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Evaluation of Somatosensory Evoked Potential in Temporary Occlusion of Cerebral Artery 1. Experimental Study in Cats

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Introduction

Technical difficulties of direct operations for ruptured cerebral aneurysms in the acute stage comprise access to the aneurysm retracting the swollen brain, avoidance of or immediate management for the dreadful premature rupture of the aneurysm during dissection, and complete obliteration of the aneurysmal neck without encroaching on the lumen of the parent artery. Application of a temporary clip on the parent artery is a more sensible way to reduce the blood pressure within the aneurysmal sac than use of pharmacologically induced systemic hypotension. Tolerance of the brain for ischaemia and reversibility of cerebral function after reperfusion depend on the selective vulnerability of the brain tissues, amount of remaining cerebral blood flow (CBF) during temporary occlusion and duration of critical levels of ischaemia.

During an intraoperative, iatrogenic focal cerebral ischaemia secondary to temporary occlusion of the cerebral artery, close monitoring of time course of remaining brain function is mandatory. Such monitoring must be repeatable in a short time, it must reflect the changes in CBF, especially when CBF approaches the critical range, and of course it must be non-invasive and should not interrupt or prolong the surgical manoeuvre.

Use of somatosensory evoked potential (SEP) in the operating room or in the ward has been reported in patients with ruptured cerebral aneurysms[17,18]. However, interpretation of changes in the SEP during temporary clipping of the parent artery is difficult with inherent limitations because of a variety of affecting factors including mode and depth of anesthesia, and its ability to correctly predict recovery of cerebral function following temporary ischaemia remains to be confirmed. The purposes of this experimental study were to establish the tolerable threshold of ischaemia by studying relationships between components of the SEP and local CBF, and to show the possibilities, if present, of SEP to predict the outcome of animals after focal cerebral ischae-

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mia lasting one hour followed by reperfusion.

Materials and Methods

Preparation of animals

Data were obtained from 9 cats weighing from 2.3 to 4.0 kg. Anesthesia was induced with an intramuscular injection of ketamine hydrochloride (20 mg/kg) and atropine sulphate (0.01 mg/kg), and endotracheal intubation was performed. The femoral artery was catheterized for the purpose of monitoring the blood pressure (Nihon Koden, MPU-0.5 U) and sampling the blood, and the femoral vein was cannulated for administration of fluids and drugs. After immobilization by an intravenous injection of pancuronium bromide (0.1-0.25 mg/kg), animals were artificially ventilated with room air (Acoma, AR-100). Pancuronium bromide was added as necessary. Intravenous administration of ketamine hydrochloride and local infiltration with 1% lidocaine hydrochloride solution were used during surgical procedures.

$PaCO_2$ and PaO_2 were maintained within the normal ranges, namely, 30 ± 3 Torr and above 90 Torr, respectively. The pH of the arterial blood was between 7.35 and 7.45 and the core temperature at 37.5 ± 0.5°C during the study. Mean systemic blood pressure was maintained between 100 to 140 Torr. Isotonic fluids were administered intravenously at a rate of 5.7 ml/hour.

The left orbit was exenterated and the left optic foramen was enlarged using a saline-cooled dental drill, and the left middle cerebral artery (MCA) was exposed. Under an operating microscope (Zeiss OPM1), the MCA was carefully dissected from the arachnoid membrane and clipped using a Yasargil microclip immediately distal to the perforating arteries supplying the basal ganglia.

Monitoring of SEP

SEP was recorded from the left and right sensory cortex after stimulation of the contralateral median nerve (Medelec MS-6). The silver ball recording electrodes, 1 mm in diameter, were placed on the dura mater over the posterior sigmoid gyrus that corresponds to the primary somatosensory area for the forelimb[10] This point is 8 to 10 mm lateral to the midline and 2 to 3 mm anterior to the coronal suture. A reference electrode was placed on the chin. Platinum stimulating electrodes were inserted into the exposed median nerve of each forelimb as atraumatically as possible, and covered by a sheet of rubber and cottonoid pledget soaked in warm paraffin. The median nerve was stimulated with square wave pulses of the supramaximal voltage (15 to 18 V), 3 Hz in frequency and 0.2 ms in duration. Somatosensory evoked responses were amplified with a bandpass of 32 to 1600 Hz and averaged over 128 sweeps in a 50 ms time window.

