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INFLUENCE OF ACUTE CEREBELLAR LESIONS ON SOMATOSENSORY EVOKED POTENTIALS (SEPs) IN CATS

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We studied the effect of acute unilateral cerebellar lesions on the cerebello--thalamo-cortical projection in cats. The lesions were classified into two groups according to their extent. In group I the lesion only covered the cerebellar cortex, while in group II both the cerebellar cortex and deep cerebellar nuclei were removed. Early (short-latency) and late (long-latency) waves, evoked by an electrical stimulation of a forelimb, were collected contralateral to the stimulated leg hemisphere. Pre- and postsurgery recordings from primary and non-primary (motor and parietal) cortices were compared. Cerebellar impairment had a strong influence on discharges of all the considered cortical areas. Early non-primary and primary responses increased in group I and remained unchanged in group II. Late somatosensory evoked potentials components were suppressed in both groups. An inhibitory influence of the cerebellar cortex on the thalamo-cortical projection was confirmed. Changes within the primary sensory cortex may suggest an engagement of that area in the compensation process of cerebellar dysfunction shortly after cerebellar lesion. An alteration in the unaffected hemisphere activation indicate that the spino-cerebellar and cerebello-cortical inputs, responsible for somatosensory evoked potentials generation, are regulated through contralateral and ipsilateral pathways. These pathways are unmasked by cerebellar lesion.

Key words: *Cerebellar unilateral lesion; Somatosensory evoked potentials (SEPs); Cerebello-thalamo-cortical projection; Early and late SEP components.*

INTRODUCTION

Somatosensory evoked potentials (SEPs) provide the basis of a noninvasive procedure that tests the functional state of the nervous system. The method has been widely introduced into clinical practice. Abnormalities in the main SEP components may assist the determination of the place and size of brain injuries. Experimental surgeries allow the testing of well-localized infarction contributions to SEP disturbances. The aim of the study was to show a variety of

cortical SEP changes after cerebellar lesions of different extent. The functional relationship between cerebellar structures and cerebral cortex have been investigated in men (1, 2) and experimental animals (3—7). However, information about the usefulness of early and late SEP component latencies, and amplitudes recorded from various cortical areas as sensitive indicators of cerebellar dysfunction, is rather limited.

After peripheral stimulation, two kinds of cortical SEPs can be detected: primary short latency responses within somatosensory primary areas (SI, SII, S1A) and nonprimary responses, of similar or longer latency, projected more diffusely and recorded outside mentioned specific sites. It was established that delayed responses in the associated cortex are strongly depressed by barbiturates but preserved, or even enhanced, under chloralose anesthesia (8). The primary, somatotopically distributed potential is usually biphasic for chloralose anesthesia. It consists of an early, surface positive, spikelike waveform component followed by a milder negative one. The afferent projection and its processing within nonprimary cortices, is more complex than within primary sensory fields. It is reasonable to suppose that every nonprimary response represents the composite of a variety of sources, each contributing to the process of elaborating and integrating sensory and other modality information. Except for the primary somatosensory area, we focused on cortical loci where marked cerebellar evoked potentials are observed — including the motor and parietal cortex.

It is apparent that the stimulation of peripheral nerves and the inhibitory properties of the cerebellar cortex exerted on the deep cerebellar nuclei (9—11) evokes complex sequences of excitation and inhibition in neurons of deep cerebellar nuclei (12). Lesion of the cerebellar cortex allows a direct excitatory input to cerebellar nuclei to produce much stronger responses (10) and disturb impulses conducted through the thalamo-cortical linkage. In contrast, the lack of deep cerebellar nuclei results in a reduced cortical activity due to the interruption of excitatory influences of deep cerebellar nuclei exerted on the thalamo-cortical pathway (9, 10). Disorganization of cortical motor processing following acute cerebellar lesions of different extents is unquestionable but the mechanisms still remain unclear.

MATERIAL AND METHODS

Experiments were performed on 7 adult cats weighing between 2.5 and 4.0 kg. The animals were initially premedicated with ketamine (20 mg/kg) and atropine (0.25 μg) injected intramuscularly, then kept under chloralose (50 mg/kg) anesthesia. The trachea and femoral vein were cannulated for respiration and drug infusion, respectively. During the recording session, cats were placed in a Faraday cage and their heads fixed in a stereotaxic frame. To prevent muscle contractions, the animals were paralyzed by periodic intravenous infusion of small doses of

gallamine triethiodide (flaxedil, 20 mg/h) and artificially ventilated during the whole recording session. The concentration of CO_2 in expired gases was measured by the Gould Capnograph IV and maintained at approximately 4.5 %. The body temperature was sustained at around 37°C with a thermostatically controlled heating pad.

