Arch. Psychiat. Nervenkr. 228, 197–204 (1980)

Archiv für Psychiatrie und Nervenkrankheiten
Archives of Psychiatry and Neurological Sciences

The Late Facilitation in H-Reflex Recovery Cycles in Different Pyramidal Lesions*

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Summary. H-reflex amplitudes were recorded after stimulation of the tibial nerve and different electrical stimuli in 18 normal persons and 26 patients showing pyramidal spasticity (8 spastic spinal paralysis, 6 spastic hemiparesis, 12 spinal lesions).

A just subthreshold stimulus of the tibial nerve facilitated the H-reflex in spastic patients slightly after about 300 ms (up to 113%), following an early strong facilitation (10 ms) and a longer lasting depression (20–200 ms). Similar postinhibitory facilitation was obtained in spastic patients after ipsilateral stimulation of the plantar surface and after direct stimulation of the dorsal columns. Conditioning by contralateral stimuli of the posterior tibial nerve caused a slight late facilitation in both normal and spastic patients.

This late facilitation did not correlate significantly with the severity of spasticity, but it was more pronounced in cerebral pyramidal lesions than in spinal ones. It is assumed that this postinhibitory facilitation is probably generated as a spinal rhythm, similar to the clonus, and that it is modulated from supraspinal structures.

Key words: H-reflex recovery curves – Postinhibitory facilitation – Pyramidal spasticity.

Zusammenfassung. An 18 Normalpersonen und 26 Patienten mit pyramidaler Spastik (8 spastische Spinalparalysen, 6 spastische Hemiparesen, 12 spastische Spinalläsionen) wurden die H-Reflex-Amplituden des M. triceps surae nach N. tibialis-Reiz und verschiedenen elektrischen Vorreizen registriert.

Der H-Reflex wird bei pyramidaler Spastik durch motorisch eben unterschwellige Tibialis-Vorreize nach 300 ms leicht gebahnt (bis 113%), nachdem

* Supported by Sonderforschungsbereich Hirnforschung und Sinnesphysiologie (SFB 70) der Deutschen Forschungsgemeinschaft (DFG) und Bundesminister für Arbeit und Sozialordnung, Bonn-Bad Godesberg

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0003/9373/80/0228/0197/$ 01.60

die erste starke Bahnungsphase (10 ms) und die folgende Depression (20–200 ms) abgeklungen sind. Ähnliche postinhibitorische Bahnungen finden sich bei Spastikern nach ipsilateraler plantarer Hautreizung und direkter Hinterstrangsreizung. Nach kontralateraler Vorreizung des N. tibialis posterior haben Gesunde und Spastiker eine mäßige H-Reflex-Bahnung zwischen 50–300 ms.

Diese späte Bahnung korreliert nicht mit dem Schweregrad der Spastik, doch ist sie bei cerebralen Pyramidenläsionen deutlicher als bei spinalen Läsionen.

Es wird angenommen, daß die postinhibitorische Bahnung nicht durch supraspinale long-loop-Reflexe, sondern ähnlich wie der Klonus durch einen intraspinalen Rhythmus entsteht, der von supraspinal gebahnt und moduliert wird.

Schlüsselwörter: H-Reflex – Konditionierungskurven – Postinhibitorische Bahnung – Pyramidenbahnspastik.

Introduction

Conditioning the H-reflex by a preceding stimulus of the posterior tibial nerve results in a recovery cycle showing an early facilitation (0–25 ms), followed by a depression (25–75 ms) and a second late facilitation between 75–300 ms [13]. The origin of this late excitation still remains controversial. It was interpreted either as due to a brain stem or cortical long loop reflex or to propriospinal mechanisms, activated either by I a afferents or cutaneous nerve fibers [19, 23, 25, 28].

H-Reflex recovery curves are different in normal persons and patients with alteration of muscle tone, as in spasticity and extrapyramidal motor disorders [6, 10, 13, 14, 20, 26, 30, 31]. Late facilitation in the excitability profile between 200 to 350 ms has been especially discussed as a significant manifestation of spasticity [15, 18, 23, 26].

The following study reevalutes the significance of the late facilitation in the H-reflex excitability cycle for spasticity with respect to the different degrees of severity and the influence of localization of the pyramidal lesion. It should be noted that the term 'pyramidal' is used for its clinical connotation and not for the precise anatomical delineations of the lesions.