Measurement of CBF

CBF was measured at four sites using a hydrogen clearance technique[1,19] According to the SNYDER's coordinates[14], the polarographic electrodes made of platinum-iridium (90%/10%) wire, 0.2 mm in diameter, were stereotaxically inserted through the small burr holes to the left posterior sigmoid gyrus, left ectosylvian gyrus, left thalamus, and right (contralateral to the occlu-

sion of the MCA) posterior sigmoid gyrus. A silver-silver chloride reference electrode was placed in the temporal muscles. Hydrogen gas was given via an endotracheal tube in the concentration of 5% (vol/vol) for 1 minute. All CBF values were calculated using the initial slope index method[9]

Experimental protocol and statistical analysis

Nine cats underwent transorbital MCA occlusion. CBF and SEP were measured before, and 5, 15, 30, and 60 minutes after clipping of the MCA. Then the clip was released (one hour occlusion) and the orbit was sealed to prevent escape of excess amount of the cerebrospinal fluid. Recordings of CBF and SEP were repeated further 5, 15, 30, and 60 minutes after release of the clip, and thereafter at a one hour interval until three hours after start of reperfusion.

Statistical analysis was performed using a t-test for comparisons between pre- and post-ischaemic values, and with an unpaired t-test for comparisons between the groups. A p value <0.05 was regarded as statistically significant.

Results

Description of SEP peaks

In cats, the posterior sigmoid gyrus representing the primary sensory area for the forelimb corresponds to the circulatory border zone between the MCA and the anterior cerebral artery. The cortical association area situates in the anterior and middle suprasylvian gyri and a part of the ectosylvian gyrus and the lateral gyrus which are included in the central ischaemic area or ischaemic penumbra following occlusion of the MCA[11,15]

Record of SEP obtained in the posterior sigmoid gyrus is schematically shown in Fig. 1. It has a small positive peak (P_1) which is thought to be thalamocortical in origin[5,16]. This is followed by a small negative-positive complex (N_1, P_2), which is more or less variable but thought to be primary sensory cortical origin. Next major negative (N_2) and positive (P_3) peaks originate in the association cortex[5], P_1, N_2 and P_3 peaks were consistently observed in this study, and I used P_1 and N_2 peaks for analysis. Conduction time was defined as P_1-N_2 interpeak latency, and amplitude as the height of N_2 peak measured from the baseline connecting P_2 and

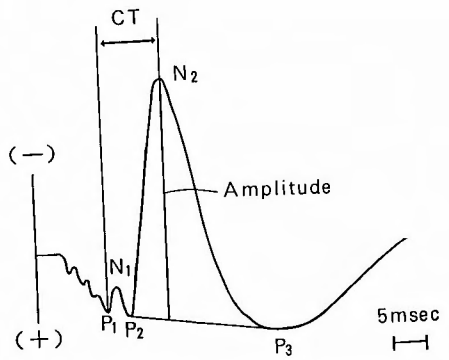

Fig. 1 : Schematic drawing of SEP recorded from the posterior sigmoid gyrus by stimulation of the contralateral median nerve. CT: conduction time=P_1-N_2 interpeak latency.

