Conditioning of the H reflex by stimulation of the posterior tibial nerve in Parkinson’s disease

P. Martinelli and P. Montagna

From the Institute of Neurology, University of Bologna, Bologna, Italy

Summary The excitability curve of the H reflex conditioned by stimulation of a mixed nerve was studied in eight Parkinsonism patients, before and after L-dopa therapy. There was no significant variation between the two curves. However, there was a reduction of the normal early inhibition of the H reflex conditioned by exteroceptive stimulation. This indicates the presence of alterations in the organisation of the reflex pathways at a spinal level in this disease.

The H reflex has been used to evaluate variations in excitability of soleus motoneurones (Magladery et al., 1952; Paillard, 1955). Several authors have demonstrated that cutaneous stimulation affects the excitability of soleus motoneurones (Bathien and Hugon, 1964; Hugon and Bathien, 1967; Gassel and Ott, 1970; Hugon, 1973). Delwaide (1971) showed that non-nociceptive stimulation of the posterior tibial nerve in normal subjects causes a marked reduction of the amplitude of the H reflex, maximal at about 100 ms of delay between the two stimulations. In lesions of the pyramidal tract a marked facilitation of the H reflex is present with the same time delay of the inhibition which is observed in normal subjects. Using a different technique, Pierrot-Desileigny et al. (1973) showed that changes of H reflex induced by cutaneous stimulation are subject to supraspinal control. This type of excitability curve is thus able to demonstrate abnormal features in spasticity.

In this study, we have used the same techniques to see if there are modifications of the excitability curve in patients with Parkinsonism. Conditioning of the H reflex by percutaneous stimulation of the posterior tibial nerve makes it possible to study not only alterations in motoneurone excitability found in Parkinsonism, but also possible variations resulting from therapy.

Patients and methods

Eight patients, seven men and one woman, were studied. All were affected by Parkinson’s disease and any other extrapyramidal syndrome was excluded. The age range of the patients was 45–67 years, and none of them was receiving therapy at the time of study.

Percutaneous stimulation was applied to the posterior tibial nerve at the ankle by a painless train of electrical stimuli at 300 Hz, each pulse lasting 20 ms, which evoked the “reflexe polysynaptique précoce du soleaire” (RPPS) described by Delwaide (1971). The effects of repetitive stimulation of the reflex were studied at various frequencies from 0.1 to 5 Hz.

The H reflex was then obtained. The methods for eliciting and recording the reflex, the positioning of the patient, the relationships of joints, and the repetition rates of stimuli were all chosen according to the suggestions of the Brussels Committee (1973). Amplitude of Hmax/2 was used in order to show a possible inhibition or facilitation of the reflex (Paillard, 1955). At least five basal values of the Hmax/2 amplitude were obtained. Amplitude variations of Hmax/2 were studied with various delays between the two stimulations, and for each delay, at least 10 values were recorded. Amplitudes of the H reflex action potential were then measured peak-to-peak on photographic paper.

The same procedure was repeated after the patients had received L-dopa alone or L-dopa plus carbidopa in such an amount as to give satisfactory clinical results. The percentage variations of the conditioned H reflex were then plotted as a function of the time delay between the two stimuli for each patient and as a single curve for the whole group.
Results

BEFORE L-DOPA THERAPY

In all patients studied it was possible to record from the soleus muscle the RPPS (Delwaide, 1971) with percutaneous stimulation of the posterior tibial nerve at the medial malleolus. No significant difference was found in the latency, amplitude, and shape of the reflex between normal subjects and Parkinsonism patients. Reduction of habituation of this reflex with increasing rates of stimulation (over 0.2 Hz) was easily demonstrated in these patients. Sometimes a reduction in latency of a few milliseconds was observed with repetition (Fig. 1).

The excitability curve of the H reflex, plotted as a function of the time delay from the conditioning stimulation of the posterior tibial nerve, showed a pattern which differed only slightly in different patients. Making allowance for individual variations, a slight increase of the H reflex amplitude could be observed, with time delays of 30–80 ms between the two stimulations (Fig. 2).

AFTER L-DOPA THERAPY

L-dopa, alone or associated with carbidopa, was administered to the patients, and the electrophysiological examination was repeated a few days after a distinct and steady clinical improvement had been achieved.

The excitability curves of the H reflex amplitude of the patients showed either a pattern similar to that observed before therapy, or a slight increase of the H reflex amplitude, more marked at 60–80 ms of interval between the two stimulations.

When plotted as a single curve for the whole group of patients, the curve showed a pattern almost the same as that observed before therapy. Statistical comparison (Student’s paired t test) did not disclose any significant variation between the two excitability curves.

Discussion

An extensive electrophysiological investigation of reflex behaviour in Parkinson’s disease was carried out to elucidate the pathophysiology of this disease and possibly to establish a method for clinical use.

