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A Study of Interhemispheric Diaschisis: Reduction of Cerebral Blood Flow in Both Hemispheres Following Unilateral Middle Cerebral Artery Occlusion in Cats†

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=Abstract=To demonstrate the effect of interhemispheric diaschisis in acute unilateral cerebral infarction, the changes of regional cerebral blood flow (rCBF) following experimental occlusion of one middle cerebral artery(MCA) for up to 16 hours were investigated in an unanesthetized cat model using the technique of transorbital snare ligature implanted in the intact cranium. rCBF of both MCA territories and both marginal zones were measured simultaneously by the hydrogen clearance technique. 25 cats were divided into 2 groups according to the size of infarct; Group A, infarct larger than 10% of ipsilateral coronal sectional surface of hemisphere and Group B, smaller than 10%. During the initial stage of ischemia, significant decrease in rCBF of the contralateral hemisphere was observed in both groups, however, in Group A, rCBF gradually increased toward preocclusion level. In Group B, rCBF decreased further and remained low till the 16th hour after occlusion. These results suggested that mild ischemic Group B would take advantage of interhemispheric diaschisis during the late phase of infarct development as well as the substantial collateral flow from the surrounding anterior and posterior cerebral artery territories.

Key Words: ***Interhemispheric diaschisis, Regional cerebral blood flow, Cerebral infarction, Hydrogen clearance method***

INTRODUCTION

From clinical observations, von Monakow, in 1914, first formulated the concept that a transient depression of the brain function can occur at a distance from a circumscribed lesion of the brain. He applied the term 'diaschisis' to designate this phenomenon. During the last several decades there were many studies showing that in acute unilateral infarction cerebral blood flow and metabolism may be reduced in both cerebral hemispheres (Lavy *et al.* 1975; Meyer *et al.* 1970; Reivich *et al.* 1977; Slater *et al.* 1977). Recently the occurrence of diaschisis at the multiple sites following cerebral infarction was well established in man with the use of positron emission tomography (PET) (Baron *et al.* 1981; Martin and Raichle 1983; Wise *et al.* 1983). There have been, however, limitations in establishment of an ideal experimental model for the production of focal cerebral ischemia and also in the methods to measure rCBF.

In the present study, we used the unanesthetized cat model implanting transorbital snare ligature in the intact cranium to occlude unilateral MCA abruptly after recovery from the surgery. The rCBF was recorded before and after MCA occlusion by the hydrogen clearance technique on both MCA territories and marginal zones. The aim of this study is to demonstrate the interhemispheric diaschisis in experimental focal ischemia and its influence, if any, upon the ischemia and the development of irreversible infarction.

MATERIALS AND METHODS

Thirty adult mongrel cats of both sexes, weighing

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2.4 to 3.3 kg, were studied. Five animals were used as sham operational controls and the remaining twenty-five were studied as MCA occlusion models.

Preparation Procedures

The animals were starved overnight and were anesthetized with ketamine (10 mg/kg intramuscularly), atropine (0.025 mg/kg intramuscularly), and pentobarbital (20 mg/kg intraperitoneally). The femoral artery was cannulated and connected to a Statham P23G blood pressure transducer via a three-way tap, to allow arterial blood gas sampling and to maintain normotensive throughout the operation. After immobilizing the animal by the headholder, the left eyelid was spread wide after incising the lateral fissure of the eyelid 1.0 cm long. After infiltration of 2% lidocaine into the orbit to avoid the vagal reflex, the globe was deflated and the orbital content was removed. The optic foramen was enlarged superomedially to make a hole of 4-5 mm diameter using a high-speed electric drill. The internal carotid artey (ICA) and proximal MCA were then exposed following dural incision. After the meticulous splitting of the arachnoid, a 8-0 nylon silk was looped carefully around the proximal MCA within 2-3 mm from its origin. This snare ligature was passed outside the incision through a narrow polyethylene tube fixed like in the Figs. 1 and 2. To identify the degree of occlusion of the artery the snare ligature was pulled and Heifetz clips were applied and the number of clips required to occlude MCA was recorded. The clips were then removed and normal blood flow was identified