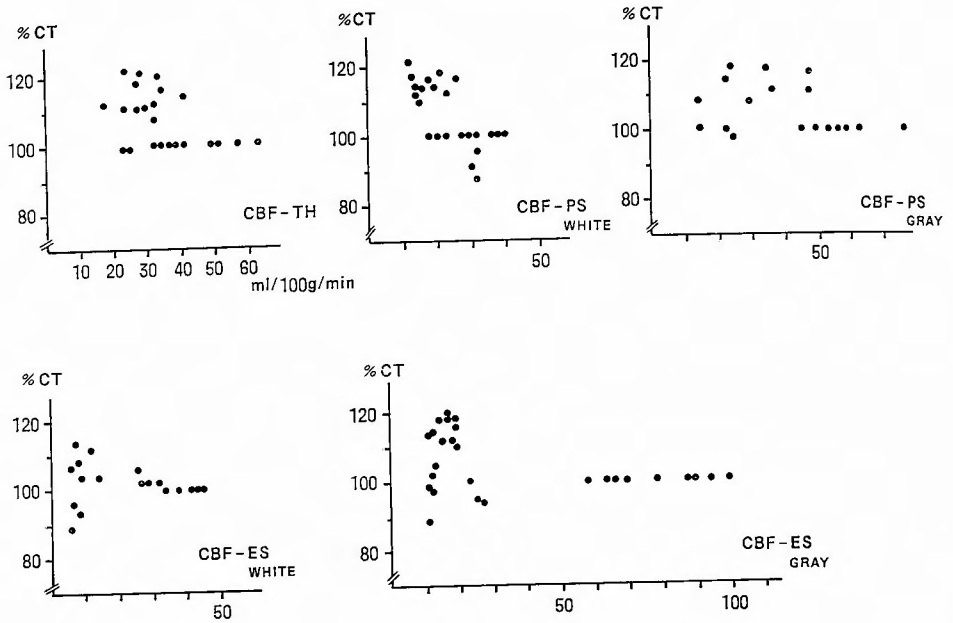

Fig. 2 Relationship between conduction time and local cerebral blood flow during occlusion of the left middle cerebral artery. % CT: conduction time in terms of % to the control value, CBF: local cerebral blood flow, TH: thalamus (left), PS: posterior sigmoid gyrus (left), ES: ectosylvian gyrus (left), WHITE: white matter, GRAY: gray matter.

P_3. Values of conduction time and amplitude were shown as the per cent to the control values obtained before occlusion of the MCA.

Changes in SEP correlated with CBF during one hour ischaemia

1 *Conduction time versus CBF* (Fig. 2)

During one hour occlusion of the left MCA, elongation of conduction time up to 120% was observed.

Left thalamus: In the left thalamus, CBF in the control state was 45.9±11.0 ml/100 g/min (mean±S.D.). Although most of the perforating arteries arising from the horizontal portion of the MCA were carefully spared, CBF was reduced to 25.8±8.1 ml/100 g/min after occlusion of the MCA ($p<0.05$). Conduction time in terms of per cent distributed between 100 and 120% of the control values with the reduced CBF ranging from 17 to 35 ml/100 g/min.

Left posterior sigmoid gyrus: CBF in the white matter of the left posterior sigmoid gyrus was 30.1±6.3 ml/100 g/min in the control state, which was reduced to 15.4±7.9 ml/100 g/min after occlusion of the MCA ($p<0.05$). In the cortical gray matter, CBF of 53.4±11.7 ml/100 g/min decreased to 26.0±15.4 ml/100 g/min after occlusion of the MCA ($p<0.05$). Prolongation of conduction time scattered against wide range of reduced CBF, the range of CBF being wider in the gray matter (15 to 50 ml/100 g/min) than in the white matter (10 to 25 ml/100 g/min). Conduction time did not show progressive Prolongation with the reduction of the blood flow.

Left ectosylvian gyrus: CBF in the white matter of the left ectosylvian gyrus was 36.7±7.3

ml/100 g/min in the control state. It decreased to as low as 9.2±6.7 ml/100 g/min during ischaemia. CBF in the gray matter decreased from a control value of 80.4±14.3 ml/100 g/min to 17.8±6.6 ml/100 g/min after occlusion of the MCA. Both these changes were statistically significant ($p<0.01$). Conduction time distributed between 90 and 120% against a wide range of reduced CBF in the gray matter (5 to 40 ml/100 g/min). Conduction time showed a tendency to elongate at the gray matter blood flow level of 10 to 18 ml/100 g/min. In the white matter, conduction time seemed to elongate when the CBF decreased to the lower level of 5 to 15 ml/100 g/min.

Right posterior sigmoid gyrus: The CBF in the gray or white matter of the right posterior sigmoid gyrus (contralateral side to the occluded MCA) showed no significant changes either during ischaemia or after recirculation. The conduction time by the left median nerve stimulation showed no significant changes throughout the experiment in the right posterior sigmoid gyrus.

2 *Amplitude of SEP versus CBF*: (Fig. 3)

Left thalamus: Amplitude of SEP declined acutely as the CBF in the thalamus was reduced to around 30 ml/100 g/min, and the SEP peaks were no longer recorded at the blood flow level of 20 ml/100 g/min or less.

Left posterior sigmoid gyrus: In the white matter of the posterior sigmoid gyrus, amplitude of SEP declined rapidly and progressively at the threshold flow level of 20 to 12 ml/100 g/min. In the gray matter, the scattering of the data did not permit definite estimation of threshold