The cranium surface was exposed and small holes drilled in the bones over the sensorimotor and parietal cortices for the placement of silver chloride ball electrodes. Responses evoked by an electrical stimulation of a forelimb, were collected through the skull contralateral to the stimulated leg hemisphere. Eight active, gross electrodes were positioned over the sensorimotor and along the suprasylvian and lateral gyri. No electrodes penetrated the dura mater. *Fig. 1* shows the arrangement of the electrode sites on the surface of the skull. The relationship of active electrodes to the underlying cerebral cortex was determined on the basis of a cranium geometry and cranial sutures positions. Coordinates calculated due to a stereotaxic atlas also favored localization of cortical structures. However, according to the anatomical variations, coordinates and craniocerebral landmarks could not be sufficient in identifying the cortical regions in all cases. At the end of the experiment the holes in the cranium were deepened and the active electrode sites marked on the underlying cerebral structures to precise relationship between them. A relatively large number of electrodes let us to locate the largest potentials within considered cortical areas. A reference electrode was inserted in neck muscles. Recordings were performed before and after ablation of the right hemicerebellum. A craniotomy was performed — the dorsal surface of the right cerebellar hemisphere exposed and gently sucked out. The operation was always confined to one side. However, the extension of the lesions differed and the animals were divided into two groups according to the kind of impairment. In the first group, the injury covered the cerebellar cortex only while in the second group the whole hemicerebellum was sucked out. Care was taken not to injure the pons or other extracerebellar structures. Following ablation, the wounded edges were

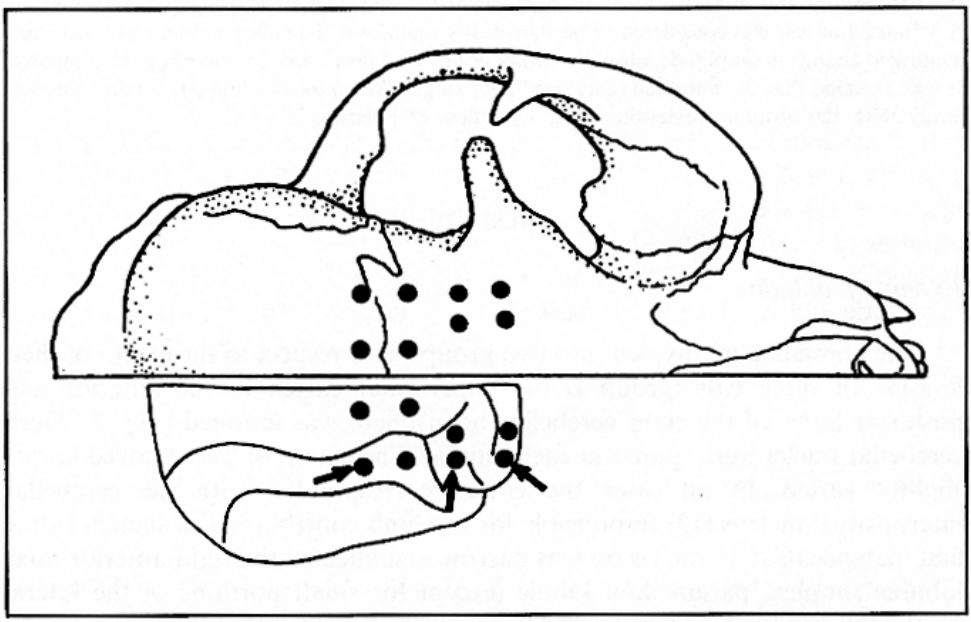

Fig. 1. The arrangement of active electrodes on the dorsal cranium surface (upper part of a figure) in relation with cerebral cortex (lower part of a figure). Dots show approximate electrode sites used in each animal to locate the largest potentials. Pericruciate (MI), somatosensory (SI) and parietal areas were under consideration. Optimal recording sites are indicated by arrows.

covered with spongostan (Ferrosan, Denmark) and the animals allowed to recover from the operation for 0.5—1 h. At the end of the experiments the animals received an overdose of anesthetic and the brains were perfused intravenously with physiological saline, followed by 10% formaldehyde. All solutions were administered at body temperature. The extent of the lesions were verified visually and by a subsequent histological examination. After the brain was removed from the cranium, it was immersed overnight in a cold (5°C) solution consisting of 30% sucrose in 0.1 M phosphate buffer. A frozen cerebellum was cut into 50 μm frontal sections. Finally, the sections were stained by the Nissl technique and mounted on gelatin-coated slides, air-dried, cleared and coverslipped with DPX (Serva).

All surgical procedures were carried out in accordance to Polish regulations (which closely match Convention No. 123 of the Council of Europe) and with the approval of the Ethics Committee at the Nencki Institute.