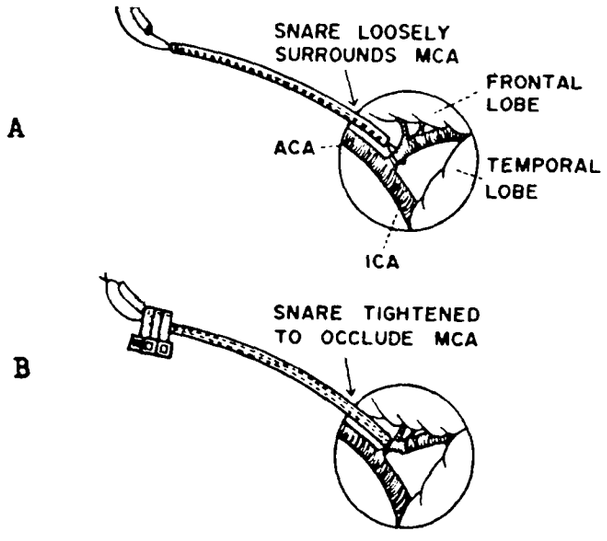

Fig. 1. Schematic drawing of the implanted snare ligature device (A: before occlusion, B: after occlusion).

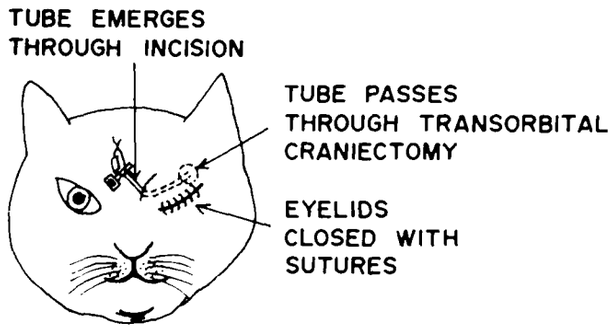

Fig. 2. In the unanesthetized state, snare ligature is tightened to occlude the left MCA.

under the microscope. Protecting the exposed cortex with the gelfoam and muscle pieces, the orbit was filled with methyl metacrylate resin and the wound was closed. After recovery from surgery requiring 4 to 5 days of hydration, feeding, antibiotics and wound care, cats without evidence of infection or neurological deficits were selected for measurement of rCBF.

Measurement of rCBF

Four hours before the study the animals were lightly anesthetized with intramuscular ketamine and transfemoral arterial and venous catheters were inserted to monitor the blood pressure and to sample the arterial blood. Rectal temperature was maintained between 37° and 38°C. Under local anesthesia the scalp was incised to make 2 mm burrholes using high-speed electric drill through which electrodes were placed at both sides of MCA territory and marginal zone under microscopic control. Each electrode was 250 micron in diameter, 30 mm in length, composed of epoxy-insulated platinum(90%)-iridium(10%) wire with a 0.5 mm terminal tip. Hydrogen clearance curve was then obtained after abrupt cessation of inhalation of hydrogen gas through a nasopharyngeal teflon tube at the rate of 1.5 liter/min. The microcomputer was programmed to calculate rCBF from the clearance value for an average 2 minutes after the initial 45 seconds, using so-called the initial slope index method. Control values were determined by repeating three to four times. Animals were again saturated with hydrogen and the MCA was occluded by pulling and tightening the snare ligature with application of previously noted number of Heifetz clips. The rCBF was measured immediately following MCA occlusion and 1, 2, 4, 8, 12 and 16 hours after the occlusion.