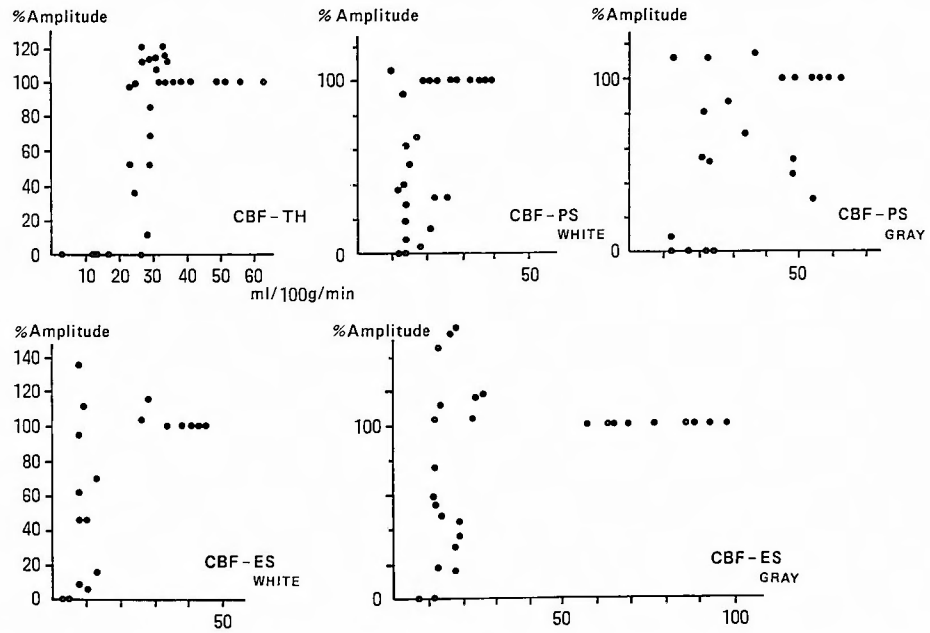

Fig. 3 Relationship between amplitude and local cerebral blood flow during occlusion of the left middle cerebral artery. % Amplitude: amplitude in terms of % to the control value. Other Other abbreviations are the same as used in fig. 2.

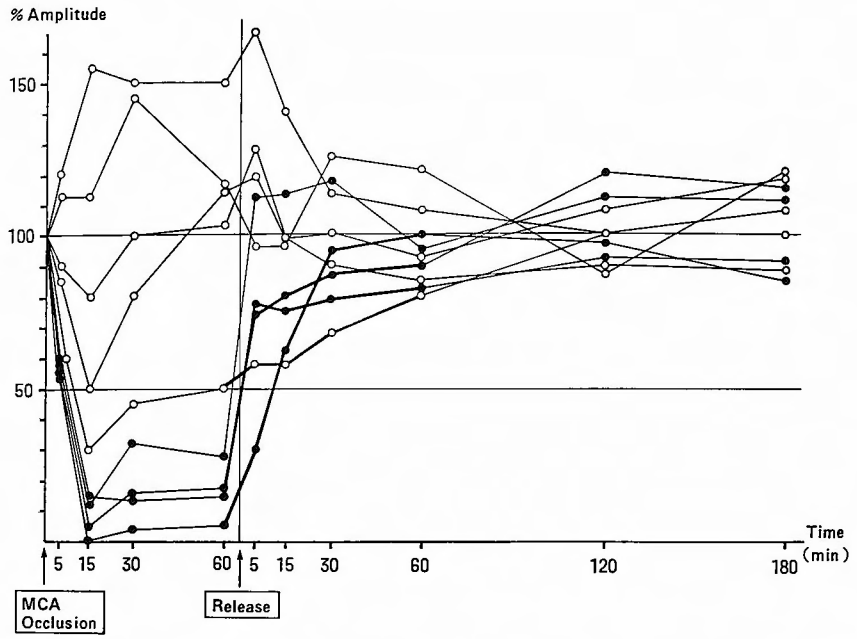

Fig. 4 Time course of amplitude during and after occlusion of the middle cerebral artery. % Amplitude: amplitude in terms of % to the control value, MCA: middle cerebral artery.

flow level, but it seems that the amplitude of SEP decline at a higher level of blood flow than in the white matter, at around 20 to 30 ml/100 g/min.

Left ectosylvian gyrus: Amplitude of SEP showed a clear threshold value of blood flow at 10 to 18 ml/100 g/min in the gray matter of the ectosylvian gyrus. In a few studies, however, some increase in amplitude was also recorded at the low blood flow level between 30 to 12 ml/100 g/min. Amplitude of the SEP showed a threshold blood flow also in the white matter of the ectosylvian gyrus, at the level of 5 to 13 ml/100 g/min.

Right posterior sigmoid gyrus: The amplitude recorded on the right posterior sigmoid gyrus (contralateral to the MCA occlusion) by the left median nerve stimulation showed no significant changes throughout the experiment.

