subjected to phasic innervation because they are not used in man to counteract gravity[11]. Such is probably the case of the intrinsic hand muscles that are used to perform fine phasic independent finger movements. In fact the hand muscles are muscles that are used for fine tactile exploration of the environment and have the largest cortical representation in man[16]. We found that in about 30 per cent of normal subjects a small amplitude H reflex may be elicited with a submaximal stimulus to the M wave in the APB muscle at rest (figure 1A). This has not been previously reported probably because had not been systematically studied.

Teasdall *et al,* in 1952 discovered that in patients with spasticity the H reflex of the anterior tibial muscles, that is a normally suppressed monosynaptic reflex, could be elicited with electrical submaximal stimulation of the peroneal nerve[18]. Hohman and Goodgold observed that H reflexes in spastic patients could easily be obtained in muscles other than the soleus[10]. They reasoned that the presence of the H reflex in various muscles in patients with spasticity was indicative of an increase in the excitability of the segmental alpha motoneurons innervating these muscles. Yanagisawa et al. encountered that in patients with spastic hemiplegia the H reflex of the pretibial muscles could also be obtained[19]. The stimulus given in all these studies was threshold for the M response as that intensity was found optimal for the elicitation of the H reflex of the soleus. The stimulation was also given under relaxed conditions. These studies seem to indicate that in patients with spasticity the H reflexes of certain muscles, that in normal subjects are usually absent, appear with submaximal or threshold stimulation to the M wave.

The above studies seem to indicate that damage to the descending supraspinal influences increases the excitability of the alpha motoneuron pool in various spinal segments[10,11,18-21]. The amplitude of the maximal H reflex in spastic subjects is also increased[14] and therefore the relationship between the maximal H reflex and the maximal M response (H_{max}/M_{max}) is also increased beyond that reported in normal subjects (about 0.59)[3,11]. We found that the H reflexes of the thenar and hypothenar muscles in spastic subjects can be easily elicited in all trials under a relaxed condition. In patients with spasticity of the upper extremities in whom the myotatic reflexes of the fingers flexors are hyperactive it is likely that the segmental alpha motoneurons subserving these muscles have a resting excitability greater than normal. This has been described in spastics subjects in muscles other than the soleus[18,19,21]. Thus when a peripheral volley of impulses through the Ia afferent fibers arrive to the alpha motoneuron pool an H reflex of these muscles is induced under a relaxed condition because of an increase in basal alpha excitability. Other factors that may increase the basal excitability of the alpha motoneuron pool include a decrease in presynaptic and Renshaw cell inhibition[22]. We found that the H reflex of the intrinsic hand muscles, at rest, had frequently an amplitude of the same size of the M response. Therefore we found that the index H_{rest}/M_{rest} may be useful as an index of spasticity. H reflexes of the same amplitude of the M response are never seen with

relaxed intrinsic hand muscles in normal subjects.

Under muscle contraction in normal subjects it is likely that the excitability of the alpha motoneurons should increase due to the excitatory descending facilitatory influences[1,7,9,23]. We found that the H response in spastics during muscle contraction increases in amplitude. Sica *et al* [24] found in spastics a "lack of potentiation" of the amplitude of the H response in the same muscles. We believe this difference is probably related to the inability of some patients to contract the muscles as some spastic patients are paralyzed or may even have flexor contractures. We carefully excluded all the patients who were unable to make an abducting movement of the thumb or fifth finger. We observed that the increase in the amplitude of the H reflex with muscle contraction was much higher than the amplitude observed in the sound side in spastic subjects or to that observed in normal subjects. In some cases the H reflex amplitude during muscle contraction may be up to eight times that of the M wave. This increment was particularly noticeable in patients with cerebral palsy. This indicates that supraspinal influences are able to recruit more alpha motoneurons during muscle contraction and make them fire synchronously. The index relating the amplitude of the H reflex during contraction to the H reflex at rest (H contraction/ H rest) H_{cont}/H_{rest} may prove to be useful as a measure of this increased recruitment in the evaluation of spasticity. We found that this index may be as high as 8. All these indexes need further evaluation in spastic subjects but we have found them highly useful and reproducible.

Long latency reflexes in the intrinsic hand muscles.

In normal subjects, when the median or ulnar nerves are stimulated at the wrist with the cathode proximal and the thenar or hypothenar muscles exert a contraction an H reflex develops at around 28 milliseconds[2,3,8,9] and three successive responses are seen after the H reflex. The first response has been termed long latency response I (LLRI), that appears at approximately 40 ms, the second, long latency response II (LLRII), that appears at 50 ms and a third wave (LLRIII) may appear at 75 ms[2,,3,8,9]. The most consistent and most studied of these LLRs is LLRII. Deuschl *et al,* thought that the LLRII was mediated by fast conducting muscle and cutaneous afferents[3]. The LLRII has been thought to represent a transcortical reflex: ascending through the dorsal columns and the thalamus, integrated at the sensory motor cortex and descending through the pyramidal tract [3-5,8,9,16,17,21,23,25,26].