All recordings were monopolar. The impedances between the active and reference electrodes were less than 5 kΩ. The responses were always evoked by electrical stimuli delivered through a pair of fine steel needles stuck into the skin of the forelimbs. Stimuli, applied to the right or left paw pads, were square-wave constant-current pulses of 140—250 μA, 0.5 ms duration with a repetitive rate of 0.2 — 0.5 Hz. A total of 100 sweeps were averaged. Two consecutive averages were obtained for each sample in order to clearly identify time-locked responses from random activity and to assess response variability. The collected signals were transmitted to a Grass model 12 Neurodata Acquisition System for amplification and filtering. The time constant for recordings was 0.1 s and the high frequency filter settings for all channels was 1 kHz. The amplified signals were digitized with a sampling rate of 2 kHz and stored for off-line analysis. Primary and nonprimary responses, collected contralaterally to the stimulated forelimb, were analyzed statistically. Latencies-to-peak and amplitudes between baseline and positive or negative peaks recorded before and after the ablation were calculated and compared using the ANOVA test. A 3 % level or less was considered to be statistically significant. To define a course of statistically confirmed change in amplitude, absolute values of pre- and postlesion amplitudes were compared. It was assumed that the amplitude increased after surgery when its absolute postlesion value was larger than the absolute prelesion value, regardless of polarity.

RESULTS

Extent of ablation

The animals were divided into two groups with respect to the extent of their lesions. In three cats (group I) the paravermal cortex of the anterior and posterior lobes of the right cerebellar hemisphere was lesioned (*Fig. 2*). Deep cerebellar nuclei were spared in each animal. The extent of the removed cortex slightly varied. In all cases the cortex corresponded with the cerebellar interpositus nucleus (13) responsible for forelimb control (5) was sucted. In the first instance (Cat 1), the lesion was narrow and affected the right anterior lobe, lobulus simplex, paramedian lobule (except for small portions of the lateral part of the paramedian lobule) and flocculus. In the two other animals of group I, almost the entire intermediate and lateral part of the cerebellar cortex, including Crus I and II, flocculus and paraflocculus, was sucked out (Cat 2 and 3). The vermal part of the cerebellum was left intact or only slightly affected.

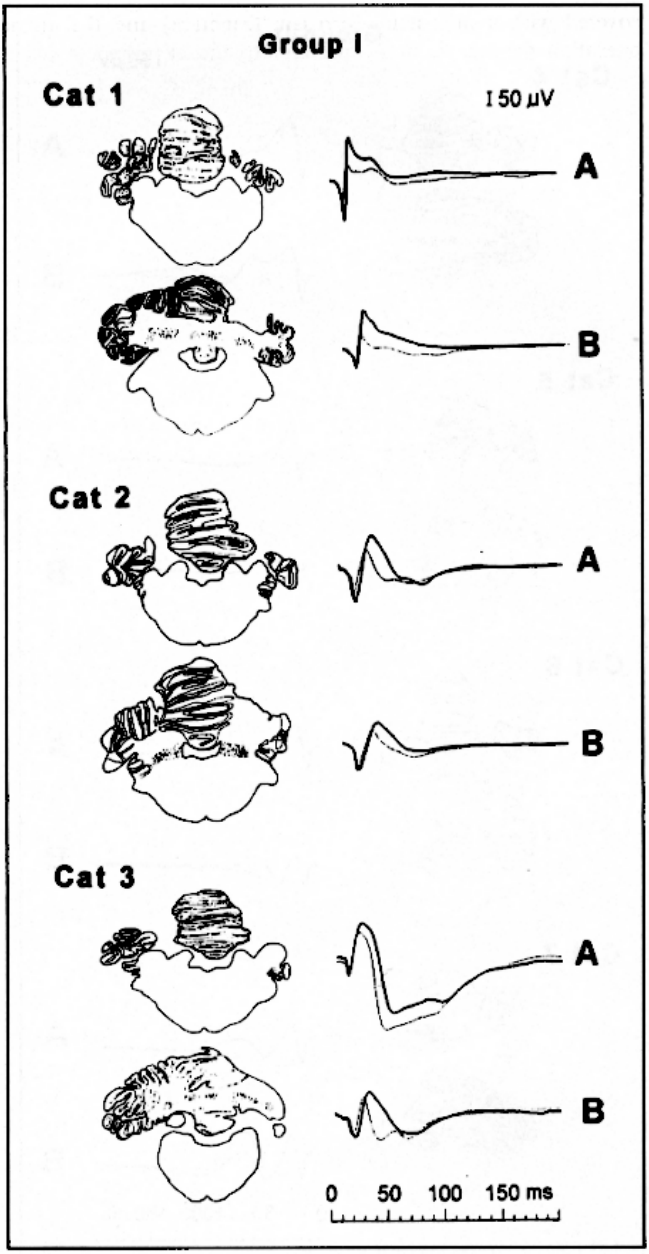

Fig. 2. Extent of lesions in the group I. Frontal sections of a partially damaged hemicerebellum. Each pair of sections is accompanied by SEPs of the animal. Potentials are collected from contralateral to the stimulated leg somatosensory primary cortex (SI). The thick trace corresponds to the postlesion period and is superimposed on the thin trace corresponding to the prelesion period. "A" response is evoked by the right forelimb stimulation and recorded on the left, affected hemisphere. "B" response is evoked by the left forelimb stimulation and recorded on the right hemisphere. The polarity is reversed.