3 *Time course of changes of amplitude*: (Fig. 4)

Two out 9 cats showed an increase in amplitude of SEP throughout the period of focal ischaemia lasting 60 minutes. In the remaining 7 cats, the lowest value of amplitude was reached 15 minutes after occlusion of the MCA. In 3 of them, amplitude reached the nadir at 15 minutes postocclusion, but the decrease was relatively small and the amplitude gradually recovered toward the control level, reaching at least the level of 50% at the end of occlusion. In other 4 cats, amplitude remained reduced at 0 to 30% level during the whole period of measurements up to 60 minutes postocclusion. These 4 cats showed significantly lower amplitude already at an earlier measurements at 5 minutes postocclusion, and in all of them CBF in the posterior sigmoid and ectosylvian gyri on the left side was significantly lower when compared to the CBF in other cats (Fig. 5).

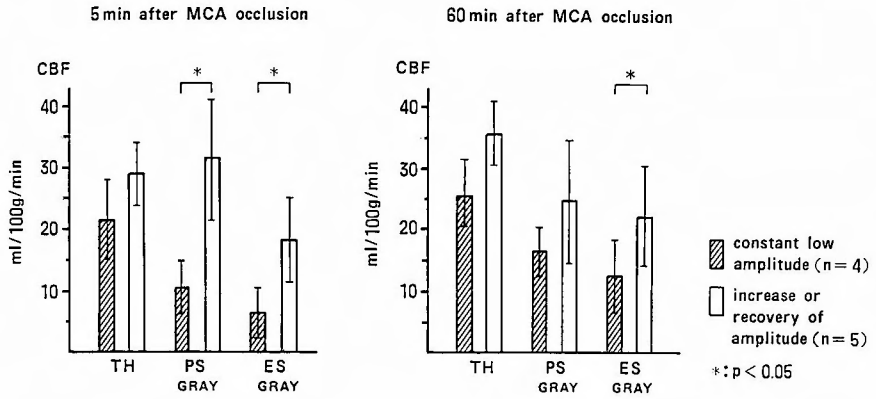

Fig. 5 Comparison of remaining local cerebral blood flow during occlusion of the middle cerebral artery between two groups of cats, one group of 4 cats with constant low amplitude and the other group of 5 cats that showed increase, or decrease but tendency to recover, of amplitude. Mean±SD. MCA; middle cerebral artery, TH: thalamus (left), PS GRAY: gray matter of left posterior sigmoid gyrus, ES GRAY: Gray matter of left ectosylvian gyrus, *: $p<0.05$.

Changes in SEP correlated with CBF after reperfusion

1 *Conduction time versus CBF* (Fig. 6)

In analysing the relationships between the conduction time in terms of per cent to the control value and CBF in the thalamus, the posterior sigmoid gyrus and the ectosylvian gyrus,

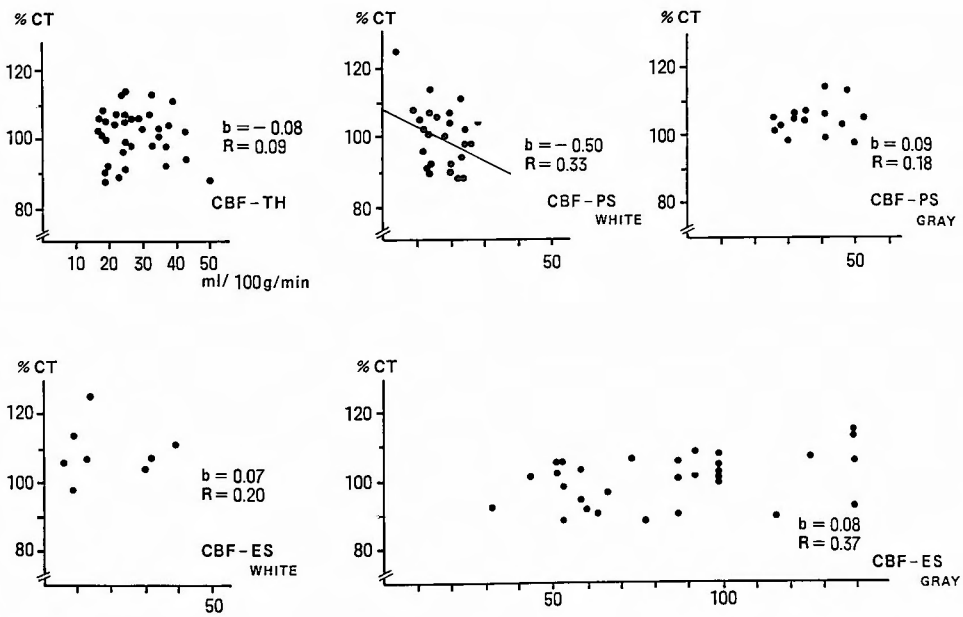

Fig. 6 Relationship between conduction time and local cerebral blood flow after reperfusion. b: slope of regression line, R: correlation coefficient. Other abbreviations are the same as in fig. 2.

