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Effect of Stereoencephalotomy on Long-Latency EMG Responses and Motor Control of Arm Movements in Parkinson's Syndrome

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It is well known that stereotactic lesions such as thalamotomy and subthalamotomy suppress tremor and reduce muscle tone. Even increased muscular effort, as for instance in Jendrassik's maneuver, or mental effort no longer induce tremor or rigidity. Immediately following the lesion, the patients show a decreased postural tone, which can be compensated for by visual control. There is no loss of muscular power. When the isometrically innervated flexor muscles of the forearm are passively stretched, the forearm yields to hyperextension on the affected side, when compared with the intact side. A positive rebound phenomenon and general symptoms of hypotonia can also be observed (12).

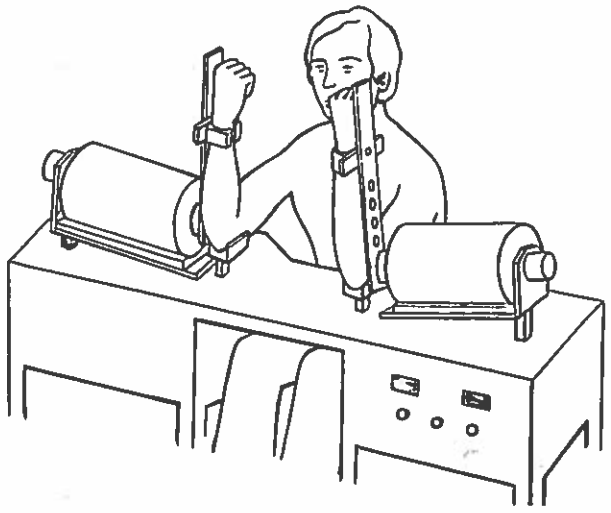

FIG. 1. Experimental setup to investigate muscle response when stretches are applied to the forearms.

To understand these motor disturbances, we studied long-latency EMG responses to muscle stretch, because parkinsonian patients exhibit increased long-latency EMG activity in comparison to normal subjects (7,16,17), and a relationship between long-latency responses and rigidity (10,11). The purpose of the present investigations was to see if the stereotactic intervention affects the increased long-latency responses and if any alteration of EMG responses could be related to the clinical phenomenon of hypotonia. The muscles moving the forearm were chosen because they support balance and skilled movements. Additionally, Parkinson tremor is often observed in muscles of the forearm. Some of the results have already been reported earlier (8).

MATERIAL AND METHODS

Seventeen parkinsonian patients, 14 of them assigned for stereotactic intervention, were included in this study. Fifteen healthy volunteers acted as controls. All patients displayed clear tremor activity and varying degrees of rigidity and akinesia. Control subjects and parkinsonians were requested to compensate a sudden stretch of their isometrically active elbow flexors ("resist") or to follow the displacement ("let go") of their forearms.

Both forearms of a subject were fixed to two levers (Fig. 1) moved by a pair of precision torque motors that add a maximal torque of 6 Nm to the effects of gravity acting on the forearm. In addition, sudden torque pulses (between 0 and 6 Nm, usually lasting 1 sec) could be applied. EMG from brachial muscle was recorded with pairs of wire electrodes, EMG from triceps muscles with surface electrodes. EMG activity together with the mechanical deviation of the forearm (elbow-joint-angle) was stored on magnetic tape for subsequent analysis. EMG activity of eight trials was rectified, averaged, and integrated. The integral value of each single M-response was calculated and normalized to the total integral of all three M-waves. Additionally, in a small group of patients, single-unit recording was done by means of commercially available concentric needle electrodes.

RESULTS

Sudden stretching of isometrically innervated brachial muscle resulted in a segmented EMG response. In control subjects performing the resist task, three bursts were usually observed: M1, M2, M3 (see Fig. 2A). Onset latencies of M1 were 22 ± 2 msec, of M2, 46 ± 6 msec, and of M3, 78 ± 5 msec. The kinesthetic reaction times were always longer than 100 msec. Thus, 100 msec can be used as a reasonable critical value between reflex and voluntary activity in the EMG.