Histopathological examination

The animals were sacrificed after rCBF measurements and immediately thereafter 25 ml of 2% triphenyl tetrazolium chloride (TTC) solution was injected through each ICA. After injection of 0.5 ml of Evan's blue solution through the left ICA to confirm the occlusion of ipsilateral MCA, the brain was removed and immersed in 10% buffered formalin solution for two weeks, then the whole brain was sectioned in the coronal plane at 5 mm thickness. Infarcted area of the hemisphere was examined in the two sections which cross the optic chiasm and the mammilary body levels. The percentage of the infarcted area which was unstained by 2% TTC solution two sections were added and divided by in two. The animals were divided into two groups according to the size of infarct. Group A consisted of 18 aniamls (72%) of which the infarcted area was larger than 10% of ipsilateral coronal sectional surface of hemisphere and Group B consisted of 7 animals (28%), in which infarcted area was 10% or smaller.

RESULTS

Systemic Variables

The blood pressure and pulse rate decreased slightly immediately after MCA occlusion and then slowly increased to the level of control values. There were no obvious changes in arterial blood gases either before and after the occlusion. Data in which $PaCO_2$ difference were greater than 10 torr were eliminated from the anlaysis. There were no significant changes in systemic variables between Group A and Group B.

Changes of rCBF

Table 1 shows the average rCBF values before and after MCA occlusion recorded from the various anatomic locations of Group A and B. Before MCA occlusion the rCBF of ilpsilateral MCA territory of Group A was 61.2 ± 5.9 ml/100g/min, while Group B was 68.9 ± 6.3 ml/100g/min. On the contralateral MCA territory, the rCBF of Group A was 74.4 ± 11.2 ml/100g/min and that of Group B was 73.5 ± 9.2 ml/100g/min. There were no statistical differences between both groups ($p > 0.05$). After occlusion, the average rCBF of MCA terriotry in Group A reduced markedly to 6.51 ± 2.2 ml/100g/min which was below the ischemic flow threshold, while Group B decreased to 32.6 ± 2.23 ml/100g/min, showing a significant difference between two groups ($p < 0.01$). Postocclusion rCBF of Group B at MCA territory remained greater than the flow threshold of synaptic transmission failure (16-20 ml/100g/min). On the contralateral MCA territory, rCBF of both groups decreased initially, however, in Group A, CBF reduced a little less than Group B (Fig. 3 and 4). In Group A, the flow gradually decreased till the 8th hour when it reached 44.7 ml/100g/min as the lowest value and then increased slowly to the control value at the 16th hour after occlusion (Fig. 3). However in Group B, rCBF decreased more to reach 35.7 ml/100g/min at the postocclusion 16th hour, which was 45% reduction of control value (Fig. 4) and

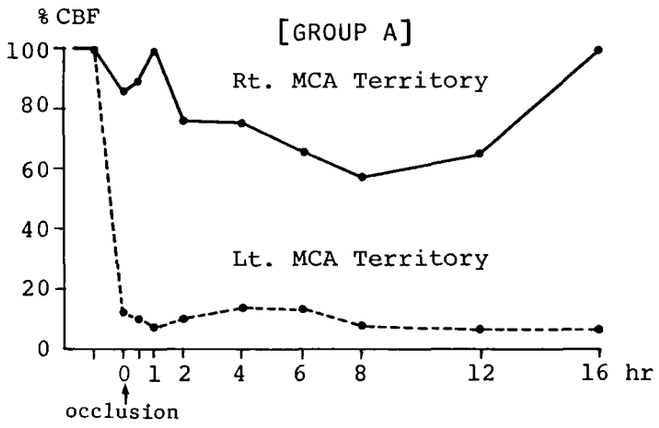

Fig. 3. Pattern of rCBF changes in group A on both MCA territories. Lt MCA territory shows profound ischemia and persists throughout the experiment. Regional CBF of Rt MCA territory decreased for 8 hours then subsequently increased to approximately control value in 16 hours.