It has been thought that the final common pathway for the LLRII are the same motoneurons of the preceding H reflex. However the presence of the LLRs does not invariably correlates with an increase in the excitability of the alpha motoneurons of the H reflex. The alpha motoneurons recruited for the LLRs fire less synchronously than the preceding H reflex. The H reflex is more synchronous with less duration and greater amplitude than the LLRII [5]. This could reflect different times of activation of the alpha motoneurons by the descending impulses, a different set of motoneurons or a polysynaptic reflex [24]. Adam *et al,* first discovered that lesions of the internal capsule in man abolished the long latency reflexes induced by mechanical stretch of a hand muscle[14] and subsequently this was confirmed by many others in man and in the experimental animal [16,17,23,25,]. This was confirmed by Conrad and Aschoff[8] with electrical stimulation of the median nerve in hemiparetic patients and confirmed by many others[3-5,9]. Components M1 and M2 induced by mechanical traction of a muscle are differentially affected by fatigue. The short-latency reflex response M1 (akin to the H reflex) is abolished by a brief maximal voluntary contraction in a small intrinsic hand muscle whereas the long-latency component (akin to the LLR) is not[27]. This also points to a mechanism responsible for M1 that is different from that of M2. Other ideas of the mechanism of production of LLRs includes activation of spindle secondary afferents and type II nerve afferent fibers (Matthews)[24] and the idea that cutaneous afferents could be the primary source of the reflex (Corden *et al)* [28]. It is difficult to conceive a mechanism based on these two theories to explain the absence of the LLRs in spasticity as polysynaptic flexor responses are also hyperactive in spasticity[10].

The LLRII has been reported to be absent in the intrinsic hand muscles in patients with Huntington disease[22] and in the ankle, to mechanical stretch, in multiple sclerosis[15]. In spastic patients mechanical stretch showed an absence of the M2 myotatic response[16,17,23]. The dissociation of the H reflex and the LLRs is of interest. It is in accordance to the theory that the LLRII response may be a transcortical reflex[2-4,27]. However it is difficult to assess in such a long circuit whether only the damaged efferent part of the reflex (that is the pyramidal or descending tract) is solely responsible for its absence. The study of patients with pure motor hemiplegia of vascular origin or patients with spasticity due to ALS may be of interest in this point. It has also been suggested that the absence of LLRs in spasticity might be secondary to a refractory state of the alpha motoneurons due to the large preceding H wave[9]. We have found that the amplitude of the H response in spastics does not correlate with

the absence of the long latency responses. Relatively small amplitudes H reflexes are not followed by the long latency responses. More importantly it is not related to a lack of muscle contraction as our patients could contract the studied muscles. This suggests that the absence of the LLRs is not related to an increase of the refractory state of the alpha motoneurons due to the preceding large H wave or to the lack of muscle contraction but to some other factor among which the interruption of the efferent arm of a transcortical reflex looms large.

CONCLUSIONS

In conclusion the H and long latency reflexes of the thenar and hypothenar muscles in patients with spasticity have the following physiological characteristics: **1.** they can be readily produced by a threshold stimulation to the M wave under a relaxed condition; **2.** with the muscle at rest the H reflexes are frequently of the same size as the preceding M wave; with the ratio H_{rest}/M_{rest} increased to 0.5 as compared to 0.1 or less in normal subjects; **3.** with the muscle contracting the H reflex increases its amplitude frequently as much as eight times the size of the preceding M wave; this indicates an ability to recruit more motoneurons to discharge synchronously and an increase excitability of the alpha motoneuron pool; thus, the ratio of the amplitudes between the H reflex with muscle contraction to the H reflex with the muscle relaxed may be as high as 6 ± 2; **4.** the H reflexes of the intrinsic hand muscles in spastics are not followed by the LLRs as in normal subjects and its absence is unrelated to the size of the preceding H reflex and is not due to lack of muscle contraction; the absence of LLRs in spasticity remains unknown but the most likely possibility is interruption of the efferent arm of a transcortical reflex; **5.** the latencies of the H response are not different in spastics than in normal subjects; **6.** we have found that the relationships Hrest/ Mrest, and Hcont/Hrest may be useful as indices of spasticity at least in the intrinsic hand muscles.

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