Muscle silent period in Parkinson's disease

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SUMMARY The muscle silent period was measured in 11 patients with moderate to severe rigidity associated with Parkinson's disease. The determinations were made under conditions of maximum disability for each patient, since all medications had been withdrawn before testing. The duration of the EMG silence, produced by small and large electrical twitch contractions of the adductor pollicis muscle, fell within a range of values previously determined for normal individuals. Major alleviation of the rigidity and bradykinesia with chronic oral L-dopa therapy was not accompanied by any change in the silent period. It was concluded that in untreated Parkinsonism, and also after its treatment with L-dopa, the functioning of the muscle spindles and local inhibitory reflexes remains normal.

Previous investigators (Angel, Hofmann, and Eppler, 1966), utilizing the unloading reflex, demonstrated that the electromyographic (EMG) silent periods in four patients with Parkinsonian rigidity were similar to those obtained in normal individuals. The present investigation, using 11 patients, demonstrated normal silent periods under conditions which provide additional insight into the reflex behaviour in Parkinsonism. The silent period was obtained by an electrically induced contraction of a single muscle (adductor pollicis). During maximal stimulation the early portion of EMG silence was a manifestation of inhibitory actions, while events related to muscle spindle function were identifiable in the termination of the silence (Granit, Kellerth, and Szumski, 1966; Higgins and Lieberman, 1968a, b).

METHOD

The technique of obtaining the silent period of the adductor pollicis muscle has been described previously (Merton, 1951; Higgins and Lieberman, 1968a, b). Two modifications were made to simplify the recording procedure.

The first modification was that two skin surface electrodes with silver-silver chloride junctions (Beckman Bio-potential electrodes; diameter 2 mm) were used for EMG recording instead of needle electrodes. The active

electrode was placed over the adductor muscle, adjacent to the flexor tendon of the second digit and away from the edge of opponens pollicis muscle. The reference electrode was located on the volar surface of the hand, over the distal head of the third metacarpal bone. The centre-to-centre inter-electrode distance was approximately 5 cm. Identical silent periods were observed in the surface and needle electrode recordings obtained from a normal individual. The second modification was that stimulation of the ulnar nerve at the wrist and recording of EMG and tension responses were accomplished with a Hewlett-Packard Electromyograph (Model 1510A) equipped with a storage cathode ray tube and Polaroid camera.

The ulnar nerve was stimulated at several intensities in order to compare the degree of inhibition associated with maximal and minimal isometric contractions. The stimuli occurred at equal or unequal intervals between 1 and 5 sec. Isometric tension was recorded with a Grass strain gauge (Model FT-10), as previously described. The amplified DC output of this gauge was applied directly to the vertical amplifier of the second recording channel of the electromyograph, by-passing the AC preamplifier.

As in previous investigations of this type, close attention was given to the maintenance of steady tension and EMG levels before each electrical stimulation. Several trial recordings were required to accustom each patient to the procedure of maintaining a steady tension of 500, 1,000, or 1,500 g. The EMG was differentially amplified at a bandpass of approximately 10 Hz to 10 KHz.

RESULTS

Eleven patients were selected from a group admitted to the Yale-New Haven Hospital for initiation of oral L-dopa therapy (Larodopa, Roche brand of

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L-3,4-dihydroxyphenylalanine) in clinical trials of its value in Parkinson's disease. These patients had a distinct predominance of rigidity and bradykinesia over tremor, and they were able to co-operate fully in the procedure. The data were obtained under basal conditions since all medications for Parkinsonism had been withdrawn at least one week before testing, which was attended by a marked increase in rigidity and bradykinesia.

The silent period was measured as the time from the stimulus until the return of EMG activity to the approximate intensity present before the stimulus application. This interval had been previously termed the S-X interval (Higgins and Lieberman, 1968a). Examples of silent period determinations are shown in Fig. 1. The muscular response to the direct motor volley (M wave) and the F wave are readily identified in all records. The timing and amplitude of the various EMG events associated with the twitch were similar to those found in normal individuals (Higgins and Lieberman, 1968a).

The silent period determinations have been summarized in Table 1, along with clinical estimates of the severity and duration of Parkinson's disease. The ranges of mean (117.8 to 138.7 msec) and individual (109 to 142 msec) test values were within the predicted 95% range for normal silent periods (94-144 msec; Higgins and Lieberman, 1968a). The mean square values, as estimates of individual variance, had a similar range in the patient and normal groups.

The effect of L-dopa on the silent period was assessed in five patients, in whom testing was carried out between one and 12 months after institution of therapy. Each patient was receiving between 2 and 5 g L-dopa daily, and each had experienced a marked reduction in rigidity and bradykinesia. As shown at Fig. 1B, there was no change in the duration of the patient's silent period after the relief of rigidity with L-dopa. The M wave, F wave, and the burst of 'voluntary' activity in the middle of the silent period were like those found before treatment.