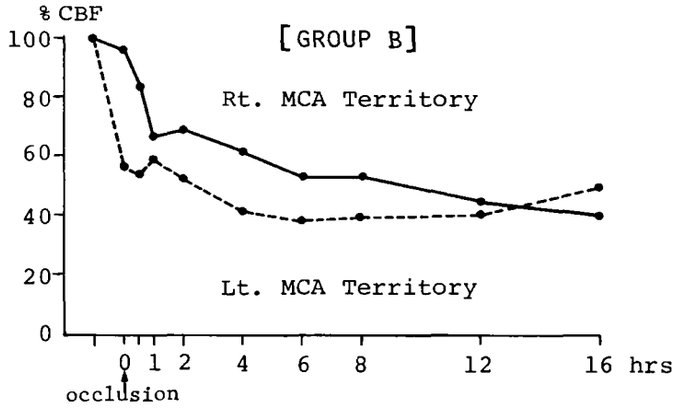

Fig. 4. Pattern of rCBF changes in group B on both MCA territories. Regional CBF of Lt MCA territory decreased less than that of Group A, maintaining more than 10% of control value. Regional CBF of Rt MCA territory decreased more than that of group B and remained low until 16 hours.

there was a statistically significant difference ($p < 0.01$). On the marginal zone, results were qualitatively similar but were less marked (Fig. 5 and 6). On the contralateral marginal zone, there was also a similar pattern of change in rCBF showing that, in Group A, rCBF returned almost to the preocclusion vaue (71.2 ml/100g/min) at the 12 to 16th hour after MCA occlusion. It was obvious, however, rCBF in Group B did not increased to reach the level of control value. This peculiar finding that contralateral reduction of rCBF in unilateral focal ischemia can be regarded as 'interhemispheric diaschisis'. Furthermore, it was obvious that there is significant difference in patterns of changes in rCBF between two groups which were divided according to size of infarct. As shown above, in Group A, of which ipsilateral rCBF decreased markedly, rCBF in contralateral hemisphere reduced far less than Group B in which mild ischemia and much smaller infarct were associated. These findings suggest that interhemispheric diaschisis have close relationship with the development of cerebral focal ischemia and infarction.

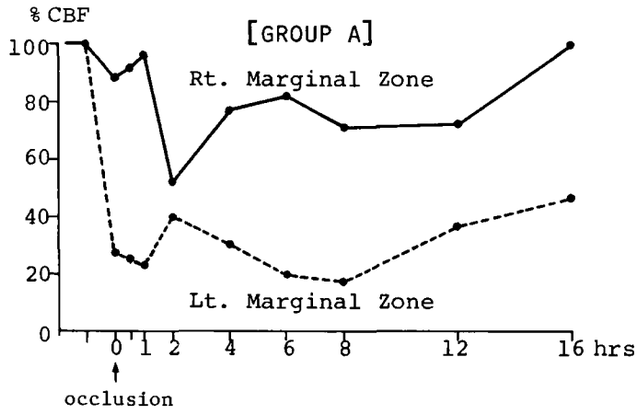

Fig. 5. Pattern of rCBF changes in group A on both marginal zones. This shows similar but less marked change than MCA territory. Regional CBF of Rt marginal zone almost returned to control value in 16 hours.

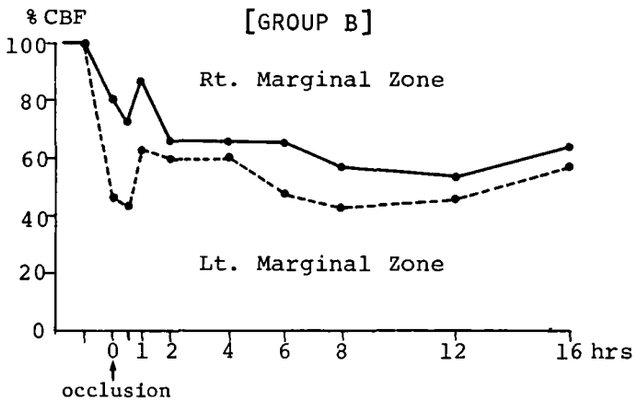

Fig. 6. Pattern of rCBF changes in group B on both marginal zones. This shows similar but less marked change than MCA territory. Regional CBF on both sides remained low until 16 hours.