The four remaining animals (group II) had both the cerebellar cortex and deep cerebellar nuclei impaired (*Fig. 3*). The lesions were deep and, in most cases, covered the whole intermediate and lateral part of the right hemicerebellum. Medial part of the fastigii nucleus wasn't impaired in some cases. In two instances the lower parts of the dentate nucleus remained intact.

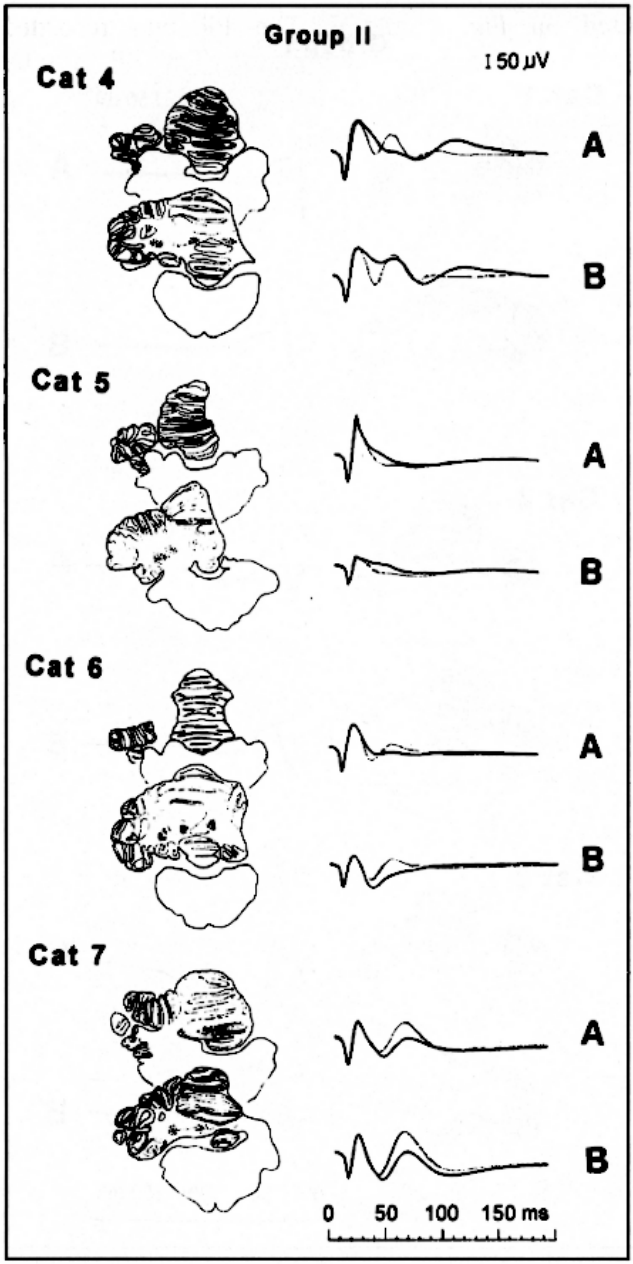

Fig. 3. Extent of lesions in the group II. Frontal sections of a complete damaged hemicerebellum. SEPs recorded from contralateral to the stimulated leg SI area are presented on the right side. All designation as in *Fig. 2.*

Prelesion primary and nonprimary SEPs

The primary and nonprimary SEPs of contralateral to the stimulated forelimb hemispheres were taken into account. The primary responses (PRs) of

individual cats are presented on *Fig. 2* and *3*. The PR was recorded in the SI field where it occurred with largest amplitude. The PR usually began approximately 12 ms after stimulation and was composed of an initial positive fast wave (P), spikelike in waveform, and 8.5 ms later a subsequent negative wave (N), milder and of longer duration. All the mentioned latencies were calculated for the wave peaks. Later discharges were often observed within the primary field but not considered here.

Nonprimary responses (NR) were explored in the pericruciate and parietal cortices. The lateral posterior portion of the Brodmann's motor area 4γ and middle suprasylvian gyrus (area 7) were the areas of the largest activation. These fields, indicated by arrows in a *Fig. 1*, showed the largest amplitudes among the NR somatic potentials and postsurgery changes were most distinct.

