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Slow Potentials in the Human Subthalamus Associated with Rapid Arm Movements*

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Summary. During stereotactic operations brain potentials were recorded from the zona incerta of Parkinsonian patients. When the patient performed a rapid contralateral elbow flexion in response to a light signal, a slow positive or positive-negative EEG potential preceded the EMG activation by 30–130 ms (average 60 ms) and extended into the biceps activation phase. It was not evoked by the light flash alone or by rapid passive flexion. The potential may be interpreted as a correlate of movement-related activity of cerebellothalamic pathways mediating command signals for rapid arm movements. In support of this assumption, a marked delay of contralateral biceps activation followed coagulation of the recording site.

Key words: Slow potentials – Human subthalamus – Cerebellothalamic connection – Rapid arm movements – Dely after coagulation

Zusammenfassung. Während stereotaktischer Operationen wurde in der Zona incerta von Parkinson-Patienten am Zielpunkt der Koagulation das EEG abgeleitet. Wenn der Patient spontan oder auf Lichtsignal hin eine rasche Beugung des kontralateralen Ellbogengelenkes ausführte, ließ sich in der Subthalamus-Ableitung ein langsames positives oder positiv-negatives Potential registrieren. Dieses begann 30–130 ms (Durchschnitt 60 ms) vor der EMG-Aktivierung des Bizeps und erreichte sein Maximum am Ende der initialen Bizeps-Aktivierung. Das Lichtsignal allein, ipsilaterale aktive oder kontralaterale passive Beugung konnten das Potential nicht erzeugen. Das Potential wird als Korrelat eines Startsignals für rasche Armbewegungen in cerebellothalamischen Bahnen gedeutet. Eine deutliche Verzögerung des Beginns der kontralateralen Bizepsaktivierung nach Koagulation des Ableitortes stützt diese Hypothese.

Schlüsselwörter: Langsame Potentiale – Subthalamus – Kleinhirn-Thalamus-Verbindung – Rasche Armbewegung – Verzögerung nach Koagulation – Mensch

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Introduction

Participation of the cerebellum in initiation of rapid movements has been suggested by von Braitenberg (1961) and Kornhuber (1971) and experimentally demonstrated in the monkey by Meyer-Lohmann et al. (1977). The aim of the present study was to record correlates of movement that precede activity in dentatothalmic pathways in the human thalamus at stereotactic target sites used for the relief of Parkinsonian tremor (Hassler et al. 1970). Further information on the functional significance of these pathways was expected by investigating the effects of coagulation of the target area on rapid arm flexions. The results suggest that cerebellothalamic activity is related to command signals of rapid arm movements.

Methods

Thalamic slow potentials were recorded by an eight-contact straight macroelectrode (contacts with 2 mm separation). Both bipolar and reference recordings were made with a conventional