Preceding the operation, more than half of the parkinsonian patients showed a markedly increased long-latency activity when compared to normals (Fig. 2B). In the other cases, the segmented activity did not distribute differently from normal. With reduced innervation, achieved by diminishing the preloads, the long-latency activity markedly decreased in normal subjects. The preloads were also varied in

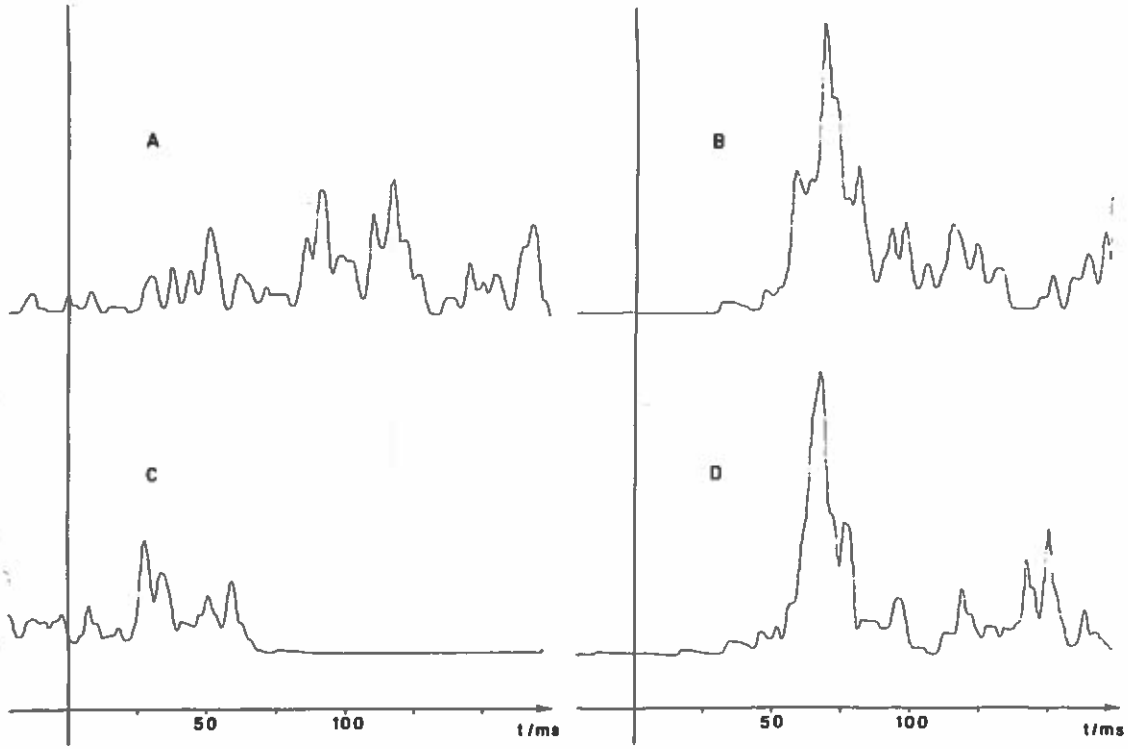

FIG. 2. Rectified and averaged EMG responses (×8) to sudden stretch of the brachial muscle ($t<0$: preload 3.4 Nm; $t>0$: additional disturbance load 3.4 Nm). **Left:** normal subject. **Right:** Parkinsonian patient. **A, B:** resist. **C, D:** let go.