Generally, the NR was a sequential occurrence of three diphasic components consisting of a positive followed by a negative wave. However, the frontal SEP pattern was distinguished from the parietal potential in proportion of amplitude and latency of successive waves. Within the pericruciate area, the NR consisted of a sharp, distinct positive wave (P_{P1}) followed by a milder negative one of lower amplitude (N_{P1}). The P_{P1} and N_{P1} waves were the earliest nonprimary components of cortical origin and are defined here as early waves. This type of response was presumably the correlation of Buser and Bignall's type II NR waves. The P_{P1} wave latency was 9.7% longer than the latency of a primary P wave recorded in the neighboring somatosensory area SI. Two other positive-negative components labeled P_{P2}-N_{P2} and P_{P3}-N_{P3} were observed. They are defined here as late waves. The last pair of waves was flat and visible in half of the animals.

The response in the suprasylvian cortex was mainly a negative wave, N_{S1}. It was preceded by a small positive deflection, called the P_{S1} component, of small amplitude — or even absent in some instances. If the P_{S1} occurred, its latency was about the same as the primary P wave. The following negative N_{S1} wave was much bigger and occurred with a latency 3.4% longer than the primary N wave latency. The P_{S1} and N_{S1} (like P_{P1} and N_{P1}) are the earliest cortical pair of nonprimary waves (early waves). Two other consecutive pairs, defined here as late waves, were labeled P_{S2}-N_{S2} and P_{S3}-N_{S3}. Their parameters (latency and amplitude) were more variable than the P_{S1}-N_{S1} parameters. The P_{S3}-N_{S3} waves, flat and diffused, were occasionally visible but were widespread in the animal in which they occurred. They sometimes consisted only of one monophasic wave (and then not considered).

The prelesion amplitude and latency values of the above mentioned PR and NR components are presented in *Tab. 1*. They are in agreement with previous studies (8, 14).

Table 1. Amplitudes [μV] and latencies [ms] of primary and nonprimary prelesion components recorded from the examined areas. Names of the waves correspond with the wave nomenclature from fig. 4—5. The mean values averaged for all animals are shown in the first row and extreme values recorded within both animal groups are presented underneath.

	somatosensory cortex		pericruciate cortex					
	P	N	P_{P1}	N_{P1}	P_{P2}	N_{P2}	P_{P3}	N_{P3}
amplitudes [μV]	130.9 83 ÷ 173.5	-123.1 -38.4 ÷ -254	211.5 28 ÷ 812	-59.1 -239.9 ÷ 55.1	70 -101 ÷ 234.6	-94.6 -285.7 ÷ 51.1	50.1 2 ÷ 157.3	9.1 -1.9 ÷ 25.3
latencies [ms]	12.3 10 ÷ 15	20.8 15.5 ÷ 26	13.5 10 ÷ 16	21 15.5 ÷ 26.5	34.7 26.5 ÷ 47	52.3 37 ÷ 64	88.4 61.3 ÷ 112.5	139.1 118 ÷ 153

	suprasylvian cortex					
	P_{S1}	N_{S1}	P_{S2}	N_{S2}	P_{S3}	N_{S3}
amplitudes [μV]	66.3 8.6 ÷ 155.5	-105.5 -193 ÷ -47.5	173.5 1.3 ÷ 573	63 -250.5 ÷ 432	168.4 33 ÷ 445	28 -66.5 ÷ 191
latencies [ms]	12.2 10 ÷ 14.5	21.5 17 ÷ 25.5	43.1 33 ÷ 51	60.4 43.5 ÷ 77.5	82.2 64 ÷ 122	118.6 109 ÷ 153

Primary postlesion changes

The PRs were only altered in group I, where the absolute amplitude of the negative component N increased dramatically (*Fig. 2, 4*). The enlargement was statistically confirmed for the contralateral N wave of both hemispheres, but especially pronounced on the left side (upon right limb stimulation) contralateral to the cerebellar lesion. This effect was observed in each animal of group I, regardless of the size of the lesion (*Fig. 2*). However, it was most visible in the case of the cat with a narrow lesion (Cat 1, the increase was equal to 142% of the prelesion value on the left and 121% on the right hemispheres) and less distinct in the two other animals with lesions covering almost the whole cerebellar cortex (Cat 2 — 62% on the left and 47% on the right hemisphere; Cat 3 — 35% on the left and 26% on the right hemisphere). The absolute value of amplitude of the primary P wave increased above the prelesion level in all animals of group I, but that change was not statistically confirmed. After ablation, the latencies of both P and N waves were longer on both hemispheres.

It is worth emphasizing that any differences in amplitudes and latencies of the primary response components were unobserved in group II (compare *Fig. 2 and 3, 4 and 5*).