DISCUSSION

Diaschisis, the peculiar phenomenon in which the local cerebral damage causes the functional depression in a remote area of the central nervous system presumably by a transneural mechanism, was first described by Von Monakow in 1914 based on clinical observations and was later more clearly demonstrated by Kempinsky (1958) in experimental animals. He also suggested the possible role of the corpus callosum in this process. The first report of bilateral reduction of cerebral blood flow in patients with unilateral cerebral infarction was that of Kempinsky *et al.* in 1961. However, for the study of diaschisis, there have been limitations in preparing the ideal animal model of cerebral infarction and in the measurement of cerebral blood flow. In the present study we used the unanesthetized cat model implanting transorbital snare ligature in the intact cranium for sudden occlusion of MCA. This was a modification of the transorbital approach of O'Brien and Waltz (1973) and considered by many as the ideal procedure for the study of cerebral ischemia, since it is less traumatic and resembles the state of the human brain at the time of the development of ischemic insult and the effect of anesthesia on cerebral blood flow can be eliminated.

For the measurement of cerebral blood flow we adopted the hydrogen clearance technique because it has the following advantages: the H_2 clearance can be determined in any tissue where a small platinum electrode can be inserted, a multiple flow determination can be obtained from the same tissue site over a long time period, blood flow can be estimated by the clearance rate of H_2 independently of the absolute amplitude of the H_2 signal and it is among the most inexpensive blood flow monitoring techniques available. It also has some disadvantages in that it may cause local injury and consequent alteration of blood flow by the implantation of an electrode. Furthermore the H_2 clearance curves are often polyexponential raising serious questions concerning the use of a single

Table 1. Regional cerebral blood flow before and after transorbital occlusion of middle cerebral artery[1)]

	Control	0	30 min	1 hr	2 hr	4 hr	8 hr	12 hr	16 hr
Ipsilateral									
MCA territory									
Group A	61.2±5.9	8.6±2.4**	7.0±2.6**	6.8±2.3**	6.9±3.1**	7.1±2.6**	5.5±2.5**	5.1±3.1**	5.1±2.3**
Group B	68.9±6.3	34.3±4.3**	32.5±3.6**	35.8±3.9**	31.1±4.2**	29.8±4.1**	30.1±3.2**	32.4±2.6**	34.9±4.3**
Marginal zone									
Group A	58.1±6.0	17.4±3.6**	16.2±3.1**	14.3±3.6**	23.3±4.8**	18.6±4.2**	12.2±2.6**	22.1±4.6**	27.3±4.4**
Group B	65.3±7.0	31.6±6.3**	30.1±4.3**	42.3±4.0**	39.2±5.8**	40.5±6.2**	27.4±3.6**	29.1±4.6**	37.9±6.3**
Contralateral									
MCA territory									
Grop A	74.4±11.2	67.3±4.9*	68.4±6.9	73.9±7.9	60.3±8.1**	61.2±5.1**	44.7±3.9**	52.1±6.0**	74.4±8.1
Group B	79.5± 9.2	72.0±7.7	71.3±4.9	55.3±4.8**	57.8±3.6**	48.3±5.4**	45.3±6.4**	38.1±7.1**	35.7±4.9**
Marginal zone									
Group A	73.4± 8.0	66.2±8.9**	67.3±8.0*	69.8±6.2	37.8±7.7**	59.4±6.2**	52.1±3.2**	34.5±4.2**	71.2±9.0
Group B	76.2± 9.4	59.3±5.5**	57.2±5.6**	64.8±7.1*	51.8±4.4**	49.6±5.7**	42.7±3.2**	35.8±4.4**	45.7±8.0**

[1)]Values are means ±SD(ml/100g/min). Statistical difference from control: *$P < 0.05$ **$P < 0.01$

exponential clearance rate for approximating blood flow. In our study the computer was programmed to calculate blood flow only by a monoexponential curve so that the exact location of the electrode was confirmed in the cerebral cortex. In order to reduce contribution from O_2 changes, we made the electrode to be polarized to +650 mV. At +650 mV polarization the sensitivity of the platinum electrode to O_2 is less. To circumvent artifacts due to recirculated arterial H_2 we discarded the first 1 to 2 minutes of the clearance data and used only the next 2 to 3 minute segments of the clearance curve (Aukland *et al.* 1964; Young 1980).