regression line and correlation coefficient were studied. Slope (b) was significantly different from zero only for the white matter blood flow in the posterior sigmoid gyrus ($b=-0.50$), but the correlation coefficient (R) between the conduction time and the CBF was low ($R=0.33$). No significant linear relationship was ever found for the slopes and the correlation coefficients were also low in any other areas.

2 *Amplitude versus CBF* (Fig. 7)

Amplitude in terms of per cent to the control value was plotted as a function of CBF measured in the thalamus, the posterior sigmoid gyrus and the ectosylvian gyrus. When a plot of the amplitude versus the gray matter blood flow in the ectosylvian gyrus was analysed as a whole, there was no correlation ($R=0.05$). When the amplitude was studied as a function of the CBF up to 130 ml/100 g/min in this area, however, the slope ($b=0.23$) was significantly different from zero, and the correlation coefficient (R) was 0.45. When the CBF values exceeded 130 ml/100 g/min, amplitude showed a lower value, that is approximately 60 to 80% of the control. The slopes were very close to zero, and correlation coefficients were low in any other regions.

3 *Pattern of postischaemic changes of amplitude* (Fig. 4)

When the clip on the MCA was released and the blood flow was restored to the ischaemic area, amplitude of SEP showed a tendency to restore the control values in all cats. Three patterns of changes are classified in this period of reperfusion, namely (A) gradual recovery towards the

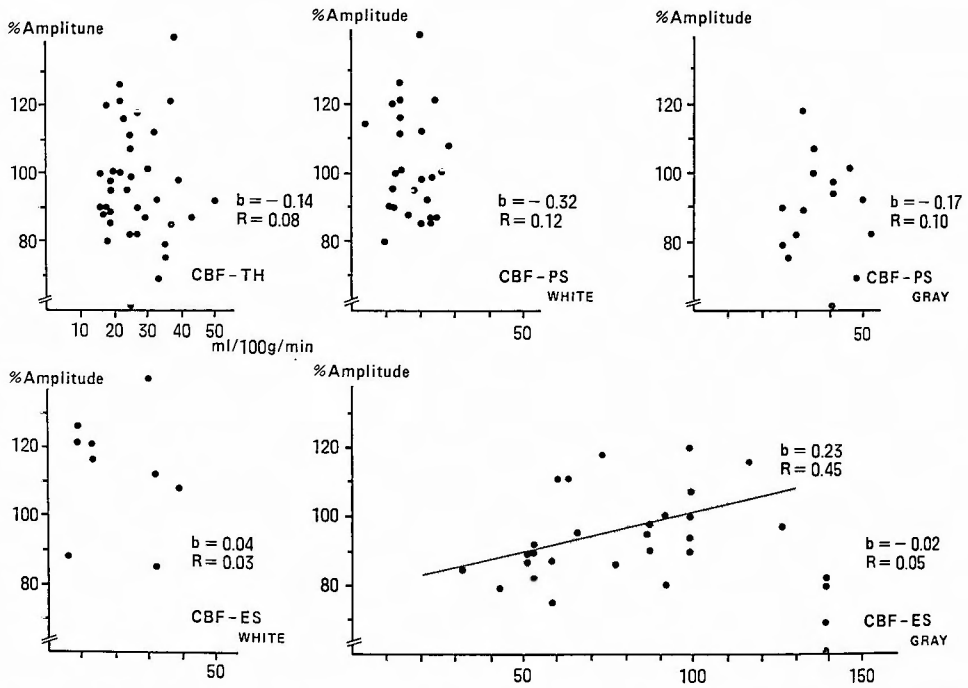

Fig. 7 Relationship between amplitude and local cerebral blood flow after reperfusion. b: slope of regression line, R: correlation coefficient. b and R above the regression line in CBF-ES GRAY are for the CBF values up to 130 ml/100 g/min. Other abbreviations are the same as in fig. 3.

control as seen in 4 cats, (B) immediate recovery, seen only in 1 cat, and (C) near normal or higher than control levels had been already reached at the measurement done 60 minutes postocclusion and immediately prior to release of clipping, seen in 4 cats. In all 4 cats of group A, amplitude of SEP was lower than 50% of the control at 60 minutes after clipping of the MCA, which was significantly low compared to those of other groups.