Nonprimary postlesion changes

The statistically confirmed postoperative changes in the NRs were observed in both groups but were more numerous in group I. The cortical cerebellar

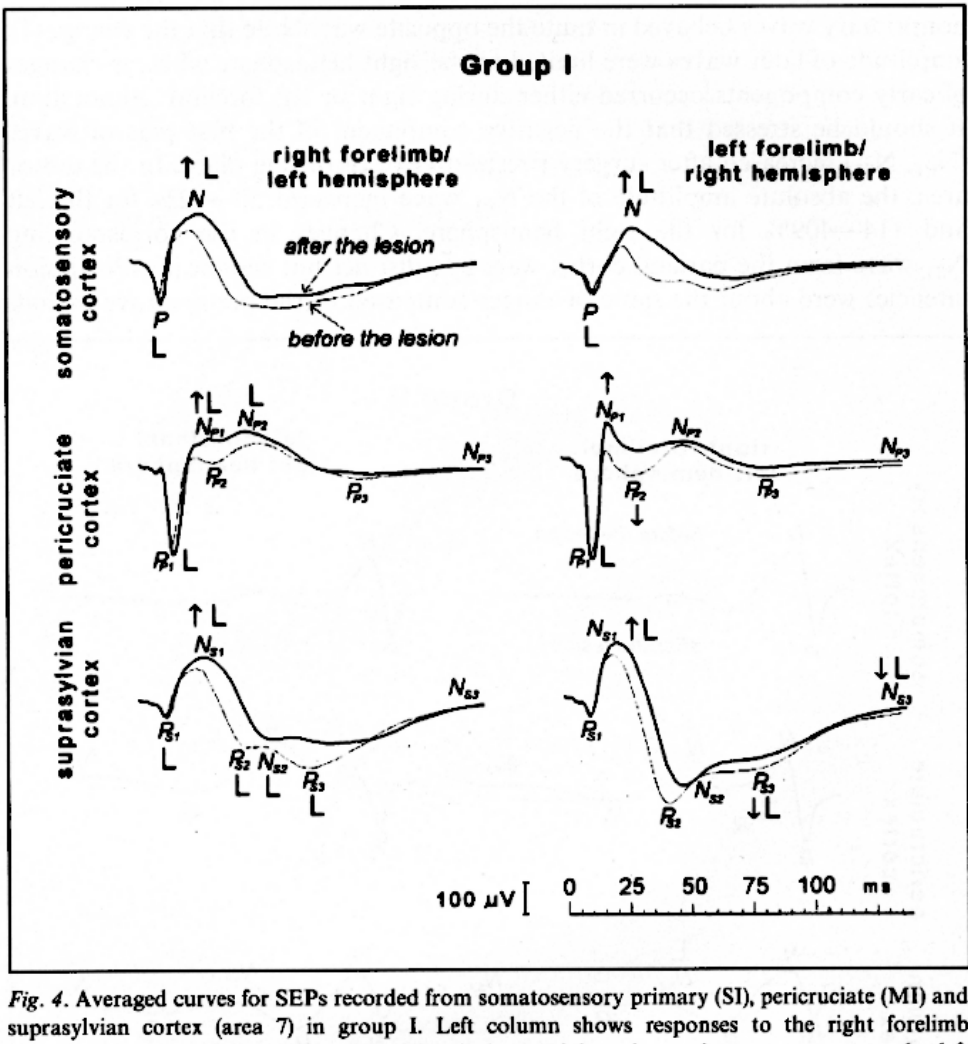

Fig. 4. Averaged curves for SEPs recorded from somatosensory primary (SI), pericruciate (MI) and suprasylvian cortex (area 7) in group I. Left column shows responses to the right forelimb stimulation, recorded from left, affected hemisphere. Right column shows responses to the left forelimb stimulation, recorded from right hemisphere. In each of the 6 parts the thick trace corresponds to the postlesion period and is superimposed on the thin trace corresponding to the prelesion period. The statistically analyzed components are denoted with their names used in the text. Postlesion changes are coded in the following way: L — statistically confirmed increase of a latency of an indicated wave, ↑ — increase in amplitude, ↓ — decrease in amplitude.

lesion in these animals influenced the potentials of both hemispheres and caused considerable changes in both early and late components (*Fig. 4*). If differences in amplitudes occurred, postlesion early waves (N_{P1}, N_{S1}) amplitudes increased while the amplitudes of the late waves were reduced (P_{P2}, P_{S3} and N_{S3} upon left forelimb stimulation). In other words early nonprimary waves reacted to lesions in the same way as primary components, while late

nonprimary waves behaved in quite the opposite way. Note that the changes in amplitude of later waves were limited to the right hemisphere whereas changes of early components occurred either during right or left forelimb stimulation. It should be stressed that the negative component of the first pair of waves (N_{P1}, N_{S1}) increased after surgery irrespective of recording place. In the motor area, the absolute amplitude of the N_{P1} wave increased 50—72% for the left and 114—409% for the right hemisphere. Changes in the corresponding N_{S1} wave from the parietal cortex were less distinct but significant. Postlesion latencies were about the same or longer compared to the preoperative period.