We checked the blood flows of the bilateral MCA territories and the marginal zones simultaneously, before and after the occlusion of the MCA, up to 16 hours repeatedly. As indicated by our results, before the MCA occlusion the regional cerebral blood flows of both groups on the ipsilateral hemisphere and contralateral hemisphere were similar ($p > 0.05$). After occlusion, ipsilateral MCA territory of group A showed profound ischemia that persisted below 10.0ml/100g/min throughout the experiment and reached 5.1 ± 2.3 ml/100g/min after 16 hours which is the low limit of cellular membrane failure. But group B showed about 40% of control value and in 16 hours it recorded 34.9 ± 4.3ml/100g/min which was much higher than the threshold of synaptic transmission failure. There was a significant statistical difference between two groups ($p < 0.01$). On the contralateral MCA territory the rCBF decreased for 2 hours in both group A and B, 60.3 ± 8.1 ml/100g/min, 57.8 ± 3.6 ml/100/min, respectively. In group A, however, the contralateral rCBF began to increase later and at 16 hours it reached the level of control value, 74.4 ± 8.1 ml/100g/min, while in group B it remained low and reached 35.7 ± 4.9 ml/100g/min, 45% of control value. There was a significant statistical difference ($p < 0.01$). On the marginal zone the rCBF of the ipsilateral territory was less markedly decreased and at 16 hours the rCBFs of group A and B were 27.3 ± 4.4 ml/100g/min and 37.9 ± 6.3 ml/100g/min, respectively. Contralateral marginal zone showed a pattern similar to that of the MCA territory but it was less marked. The rCBF of group A also increased to control level in 16 hours, 71.2 ± 9.0 ml/100g/min while the rCBF of group B remained low, 45.7 ± 8.0 ml/100g/min. In the present study both groups of animals showed decrease in rCBF of contralateral MCA territory and marginal zone.

These data could be clearly explained on the basis of interhemispheric diaschisis. This was in accordance with the findings of Hoedt-Rassmussen and Skinhoj (1964). But there was some difference in the pattern of rCBF changes between group A and B. Although both groups reduced immediately following occlusion, in group B the ipsilateral rCBF did not decrease to the ischemic threshold which can develop cerebral infarction. On the contralateral side of group B, rCBF decreased more than that of group A. This finding is a little different from that of Naritomi (1983). In his experiment of transtentorial diaschisis using gerbils the cerebellar blood flow was decreased only in the group in which the cerebral blood flow decreased below 15 to 20 ml/100g/min which induced cerebral ischemia after carotid occlusion. But in our study contralateral CBF of group B also decreased and even more decreased than that of group A. We may explain this by the species differences of animals used for experiment and locations for measurement of CBF. There were no other reports which compared the changes of contralateral rCBF between two groups as we did. As to the idea of the time course of this contralateral depression of blood flow, the decrease of rCBF in the healthy cerebral hemisphere was found to occur even at 30 minutes after the onset of unilateral cerebral ischemia in experimental aniamls (Reivich *et al.* 1977) and became progressively remarkable during the first several days of the ictus in patients with cerebral infarction (Slater *et al.* 1977; Wise *et al.* 1983). Other report showed it may persist 2 to 3 weeks after when the flow of the healthy side returned to normal (Meyer *et al.* 1970). In our results it reduced immediately, but in group B it returned to normal within 16 hours as compared to group A. Although we did not measure long enough, we can suggest that collateral circulation as well as decrease of CBF of contralateral hemisphere can compensate the flow of ipsilateral hemisphere in group B. Yet this interpretation is only speculative and further investigations are needed to clarify the detailed reason for the difference between group A and B.