Discussion

Of essential importance for interpreting SEP during cerebral ischaemia is to understand the relationship between the anatomy of somatosensory pathways which contribute to SEP and the area of the brain suffering from focal ischaemia. In cats, the first primary somatosensory area for the forelimb is located in the posterior sigmoid gyrus, which corresponds to the border zone of the MCA and the anterior cerebral artery. The second primary somatosensory area is in the anterior ectosylvian gyrus, the association cortex is in the parietal convexity, where the penumbra and the central ischaemic area are known to arise following clipping of the MCA[10,11,19].

SEP and ischaemia

COYER et al. reported that there was no relationship between the cortical component latency of SEP and the white matter blood flow of the ectosylvian gyrus during focal ischaemia of cat cerebral hemisphere, although white matter blood flow is well correlated with the latency on the contralateal hemisphere[4]. In the present experiments, however, conduction time elongated up to 120% at CBF values of 10 to 18 ml/100 g/min in the gray matter of the ectosylvian gyrus. On the other hand, the prolongation of the conduction time was seen at a wider range of CBF in the gray matter of the posterior sigmoid gyrus, ranging from 15 to 50 ml/100 g/min.

In the centre of cerebral ischaemia that is the ectosylvian gyrus in this study in cats, we found that amplitude of SEP had a threshold of gray matter blood flow at 12 to 18 ml/100 g/min.

In the centre of cerebral ischaemia that is the ectosylvian gyrus in this study in cats, we found that amplitude of SEP had a threshold of gray matter blood flow at 12 to 18 ml/100 g/min. This result is similar to that of BRANSTON et al., who used a model of MCA occlusion associated with hypotension in baboons[2].

For the gray matter blood flow in the ectosylvian gyrus ranging from 12 to 25 ml/100 g/min, amplitude of SEP actually showed either an increase or a decrease, ranging from 0 to 155%. An increase in amplitude in the face of reduction of blood flow is thought to be caused by disturbed suppression from higher cortical systems in the territory of supply of the MCA.

Some authors reported that the latency changes of evoked potentials were too variable to be measured during ischaemia[7,9]. Others described that amplitude changes were more variable than changes in latency during focal ischaemia[4]. When cerebral blood flow was reduced by occlusion of the MCA, drifts of conduction time, and especially of amplitude were found in the present study, too. However, such drifts were not severe and accurate measurements of amplitude and latency were possible. Less drifts in our study compared to those seen in others might be due to the facts that in our model the site of occlusion of the MCA was constant and

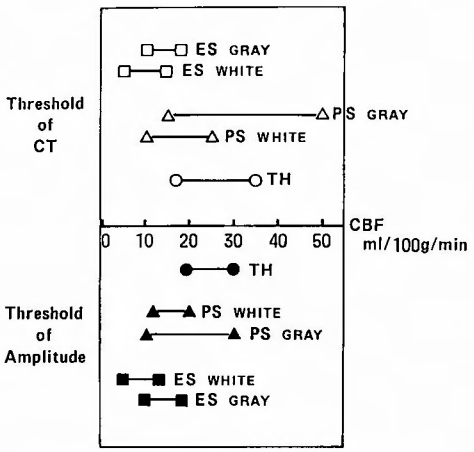

Fig. 8 Threshold of CBF for SEP changes during occlusion of the middle cerebral artery. CT: conduction time, ES: ectosylvian gyrus, PS: posterior sigmoid gyrus, TH: thalamus, GRAY: gray matter, WHITE: white matter.

pin-pointed, that the ischaemia was probably less severe, that the stable condition of the animal could be maintained, and to some other unknown factors. Besides progressive deterioration, it should be stressed that an increase of cortical component amplitude was occasionally seen as the CBF was reduced in the parietal lobe. It happened when the CBF was at least preserved to maintain SEP in primary sensory area and the association cortex.

In the present study, both conduction time and amplitude of SEP showed almost the same ranges of threshold blood flow values in all regions, except for in the white matter of the posterior sigmoid gyrus where the threshold of blood flow for amplitude changes was narrower (Fig. 8). This result indicates that conduction time and amplitude are sensitive to ischaemia almost in the same scale. Both conduction time and amplitude were sometimes seemingly vulnerable to slight reduction of cerebral blood flow in the posterior sigmoid gyrus. Those changes were in fact owing to the reduced blood flow in the association cortex, which was lower than that in the posterior sigmoid gyrus in such an occasion.