Subjects and Methods

Of the 44 persons participating in the study, 18 were healthy adults (mean age 28 years) with no history or clinical findings suggestive of neurological disorder, and 26 were patients with pyramidal lesions of different etiology. The recordings were performed on 12 patients with supraspinal lesions (8 spastic spinal paralysis, mean age 45 years; 6 hemiparesis due to cerebrovascular lesions, mean age 45 years) and 12 patients with spinal lesions (1 meningocele, 17 years old; 1 cervical myelopathy, 38 years old; 1 lymphocytic myelitis, 30 years old; 1 spinal angioma, 16 years old; and 8 spinal forms of multiple sclerosis, mean age 42 years).

A dorsal column stimulator was implanted at the level of the middle thoracic cord to reduce the amount of spasticity in two patients with spastic spinal paralysis [27]. Here single shocks of 0.2 ms were used as a conditioning stimulus.

Patients without ankle jerks or other evidence of peripheral neuropathy were not included. After a brief orientation about the purpose of the study the subjects lay prone and relaxed on a padded testing table, the knees were flexed dorsally to an angle of 30°, the ankle joint to 110°, with unrestricted movement of the feet.

The percutaneous electrical stimuli (1 ms rectangular pulses) to the posterior tibial nerve in the popliteal fossa were delivered by a bipolar stimulating electrode fixed by an elastic tape. Stimuli were generated by a Tönnies stimulation unit type D in series with two constant current stimulus isolation units.

Placement of the electrodes was considered adequate if a threshold stimulus was eliciting an H-reflex with a direct M-response as small as possible. The reflex responses were recorded by two silver disk surface electrodes, the active one placed over the soleus muscle with the reference electrode taped on the Achilles tendon. Both electrodes were each applied to a preamplifier and the potentials recorded on a cathode ray oscilloscope [9].

The conditioning stimulus of the posterior tibial nerve was adapted to threshold strength for the H-reflex, the intensity of the test stimulus was adjusted to obtain half maximum amplitudes of the reference H-reflex.

For cutaneous conditioning stimulation in the area of the tibial nerve, the electrodes were placed on the plantar surface of the foot, the rectangular impulses of 1 ms were below painful sensation.

The interval between the paired shocks was 0,1 to 0,2 Hz, the interpulse interval (IPI) varied 1–3000 ms. At each IPI 8 reflex responses were averaged by a 200-point averager. After three measurements, controls were repeated.

Statistical Methods. The results were expressed by the mean relative amplitude ± standard deviation for a test reflex equal to 100%. Comparison between two groups of data was made for the stimulation interpulse interval of 300 ms, using the Student's *t*-test. As level of significance, $P < 0.01$ was chosen, and the F-ratio was used for analysis of variance.

Results

Recovery Curves in Healthy and Spastic Persons. The H-reflex recovery profile in spastic persons shows an increase in both facilitating periods and a slightly minor depression as compared to normals (Fig. 1). The initial facilitation 2–15 ms after the conditioning shock has been noted to be unstable with a high standard deviation and is therefore omitted in the present study. This early facilitation is followed by a period of depression at 20–150 ms. As the inhibitory period is terminated, the H-reflex is recovering again with its maximum at 250 ms. A statistically significant difference between normals and spastic persons ($P < 0.01$) was found between 200 and 350 ms, with best statistical values obtained at 300 ms.

EMG-recordings over the triceps surae muscle after a single stimulus to the posterior tibial nerve in case of spasticity sometimes reveals a late muscle reflex activity with identical latency to the second facilitation of the recovery profile (see insert in Fig. 1). A second, very slight EMG response was recorded after 400–450 ms in two patients (Fig. 2f).

Localization of Pyramidal Lesions. The spastic patients were grouped according to the level of the lesion causing spasticity. While there was no statistical difference in H-reflex recovery curve between spastic spinal paralysis and hemiparesis, the late facilitation in the curves of paraspastic patients was significantly ($P < 0.01$) lower compared to supraspinal spasticity (Fig. 2a and b).