In order to test the possibility that the potential for development of central inhibition might have changed either in Parkinson's disease or after L-dopa therapy, small stimuli were applied to produce minimal twitch contractions. Ordinarily, reduction of stimulus strength is accompanied by less inhibition and a filling-in of the first half of the silent period with EMG potentials (Merton, 1951; Higgins and Lieberman, 1968b). The same effect was observed among these patients, both before and after L-dopa treatment (Fig. 1C). Therefore, there was no evidence of a change in the status of local reflex inhibition during the course of treatment and the reduction of rigidity.

FIG. 1. Silent period before and after L-dopa therapy. A. Associated with marked rigidity before treatment. Single response; maximal stimulation. B. After one month of L-dopa therapy. Five superimposed consecutive responses, maximal stimulation. C. After L-dopa therapy, but with small twitch, showing filling-in of early part of silent period. Five consecutive responses. Time base mark: 40 msec. Tension calibration mark: 1.0 kg. Tension base line: horizontal broken line indicates 1.0 kg. EMG calibration mark: 0.2 mV. All data from the same patient.

TABLE
SUMMARY OF SILENT PERIOD DETERMINATIONS

Patient	Age (yr)	Sex	Duration of disease (yr)	Rigidity	Rest tremor	Mean S-X msec	s ^a	Number (determinations)
K.D.	46	M	7	+++	+	138.7	3.7	15
J.R.	75	F	12	++	+	125.6	101.4	26
F.S.	75	M	6	++	++	121.3	82.0	40
A.M.	68	M	3	+++	++	128.3	35.8	37
K.L.	66	F	3	++	+	120.0	9.1	36
R.A.	50	F	8	+++	+	117.8	10.7	20
R.D.	48	M	26	+++	++	125.8	9.1	12
J.E.	58	M	2	+++	++	121.3	25.3	21
L.S.	56	M	15	+++	+	123.6	24.5	12
E.P.	62	F	11	+++	0	132.5	41.9	26
G.T.	45	M	2	++	0	123.9	26.2	29

Rigidity and rest tremor are graded according to their intensity in the left upper extremity, since the left adductor pollicis was tested: 0, none; +, trace to mild; ++, moderate; +++ marked.

s^a, mean square, expresses the variance encountered for each sample mean.

DISCUSSION

The present findings corroborate the silent period determinations obtained with the unloading reflex (*Entlastungsreflex*) in Parkinson's disease (Angel *et al.*, 1966). Both studies show that the EMG silence is within the normal range. The present investigation, carried out in a larger number of patients, also eliminates the possibility of a subtle modification of responses in individuals receiving anti-Parkinsonism drugs, since all medications had been withdrawn before testing was conducted. The production of stretch reflexes in antagonists was unlikely in the present investigation because the isometric conditions prevented any appreciable external shortening of the twitched muscle. The electrically induced twitch provided an estimate of inhibitory reflex activity as well as aspects related to the muscle spindles.

Muscle contraction produced by a maximal electrical stimulus of the motor nerve unloads the muscle spindles, causing a period of EMG silence during voluntary muscular activity (Merton, 1951; Granit *et al.*, 1966; Higgins and Lieberman, 1968b). It has been pointed out that silence produced in this manner may also develop from synchronous refractoriness of motor neurones, Renshaw inhibition (antidromic), and post-synaptic inhibition related to the discharge of Golgi tendon afferent fibres (Matthews, 1964; Granit *et al.*, 1966). These inhibitory features, which are most intense early in the twitch contraction, can be reduced or eliminated with weak stimuli. Under these conditions, the later part of the silent period is retained, and it terminates at about the same time as the silence which appears with maximal stimulation (Higgins and Lieberman, 1968b). Therefore, it seems possible to distinguish

between predominantly inhibitory mechanisms contributing to the early portion of the silence and a later mechanism of spindle slackness and re-extension which is influenced by fusimotor control. Both aspects were examined in this study of Parkinsonian rigidity.

Before and after L-dopa treatment, the reduction of stimulus strength below maximal levels was accompanied by the usual filling-in of the early part of the silent period. The later portion of the silence and its termination were like those observed in normal individuals. It is concluded that all aspects of EMG silence produced in an isometric twitch contraction are normal in Parkinsonian rigidity. It is doubtful, therefore, that there has been any significant change in the behaviour of spindles, fusimotor neurones and local inhibitory reflexes in this disorder and in L-dopa therapy. These conclusions confirm those of others working with H-reflexes and tendon jerks (Angel and Hofmann, 1963; Landau and Clare, 1964).

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