It has been suggested that in terms of amplitude of SEP, the phylogenetically older structures of the neuraxis are more resistant to ischaemia[2], and MARCOUX et al. also suggested that the subcortical gray matter was histologically more vulnerable to ischaemia than the cortex[13]. Contrary to these hypotheses, results of our experiment indicate that the white matter of the association cortex is most resistant to ischaemia. We conclude that there are regional difference in sensitivity to ischaemia among the brain structures, and that sensitivity is influenced by many factors especially by eletrophysiological susceptibility of synaptic and axonal transports.

SEP after removal of clip

There have been only few reports on the changes in SEP after temporary occlusion of the MCA in experimental animals[9]. In the present study, white matter blood flow in the posterior sigmoid gyrus showed a higher correlation with conduction time than the CBF in other areas.

This result is resonable as the conduction time is mainly controlled by transmission in the white matter. On the other hand, amplitude had a positive linear relationship with gray matter blood flow in the ectosylvian gyrus when the flow did not exceed 130 ml/100 g/min. It is of interest to note that amplitude of cortical component of SEP remained almost stable within the range of 80 to 120% for the cerebral blood flow values of 60 to 130 ml/100 g/min, and that amplitude declined abruptly to 60 to 80% when the cerebral blood flow exceeded 130 ml/100 g/min in the gray matter of the ectosylvian gyrus. This result is consonant with the theory that hyperemic luxury perfusion during reperfusion after temporary ischemia is rather harmful to the cerebral function[12].

Prediction of postischaemic SEP

In this experimental study, we used animal model of focal cerebral ischaemia produced by microsurgical occlusion of the MCA without an induction of systemic hypotension. Cerebral blood flow in the territory of the MCA was reduced to 16.2±11.6 ml/100 g/min (approximately 20% of control) for one hour. According to CARTER et al. and JONES et al., severity of ischaemia obtained in our model was so mild that probably it would not cause disappearance of direct cortical responses or permanent histological changes of infarction[3,8].

Amplitude of SEP recovered either promptly or gradually toward the control levels in all nine animals after release of an occluding clip. When amplitude decreased during occlusion either to the levels lower than 60% at 5 minutes (5 cats), or to the level lower than 50% between 15 and 60 minutes after clipping (5 cats), its recovery was gradual during postischaemic period, except for one cat which showed very low amplitudes during the period of ischaemia but showed a prompt recovery after reperfusion. We could not find adequate explanation for this particular phenomenon. Those animals with low amplitudes and slow recovery were found to have had significantly low blood flow in the posterior sigmoid and ectosylvian gyri.

In conclusion, our results imply that SEP is an adequately sensitive monitor, not only to display brain function during ischaemia, but also to predict the recovery of function after temporary focal ischaemia of the brain in cats. Clinical application of SEP as a monitoring during temporary occlusion of the cerebral artery in aneurysmal surgery in humans may well be useful and seems to deserve detailed study.

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和文抄録

一時的脳血流遮断における体性知覚誘発電位
1. 実験的評価

滋賀医科大学脳神経外科
中洲庸子

成猫の一側中大脳動脈を1時間閉塞して，虚血中および再開通後に正中神経刺激による体性知覚誘発電位(SEP)を測定し，水素クリアランス法による脳血流量との比較を行なった．

中大脳動脈の潅流域の中心は知覚統合野にあたり，SEPの皮質成分N_2の振幅はこの領域の皮質脳血流量が12-18 ml/100 g/minのとき急激に減少した．また，この閾値以上の皮質血流量に対しては，振幅の増大する例も観察された．視床から皮質への伝導時間は，皮質血流量に対しほぼ同じ閾値内で最大120％まで延長を認めた．再開通後，振幅は知覚統合野の皮質血流量と，伝導時間は一次知覚領野の白質血流量とほぼ直線関係を示した．注目されるのは，皮質血流量が130 ml/100 g/min を越えると振幅が急激に減少したこと

で，虚血後の過灌流状態が神経機能に悪影響を有することが示唆された．

虚血後5分に振幅が60%以下まで，又は15～60分後に50%以下まで低下する場合は，有意に低い皮質脳血流量を示し，再灌流後の振幅の回復が遅延した．

これらの結果から，一時的な中大脳動脈閉塞における SEP は脳血流量の減少に際して，神経機能の可逆的な段階で一定の変化を表わし，中大脳動脈領域の血流遮断中およびその後の脳血流量，機能の指標として有用であるとの結論を得た．