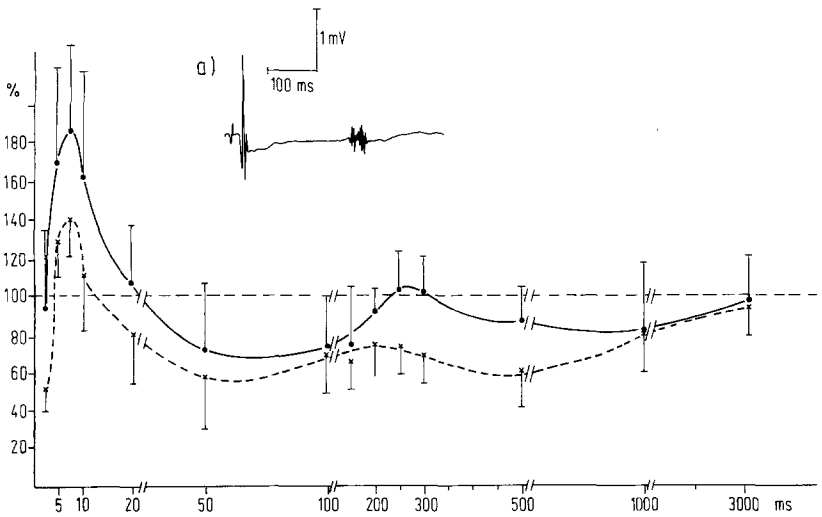

Fig. 1. Recovery curve of the half-maximum H-reflex conditioned by a just suprathreshold stimulus to the posterior tibial nerve. Each graphic point represents the average of eight reflexes with standard deviations normalized to control and evaluated at each interval represented: x--------x healthy adults; ●———● total group of spastic patients. The insert (**a**) shows an EMG recording over the triceps surae muscle after a single stimulus to the posterior tibial nerve in a spastic patient

Severity of Spastic Syndromes. For both supraspinal and spinal lesions, spasticity was grouped according to severity following clinical parameters. Clearly raised muscle tone with increased reflex activity corresponded to a high degree of spasticity; elevated reflex activity only with positive sign of Babinski, but without manifest elevated muscle tone indicated only slight spasticity. The corresponding recovery curves for the groups of different severity in supraspinal lesions differed, but showed no statistically significant difference in the range of 200 to 300 ms. There was also no statistically significant difference in the severity of spinal lesions (Fig. 2a and b).

Conditioning stimulation of the contralateral posterior tibial nerve at threshold strength facilitated the H-reflexes up to 125% in the range between 50 and 300 ms with maximal values at 150–200 ms. But there was no difference between normal and spastic persons (Fig. 2c).

Conditioning stimulation to the plantar surface of the ipsilateral foot with intensities just below painful sensation was followed by a slight facilitation of the test reflexes between 50 and 500 ms with a maximum at 250–300 ms only in the spastic group (Fig. 2d).

Conditioning stimulation of the dorsal columns by a single pulses of 0.2 ms by chronically implanted DCS-electrodes at midthoracic levels caused an early depression (30–70 ms) followed by a facilitation between 120–350 ms, maximally at 150–200 ms. This facilitation depended on DC-stimulus intensity, and was only found if the stimulus produced strong paresthesias below the segment of stimulation (Fig. 2e).