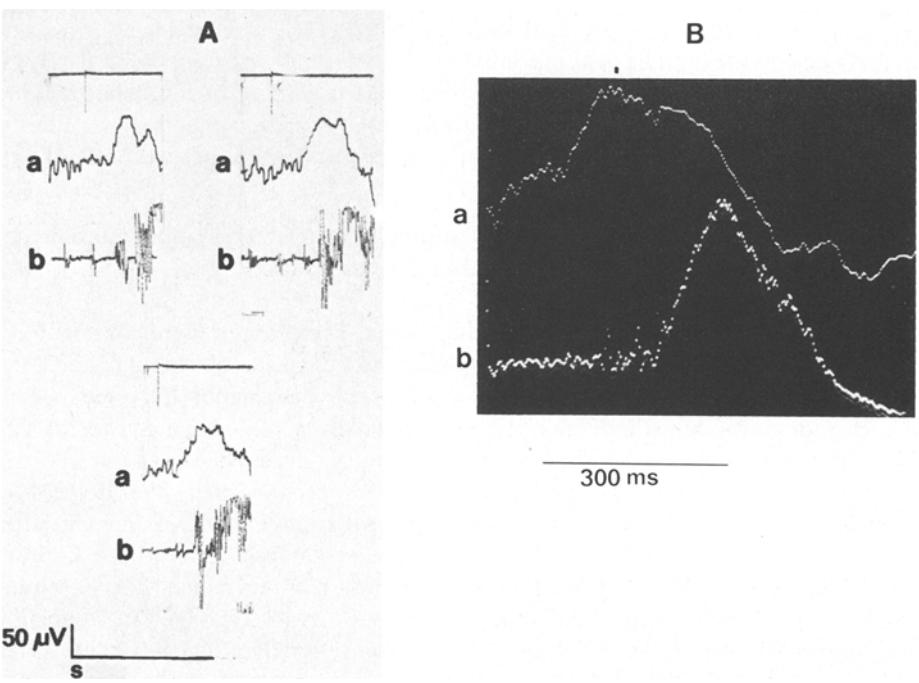

Fig. 1A and B. Subthalmic potentials related to contralateral elbow flexion. **A** Single records of EEG potentials *(a)* preceding contralateral biceps activation *(b)* associated with light triggered (light signal: upper trace) rapid elbow flexions. Reference recording from human zona incerta with 1-s time constant. Positivity upward. **B** Summated subthalamic EEG and EMG potentials (contralateral biceps) associated with rapid elbow flexion of the same patient. Summation of 32 single reactions. The avarager was triggered by a light signal at the beginning of the traces. Recording as in 1A

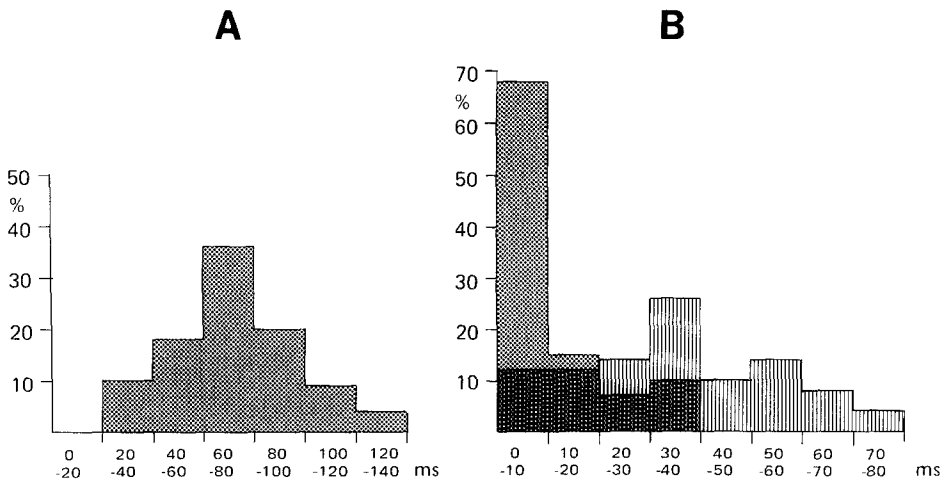

Fig. 2A and B. Timing of subthalamic motor potentials and EMG activation. **A** Distribution of time intervals between onset of the subthalamic movement related potential and onset of contralateral signal triggered biceps activation. Sample: 100 single reactions (as shown in Fig. 1A) obtained from 6 patients. **B** Distribution of the delay of the onset of contralateral (related to the side of stereotactic lesions) biceps EMG activations associated with bilateral signal triggered rapid elbow flexions. Stippled area: before coagulation. Vertical bars: after coagulation of the zona incerta. Each distribution is based on 100 values obtained from 6 patients before and after the stereotactic operation

EEG apparatus with a time constant of 0.5 or 1 s and an upper frequency cut of 70 c/s. Patients were instructed to rapidly flex the right or left elbow in response to a light signal. EMG signals of biceps and triceps muscles were recorded with surface electrodes, amplified by EEG amplifiers, and displayed with the thalamic EEG on a storage oscilloscope whose sweep was triggered by the light signals. Signals were also stored on magnetic tape for further analysis. EEG potential changes and EMG responses were photographed from the oscilloscope or summated anterogradely (trigger: light signal) or retrogradely (trigger signal: EMG activation). The recording site was the zona incerta, the subthalamic target area of coagulation for relief of Parkinsonian tremor (Mundinger 1965) (Fig. 5).