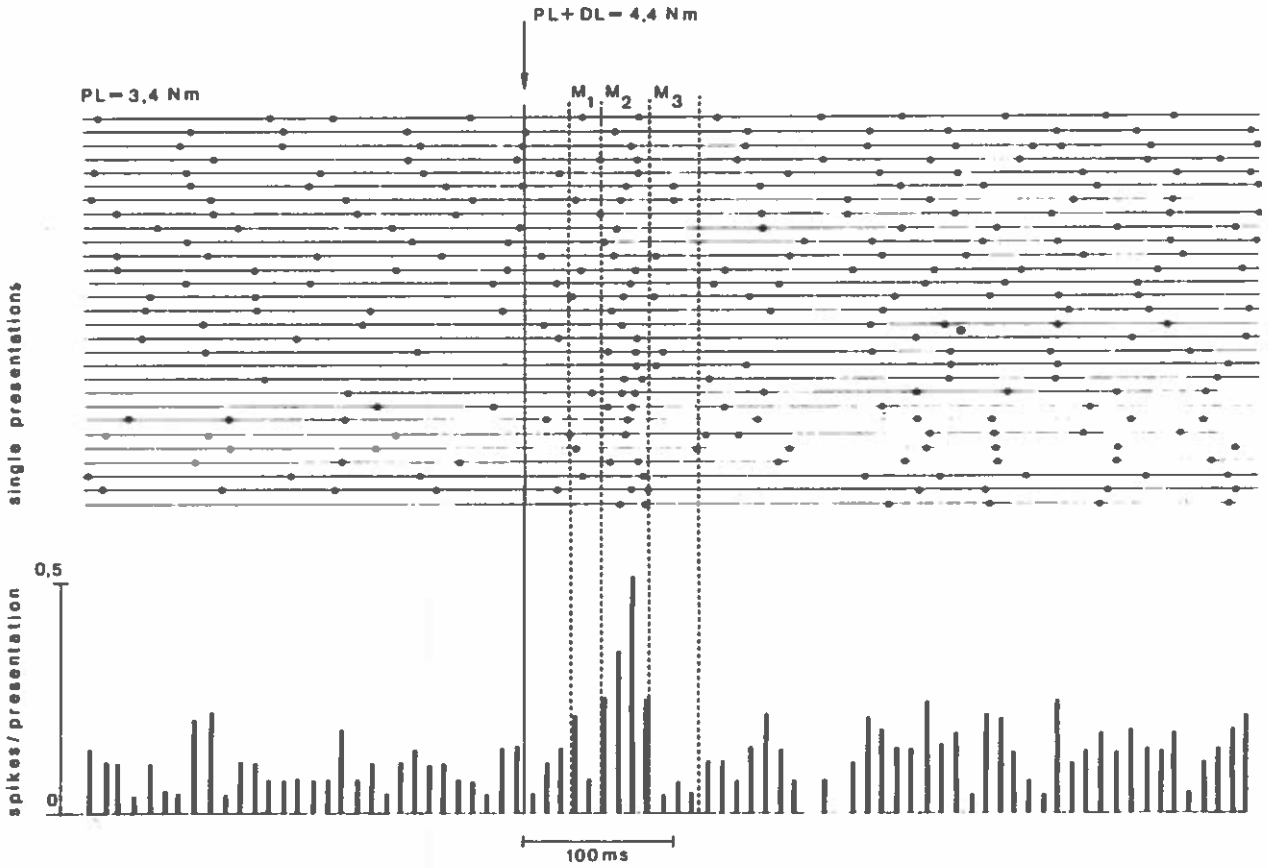

FIG. 3. Top: Discharge pattern of a single motor unit (29 sweeps) from brachial muscle before and during muscle stretch under resist condition (PL, preload; DL, disturbance load). Note the unit increases its discharge rate in the first 80 msec after stretch. **Bottom:** The discharge rate of the unit is expressed as its average firing within 10-msec intervals.

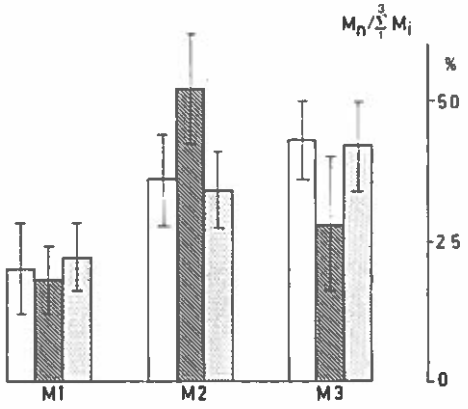

FIG. 4. Synopsis of the integrated EMG activity in normal subjects *(open bars)*, parkinsonian patients before *(hatched bars)*, and after operation *(dotted bars)*. Each M-component is expressed as a percentage of the whole M-activity (M1 + M2 + M3 = 100%).

2 parkinsonian patients. When low background activity was attained, the M2-response still remained sizeable.

The effect of the instructions on the long-latency EMG response by introducing the let-go command was similar to the results described by Hammond (3) in normal subjects, the M2-activity was reduced, M3-response and voluntary activity were absent (Fig. 2C). The long-latency activity in parkinsonian patients, however, was found to be less instruction-dependent (Fig. 2D), as observed earlier (17).