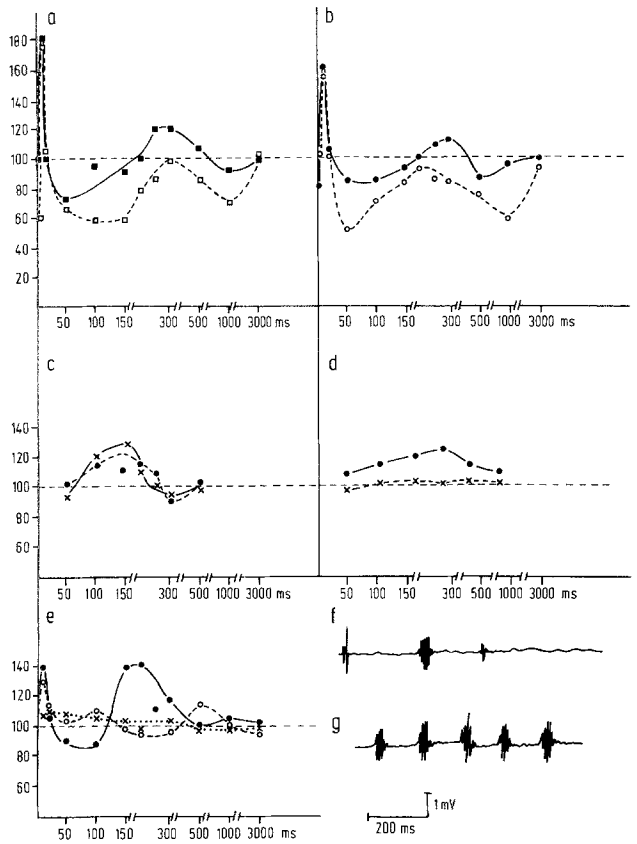

Fig. 2. **a** Comparison of recovery curve in *severe* supraspinal (■———■) and spinal (□--------□) spasticity. **b** Comparison of recovery curve in *slight* supraspinal (●———●) and spinal (○--------○) spasticity. **c** Recovery curve after contralateral conditioning stimulus in normal (●--------●) and spastic (x———x) persons. **d** Recovery curve after plantar surface stimulation in normal (x--------x) and spastic (●———●) persons. **e** Recovery curve after a dorsal column stimulus of different intensity: (x· · · · · x) first, just suprathreshold paresthesia in thoracic spinal region. ○--------○ local paresthesia of medium intensity, ●———● strong paresthesia reaching from thoracic area to the calf muscle. **f.** Long-lasting direct recording of H-reflex in a patient with a spinal angioma in D8/9 and severe spasticity of both legs. Polyphasic potentials can be recorded about 200–250 ms after stimulation. A second period with very slight polyphasic potentials can be recorded about 400 ms after stimulus. **g** Registration of the same patient's clonic jerks

Discussion

The main finding of our study is that late excitability depends less on the intensity of spasticity than on the localization of the pyramidal lesion causing it. The differences are significant when comparing supraspinal lesions to spinal ones. Facilitation is more pronounced in supraspinal spasticity and is independent of different localization. Similar facilitation is evoked by ipsilateral nonpainful cutaneous stimulation of the plantar surface in spastic patients and was also described in normals [21]. A contralateral tibial nerve conditioning stimulus was followed by an earlier facilitation without difference between healthy and spastic

persons. A single shock conditioning of the dorsal columns was also followed by late excitation, but about 50 ms earlier than after peripheral nerve conditioning.

Since the conditioning stimulus did not evoke any movements in our experiments late excitation of the test reflex is due to the electrically elicited afferent volley and not to the mechanical movements of the foot as originally suggested [2]. From the parameters of nonpainful conditioning stimulation it can be excluded that the afferent input itself reflects the late response via a segmental spinal reflex [7]. This interpretation is in accordance with the effects of dorsal column stimulation.

According to different authors a conditioning tap to the Achilles tendon leads only to a long lasting depression up to 1000 ms without any facilitation [12, 18]. This finding was used as a strong argument against participation of Ia afferents in late facilitation. As first shown by Gassel et al. [8] and confirmed in this study, late facilitation in the recovery cycle may be evoked by fast cutaneous afferent input, arising from the same segment [1]. Nevertheless, a participation of muscle afferents, as demonstrated by Szumski et al. [24], cannot be ruled out by our data.

Taborikova et al. [23] assumed that late facilitation was due to long loop reflexes activated by Ia afferents via cerebellar or vestibulospinal pathways. Moreover it could be shown by cooling experiments in monkeys that cortical motor areas are essential for late excitation in the H-reflex recovery curve [28].

On the other hand, Shimamura et al. [22] calculated from their data that a long loop reflex for the hind limb in man with a pathway up to the brain stem or even higher would have a latency of 60–85 ms. These suggestions were confirmed later on [3, 4, 16, 17]. In our opinion the late excitation seems inconsistent with the proposed bulbospinal reflex because the latency seems to be too long for a bulbospinal long loop reflex.

Late facilitation is found, though less pronounced, in complete transverse spinal lesions of humans [15] and in the spinalized cat [29] too. Late facilitation is lacking during the phase of spinal shock one to four days following spinal cord transsection [5].

From our data it seems probable that cutaneous or muscle afferent impulses activate a propriospinal oscillating reflex loop. In 1941 Jung [11] postulated a spinal automatic rhythmicity by interneuronal circuits as a common source of clonus and tremor rhythms. There is no other known sensory spinal mechanism that produce such delayed facilitation following an afferent volley. Late facilitation in spastics may be caused by tonic disinhibition of the propriospinal system after supraspinal lesions. Vestibulospinal pathways activating extensor tonus may facilitate the extensor motoneurons by disinhibition. However, cerebral long loop reflexes for the activation of these spinal mechanisms can only be excluded in total spinal lesions [15].

The latency of the late activation is longer than the intervals between reflex clonus, obtained in relaxation (Fig. 2f and g). The difference between the intervals of clonic jerks (120–180 ms) and the latency of late excitation (200 ms) may be explained by tonic peripheral input to the spinal cord [2].

In summary, we assume that late facilitation at about 200–300 ms is due to an interaction of afferent activity either of cutaneous or muscular origin with

endogenous propriospinal reflex oscillations which may be facilitated and modified by supraspinal mechanisms.

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Received June 18, 1979