EMG reactions associated with signal-triggered flexions were recorded several times before and after the coagulation to determine latencies of EMG activations or suppressions.

Results

Movement-correlated positive or positive-negative potential changes were obtained in the zona incerta in six of eight Parkinsonian patients. Onset of single slow potentials preceded onset of contralateral biceps activation by 30–130 ms (Figs. 1 and 2). The peak latency (in relation to the command signal) was 200–450 ms, and correlated well with maximal biceps activation or with the end of the initial burst of biceps high frequency discharge (Fig. 3). Amplitudes of single potentials were 20–100 μV. The potential was exclusively associated with active contralateral arm movements. Ipsilateral active or contralateral passive arm flexion did not evoke the potential. The slow potential preceded light-triggered and spontaneous arm flexions. The light signal alone evoked a rather inconspicious and inconstant

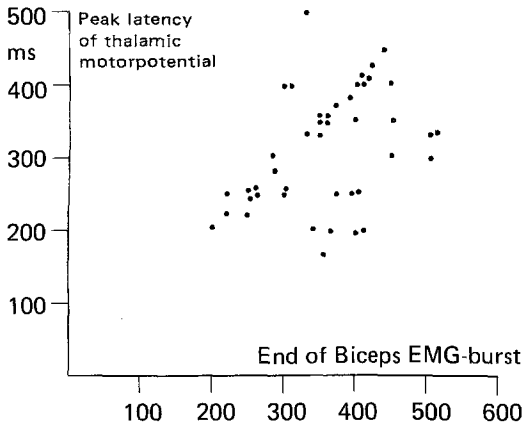

Fig. 3. Correlation between the peak latency of thalamic motor potential and the end of EMG burst, which initiates rapid elbow flexion. *Abscissa:* End of initial biceps EMG discharge (in relation to the light signal). *Ordinate:* Peak latency of thalamic motor potential

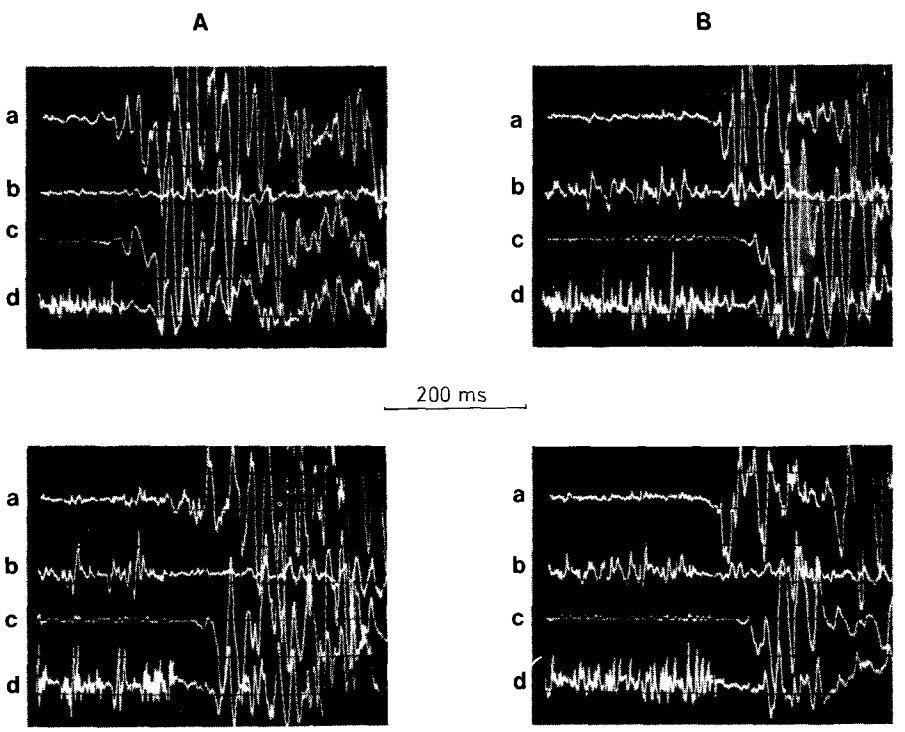

Fig. 4A and B. EMG recordings of ipsilateral *(a, b)* and contralateral *(c, d)* biceps *(a, c)* and triceps *(b, d)* activity, associated with rapid bilateral elbow flexions of the patient whose intraoperative records are shown in Fig. 1. **A** Before coagulation of the zona incerta. **B** 24 h after stereotatic coagulation of the recording site of Fig. 1. There is a marked delay of the reactions of contralateral biceps and triceps

response of shorter poststimulus latency requiring summation of at least 100 reactions for visualization.