To gain a better understanding of increased long-latency activity in parkinsonian patients, we also recorded single motor unit activity, as shown in Fig. 3. Preliminary studies suggest that tonically firing units can discharge twice within the M2-interval. New units, however, also may be recruited.

Stereoencephalotomy in the caudal subthalamus (7 patients) (1,9) or subthalamotomy combined with a lesion located in the thalamic V.o.p. (7 patients) was performed to relieve Parkinson tremor. After the stereotactic intervention, the M-responses could still be recognized. No changes in onset latencies could be detected, but all patients showed reduced M2-activity (Fig. 4, Fig. 5B), regardless of whether the lesion was restricted to the subthalamus or extended into V.o.p. The reduction is statistically significant at 1% level (*t*-test, maximum test). Postoperative investigations were done 7 to 10 days after stereotactic intervention. The mechanograms revealed an increased displacement and in some patients an overshoot when trying to compensate deflection of the forearm contralateral to the lesion (Fig. 5). The operation had no significant effect on the kinesthetic reaction times (161 ± 50 msec prior to operation and 147 ± 18 msec afterwards). Three patients were examined 1 year following the operation. The observed clinical or electrophysiological phenomena were not different from those obtained 7 to 10 days after stereoencephalotomy.

DISCUSSION

The EMG investigations showed that M2 was preoperatively increased in more than 50% of the parkinsonians, when compared with normal subjects. The result

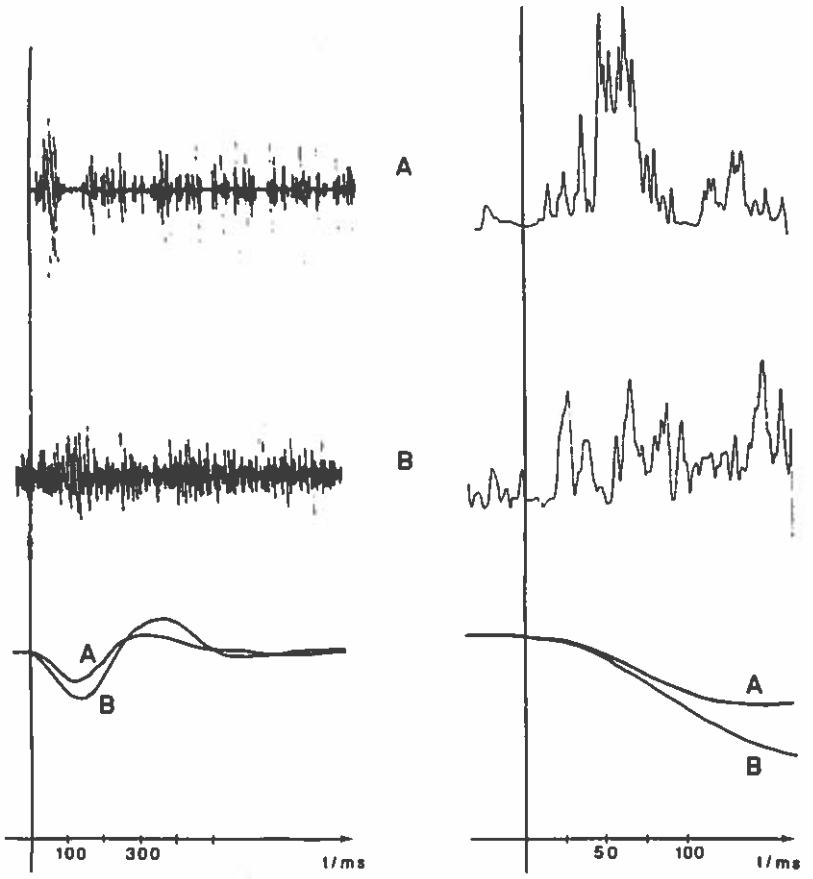

FIG. 5. Electromyographic **(upper and middle traces)** and mechanographic **(lower traces)** recordings before *(A)* and after operation *(B)*. EMG recordings from M. brachialis, mechanical deviation of the forearm recorded at the elbow joints. Note the larger mechanical deflection following operation. **Left:** Single sweep. **Right:** Average of eight sweeps. EMG activity rectified.

of increased long-latency responses is in agreement with earlier studies (7,10,11,16,17). An increased discharge rate of motor units within the M2-interval and recruitment of subliminal fringe motor units could account for this increased M2-activity.