In Parkinsonism patients the amplitude of the tendon jerk is normal. The H reflex, its recruitment curve, and the Hmax/Mmax ratio do not show any significant difference from normal subjects (Angel and Hofmann, 1963; Dietrichson, 1971; Krassoievitch and Tissot, 1971). Both the behaviour of the tonic vibration reflex alone (Hagbarth and Eklund, 1968) and the inhibition of the H reflex by vibration were described as normal by Delwaide (1971). The proprioceptive silent period was described as increased in Parkinson’s disease (Liberson, 1962; Arrigo, 1963) or, on the contrary, reduced (Hufschmidt, 1959; Hofmann, 1962). Higgins and Lieberman (1968) remarked that methods of eliciting the proprioceptive silent period lacked adequate standardisation. The exteroceptive silent period is markedly reduced in Parkinsonism (Delwaide et al., 1974). Thus these procedures hardly help to reveal any abnormality in motoneurone excitability in Parkinsonism.

A different method, involving the study of the recovery cycle of the H reflex, showed variations...
Conditioning of the H reflex in Parkinson's disease

Fig. 2 Excitability curve of the H reflex conditioned by stimulation of the ipsilateral posterior tibial nerve before therapy. Abscissa: delays (5, 10, 20, 40, 60, 80, 100, 150, 300, 500, 700, 800, 1000, 1500 ms) between the two stimulations (logarithmic scale). Ordinate: percentage variations of test H reflex. Dotted lines show SD. A slight facilitation or reduction of normal inhibition is observed with 30–80 ms of delay between the two stimulations.

from the normal pattern such as a facilitation of the so-called phase 4 and a reduction of the inhibition of phase 5 (Takamori, 1967; Yap, 1967; Zander Olsen and Diamantopoulos, 1967; Krassoievitch and Tissot, 1971). The effect of L-dopa therapy on the recovery cycle of the H reflex was studied by Krassoievitch and Tissot (1971) and by Sax et al. (1977), who showed that H reflex variations were reduced by L-dopa, though excitability curves did not return to normal values.

In Parkinson's disease, abnormalities are also present in the organisation of polysynaptic reflexes. The reduction of habituation of the blink reflex and of polysynaptic reflexes of the lower limbs has been described (Rushworth, 1962; Delwaide et al., 1974), and abnormal co-contraction of the antagonistic muscles during the reflex responses in the lower limbs has also been described by Delwaide et al. (1974).

Messina et al. (1972) have studied the effect of L-dopa and amantadine on the habituation of the blink reflex, and Penders and Delwaide (1971) have established an index of habituation of this reflex which can be used to evaluate the degree of akinesia and the effect of therapy.

The recovery cycle of the second component (R2) of the blink reflex is altered in patients with Parkinsonism, while the first oligosynaptic component (R1) behaves normally (Kimura, 1973). It was inferred that this finding indicated an inhibition at the interneurone level in Parkinson's disease (Kimura, 1973).

Amplitude variations of the H reflex in normal subjects as a result of a stimulation applied to the posterior tibial nerve are thought to be due to the function of chains of interneurones within the spinal cord (Delwaide, 1971). The results of our study indicate that, as a further abnormality, there is a reduction of the normal early inhibition of the H reflex conditioned by exteroceptive stimulation. This can be taken as additional proof of the presence of alterations in the organisation of the reflex pathways at a spinal level in Parkinson's disease, especially in the functioning of interneurones.

L-dopa therapy did not, in our study, result in normalisation of the excitability curve. A tendency to show nearly normal values as a result of therapy was described for habituation of blink and polysynaptic lower limb reflexes (Penders and Delwaide, 1971; Messina et al., 1972; Delwaide et al., 1974) and for the H reflex recovery cycle (Krassoievitch and Tissot, 1971; Sax et al., 1977). However, we believe that each test of reflex behaviour explores different physiological systems (Delwaide and Martinelli, 1978). We think that the method we have used tests different systems from the others described and that this can explain the different effects of L-dopa therapy in our study as compared to the results obtained by other workers.
We would like to thank Dr P. J. Delwaide for his helpful criticism and revision of the manuscript.

References


Conditioning of the H reflex by stimulation of the posterior tibial nerve in Parkinson's disease.
P Martinelli and P Montagna

*J Neurol Neurosurg Psychiatry* 1979 42: 701-704
doi: 10.1136/jnnp.42.8.701

Updated information and services can be found at: 
[http://jnnp.bmj.com/content/42/8/701](http://jnnp.bmj.com/content/42/8/701)

**Email alerting service**
Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

**Notes**

To request permissions go to:
[http://group.bmj.com/group/rights-licensing/permissions](http://group.bmj.com/group/rights-licensing/permissions)

To order reprints go to:
[http://journals.bmj.com/cgi/reprintform](http://journals.bmj.com/cgi/reprintform)

To subscribe to BMJ go to:
[http://group.bmj.com/subscribe/](http://group.bmj.com/subscribe/)