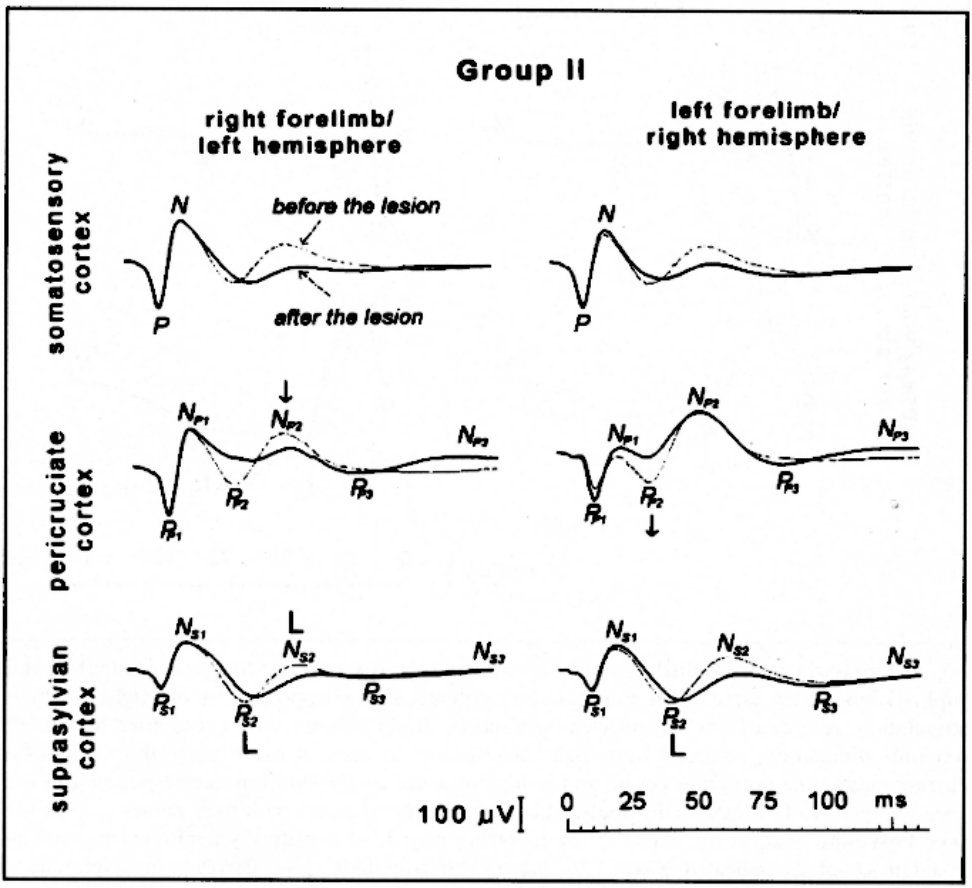

Fig. 5. Averaged curves for SEPs recorded from somatosensory (SI), pericruciate (MI) and suprasylvian (area 7) cortex in group II. All designations as in *Fig. 4.*

In group II (Fig. 5), postlesion amplitudes of parietal SEPs and latencies of motor SEPs remained unchanged. In the motor area the N_{P2} wave of the left hemisphere and P_{P2} wave of the right hemisphere were reduced in amplitude

after surgery. The latencies of hardly any wave in the parietal recordings were longer. None of the early components changed in either amplitude or latency while the corresponding waves of group I occurred later and their amplitudes increased postoperatively.

DISCUSSION

Two kinds of acute unilateral cerebellar lesion effects were studied using the SEP technique. Primary SI and nonprimary potentials, contralateral to the stimulated forelimb hemisphere, were recorded and compared before and after surgery. Among the nonprimary areas, the pericruciate cortex (lateral posterior portion of the Brodmann's motor area 4γ, which is a cortical representation of a forelimb) and middle suprasylvian gyrus (area 7) were considered.

Early nonprimary SEPs components recorded from an affected hemisphere

The pericruciate cortex and parietal areas 5 and 7 were reported to receive cerebellar influences from deep cerebellar interpositus and lateral nuclei via the ventral lateral and ventral anterior nuclei of the thalamus (4, 6, 7). Within the areas considered here, the 4γ and 7 lesions of different sizes showed a different involvement in modulation of early nonprimary components. The recorded increment of N_{P1} (area 4γ) and N_{S1} (area 7) waves after cortical, superficial lesions and a lack of their increment after deep ablation favor the inhibitory effect of the cerebellar cortex upon deep cerebellar nuclei. It should be stressed that the biggest increment of N_{P1} and N_{S1} waves were observed in a case of a superficial and narrow lesion which mainly corresponded the cerebellar cortex input to the interpositus nucleus. Unchanged early waves after deep lesions suggest that complete hemicerebellectomy, leading to the lack of cerebellar information, influences the cortical composition of discharges less than an injury limited to the cerebellar cortex. In the latter case, some distorted cerebellar input reaches the cortex and causes a severe cortical disorganization in processing. The inhibitory effect of the cerebellar cortex on the thalamo-cortical projection were reported in men with different cerebellar injuries (1) and in animals (3, 9, 10, 11) but these reports concerned changes within the sensorimotor system. As far as we know, the present results are the first confirmation of an inhibitory influence of the cerebellar cortex exerted on the parietal cortex.