A decrease of M2-response could be observed in all patients after subthalamotomy, regardless of whether the M2 was preoperatively increased or not. As this general effect of M2-reduction was found following thalamotomy and subthalamotomy as well as following subthalamotomy alone, it is suggested that the subthalamic lesion is responsible for the M2-reduction. This decreased long-latency response of the stretched brachial muscle may be the cause of the increased displacement of the affected forearm.

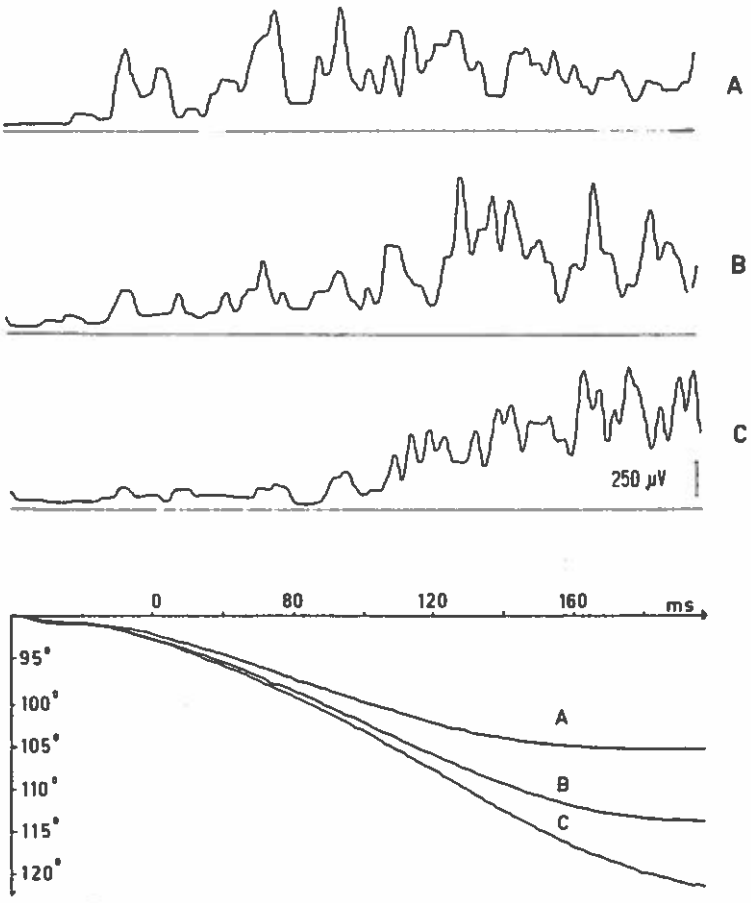

FIG. 6. The effect of differential block of the musculocutaneous nerve (Xylocaine®). **A:** Before. **B:** After 8 min. **C:** After 18 min. Note that all M-components are depressed when the block is effective. This is also reflected in the mechanical traces *A*, *B*, and *C*.

Whether or not the same mechanisms underlie the decrease of M2 and the reduction of rigidity remains to be clarified (10,11). It should be emphasized that M2 is produced under voluntary innervation. Rigidity, however, is characterized by activity at rest, increased myotatic reflex excitability, and shortening reactions, the so-called release phenomenon (6,15).

It has been shown in an earlier investigation (15) that subthreshold repetitive stimulation in the V.o.a. augments the tonic EMG activity. Such an increased tonic EMG activity could be evoked via the α- or γ-route. The γ-system is possibly involved in the fascilitation of the long-latency response, as can be deduced from earlier work using the technique of differentially blocking efferents *(unpublished observations)*. Under differential block, a general reduction of the M-waves, not

of an independent burst, was observed (Fig. 6). When following the lesion only M2 is decreased and M1 unchanged, one could assume that the lesion only affects the static γ-system. The dynamic γ-drive seems not to be primarily involved because the M1, analogous to the T-reflex, is unchanged (13).

It is not known whether the lesion interrupts ascending or descending fiber systems. Fiber tracts passing through the caudal subthalamus and related to motor functions are the dentatothalamic bundle, the pallidothalamic fibers, and the pallidotegmental fiber bundle (2,4,5,18). Which one of these systems is involved in the mediation of the long-latency responses remains to be defined.

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