Electrical stimulation of the recording site elicited mostly high amplitude 12–14 s beta rhythms, with an occasional true augmenting response in frontocentral derivations of the scalp EEG. Stimulus-evoked gross motor reactions, as observed with low frequency stimulation of the internal capsule, did not occur.

After coagulation of the recording site, onset of contralateral biceps activation and triceps suppression were delayed in rapid bilateral elbow flexions (Figs. 2 and 4B). (Ipsilateral reaction times remained unchanged from preoperative values.) The delay was usually maximal on postoperative day 1 and diminished during the first two postoperative weeks to smaller, albeit still abnormal values. On clinical examination, 80%–100% relief of tremor, discrete hypotonia, and very slight bradydysdiadochokinesia of the contralateral arm were observed. Signs of paresis, ataxia, or dysmetria were absent.

Discussion

In autopsy material from Parkinsonian patients with 100% tremor relief by stereotactic lesions in the region of the zona incerta, Hassler et al. (1970) have

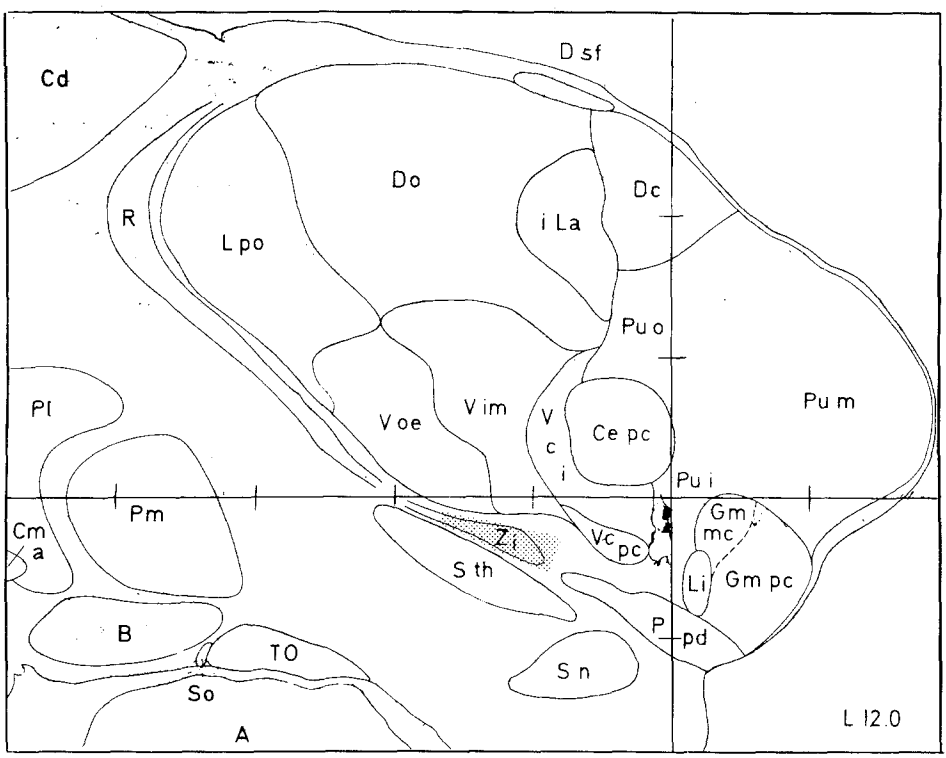

Fig. 5. Sagittal section of the human thalamus 12 mm lateral to the midline (modified from Van Buren and Borke 1972). The target region of coagulation corresponding to the recording sites of motor potentials is indicated by the cross hatched area