Changes in SI response

Previous studies demonstrated neither the existence of a transmission arising in the cerebellum and terminating in the SI area nor SEP changes

within that region following hemicerebellectomy (15). The present paper corroborates that the ablation of the whole hemicerebellum (group II) does not alter the SI response. Nevertheless, we observe a large increment in the primary, negative wave N for cats with cortical, superficial lesions (group I). This suggests a functional relationship between the sensory cortex and cerebellum. It was previously suggested that, in a chronic situation, the somatosensory cortex plays an important role during recovery when the cerebellar input to the motor cortex is eliminated (16, 17). An altered premovement activity of the somatosensory cortex following hemicerebellectomy (17) and aggravation of recovery after combined somatosensory cortex and deep cerebellar nuclei lesions (16) may reflect somatosensory processes that promote compensation in the former case and a lack of that mechanism in the latter case. The present results indicate that a change in sensory processing occurs immediately after the lesion and before any synaptic reorganization or sprouting of new terminals appear. To the best of our knowledge, it is the first electrophysiological evidence for an engagement of the somatosensory cortex in a compensation process of a cerebellar dysfunction in acute experiments. Sensory cortex activity is acutely altered as a result of a disturbed cerebellar input to the motor cortex but not after complete lack of cerebellar information. As stated before, for the acute situation a distorted cerebellar input deforms the sensorimotor cortical processing more than a lack of cerebellar information. However, both kind of lesion produces severe cerebellar symptoms.

Several weeks after impairment an entirely new functional organization might start to replace the compensatory motor function of the sensory cortex (17). The elimination of the cerebellar input induces a synaptic reorganization in the motor cortex and changes the sensory input to that area (18). Since, in normal cats, the projection from the sensory to motor cortex may represent a cortical motor regulatory mechanism, like the motor regulating mechanism in the cerebellum (19), it is tempting to say that a loss of cerebellar information may gradually increase the role of cortico-cortical transmission in sensorimotor processing followed by compensation of impaired cerebellar functions and in that way substitute cerebellar information that normally support the cortical mechanism. A capacity of the somatosensory cortex to compensate impaired motor functions is worth further, more detailed consideration. The time course of cortical activity changes after chronic lesion is the matter of our next studies (in preparation).

SEPs in the unaffected hemisphere

In the present paper we focus on SEPs contralateral to the stimulated forelimb. The potentials were recorded for the affected and unaffected cerebral hemispheres. Changes in amplitude and latency after surgery were observed in

both hemispheres, although the lesion was always confined to the right hemicerebellum. Changes of activation in the unaffected hemisphere were slightly less pronounced but statistically confirmed. This result may indicate the existence of both contra- and ipsilateral spino-cerebellar and cerebello-cortical projections that are not blocked under chloralose anesthesia and unmasked after cerebellar lesion. This mechanism — unmasking and enhancing of normally existing (especially ipsilateral) connections — is probably responsible for a change of cortical activity immediately after impairment. There is a large body of evidence for ipsilateral activation after injuries in man. Bilateral changes of cortical activity after cerebellar degeneration were previously reported (2). Ipsilateral cortico-spinal motor responses were evoked by magnetic stimulation of the affected or unaffected hemisphere (or both of them) after stroke and during recovery (20—23). The significance of ipsilateral activation is a matter of discussion. Undoubtedly, motor outputs in the unaffected hemisphere may be significantly changed after stroke, including ipsi- and contralateral projections (22). Our results show changes in the contralateral projection to the unaffected hemisphere caused by cerebellar unilateral deprivation in cats. The conclusion may be reached that the activation of the unaffected hemisphere cannot be used as a control and bilateral recordings may not detect cerebellar unilateral impairments or any asymmetries of injury.

Early versus late nonprimary SEP components

According to the authors knowledge, an effect of cerebellar lesion on late SEP components was not published previously. Our results indicate that early and late waves are affected in different ways. Early nonspecific and SI waves were increased after cortical cerebellar lesion only in group I. Late nonspecific waves amplitudes were suppressed below the prelesion level in both groups. Two different mechanisms of cerebellar lesion impact on SEPs should be considered and they are both present in the animals of group I. It was previously suggested that early and late responses come from different sources. The generation of an initial pair of positive-negative waves is associated with transmission throughout the spinocerebellar tract (24). Due to our results, these early, exogenous SEP components' behavior depends on the kind of cerebellar injury and cerebello-thalamo-cortical projection. Changes in late, endogenous waves are unrelated to the extent of the lesion and probably reflect a reorganization of cortico-cortical transmissions compensating for the lack or disturbed cerebellar input.

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