demonstrated that the stereotactic lesion interrupts dentatothalmic afferents to the v.o.p. (posterior basal part of VL). It is therefore probable that the movement-correlated potential recorded from the same area, reflects movement-preceding and movement-associated activation of cerebellothalamic fibers and possibly also v.o.p. cells. Since the onset of the potential precedes limb displacement and gamma activation of muscle spindles, it cannot arise from adjacent sensory structures such as the VPL nucleus or the proprioceptive sensory pathway destined for v.i.m. (Oscarsson et al. 1966). The good relation between peak latency of the thalamic potential and the end of the first EMG burst, or the maximum of EMG activation, suggests a close relationship between movement-correlated neuronal activity in the subthalamus and EMG activation. Our potential probably does not originate in the internal capsule, since electrical stimulation of the recording site did not produce gross, stimulus-evoked motor reactions, but did produce EEG responses, which are regarded as a probable stimulation effect of thalamic input structures to motor cortex.

As is to be expected from an event reflecting input activity to the motor cortex, the subthalamic movement-related potential shares common properties with the summated human EEG movement-related negative potential called motor potential (MP) by Deecke et al. (1969). According to these authors, the MP is maximal over the contralateral precentral motor region, precedes finger movement by 60 ms and, as in the records of Gilden et al. (1965), reaches its peak slightly before maximal activation. From subcortical regions Haider et al. (1972) and McCallum et al. (1973) have also recorded slow potentials prior to movement. These, however, could not be correlated with certain cerebellothalamic pathways.

The delay of contralateral EMG activation subsequent to coagulation of the dentatothalmic pathways at the recording site in the human parallels the results obtained in the monkey by cooling the dentate nucleus (Meyer-Lohmann et al. 1977). Together with Thach's (1978) animal experimental evidence, our results provide further support for the theory of Evarts and Thach (1969) and Kornhuber (1971) that cerebellar activity may precede and initiate that of motor cortex in generation of rapid arm movements.

References

Braitenberg V v (1961) Functional interpretation of cerebellar histology. Nature 190:539–541

Deecke L, Scheid P, Kornhuber H (1969) Distribution of readiness potential, pre-motion positivity, and motor potential of the human cerebral cortex preceding voluntary finger movements. Exp Brain Res 7:158–168

Evarts EV, Thach WT (1969) Motor mechanisms of the CNS: cerebrocerebellar interrelations. Ann Rev Physiol 31:451–498

Gilden L, Vaughan HG, Costa LD (1966) Summated human electroencephalographic potentials associated with voluntary movements. Electroenceph Clin Neurophysiol 20:433–438

Haider M, Ganglberger JA, Groll-Knapp E (1972) Computer analysis of subcortical and cortical evoked potential and slow potential phenomena in humans. Confin Neurol (Basel) 34: 224–229

Hassler R, Mundinger F, Riechert T (1970) Pathophysiology of tremor at rest derived from the correlation of anatomical and clinical data. Confin Neurol 32:79–87

Kornhuber H (1971) Motor functions of cerebellum and basal ganglia: the cerebellocortical saccadic (ballistic) clock, the cerebello-nuclear hold regulator and the basal ganglia ramp (voluntary smooth movement) generator. Kybernetik 8:157–162

McCallum WC, Papakostopoulos D, Crow HJ (1973) Relationship between intracerebral slow potential changes in man. Electroenceph Clin Neurophysiol 34:754

Meyer-Lohmann I, Hore I, Brooks VB (1977) Cerebellar participation in generation of prompt arm movements. J Neurophysiol 40:1039–1050

Mundinger F (1965) Die Subthalamotomie zur Behandlung extrapyramidaler Bewegungsstörungen. Dtsch Med Wochenschr 127:2002–2007

Oscarsson O, Rosen J, Sulg J (1966) Organization of neurones in the cat cerebral cortex that are influenced from group I muscle afferents. J Physiol 183:189–210

Thach WT (1979) Correlation of neural discharge with pattern and force of muscular activity, joint position and direction of intended next movement in motor cortex and cerebellum. J Neurophysiol 41:654–676

Van Buren IM, Borke RC (1972) Variations and connections of the human thalamus. Springer, Berlin Heidelberg New York

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