The clinical significance of diaschisis remains unknown. Bossmann *et al.* (1980) said diaschisis is not only a simple functional phenomenon but also a progression of maturity in the ischemic state. Although a detailed mechanism of this phenomenon has not been fully understood to date, the nerve pathways connecting the two remote

areas seem to play an important role in the functional depression of the secondary regions (Kempinsky 1958). The best explanation for these findings is a local functional depression of neuronal activity due to the interruption of afferent fiber pathways (Hoedt-Rasmussen and Skinhoj 1964). Recent experimental studies have shown that after focal cerebral ischemia of the unilateral cerebral hemisphere, neurotransmitters such as catecholamine may be reduced in both hemispheres. Such widespread change in neurotransmitters may also play a role in the development of diaschisis (Kogure *et al.* 1975; Welch *et al.* 1977). Unilateral hemispheric damage with reduced metabolism causes transhemispheric reduction of metabolism and a consequent reduction of blood flow on the contralateral side (Meyer *et al.* 1970). After the development of PET, the occurrence of diaschisis in multiple sites following hemispheric infarction is well established, such as in the cerebellum, the thalamus, and the brain stem (Baron *et al.* 1981; Martin and Raichle 1983; Naritomi 1983; Wise *et al.* 1983). Crossed cerebellar diaschisis was first reported by Baron et al. in 1981. He performed a positron emmision tomographic study using O_{15} inhalation in patients with supratentorial cerebral infarction and found a reduction of oxygen consumption as well as blood flow, in the cerebellar hemisphere contralateral to the side of the infarct. The present authors consider that a more detailed investigation upon the measurement of rCBF and metabolism for a longer period of time and the determination of its relationship, if any, to the development and resolution of ischemic state is necessary to understand the pathophysiology of diaschisis and the development of ischemia.

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=국문초록=

대뇌반구간 기능해리 현상에 관한 연구

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급성기 뇌경색시 대뇌반구간의 기능해리현상을 실험적으로증명하고 그 임상적 의의를 규명할 목적으로 고양이에 올가미봉합사를 이용한 동맥폐쇄장치를 경안와접근법으로 중대뇌동맥에 설치한후 마취하지않은 상태에서 급격히 중대뇌동맥을 폐쇄시키고 시간경과에 따른 뇌혈류량의 변화를 폐쇄전과 폐쇄후 16시간까지 양측 중대뇌동맥 영역과 그 변연부에서 수소정화기법으로 측정하였다.

뇌경색의 크기가 대뇌반구 절편상 10%이상인군(A)과 그 미만인군(B)으로 나누어서 혈류량의 변화와의 관계를 고찰하여 다음 결과를 얻었다.

1. 폐쇄전 동측 중대뇌동맥 영역의 국소혈류는 A군 61.2 ± 5.9 ml/100g/min, B군 68.9 ± 6.3 ml/100g/min이고 반대측 중대뇌동맥영역의 국소혈류는 A군 74.4 ± 11.2 ml/100g/min, B군 79.5 ± 9.2 ml/100g/min로서 양측 및 각군간에 통계학적 유의한 차이는 없었다.

2. 중대뇌동맥 폐쇄후 A군은 혈류량이 급격히 감소하여 평균 6.51 ± 2.2 ml/100g/min로 떨어졌고 이는 신경세포막의 부전을 초래하는 한계혈류치의 범위에 들었으며 B군은 평균 32.6 ± 2.23 ml/100g/min으로 떨어졌다.

3. A군, B군 모두에서 반대측 혈류량이 감소하여 대뇌반구의 기능해리현상을 반영하였고 B군에서 좀더 감소하였다.

4. 그러나 A군, B군의 반대측 혈류 감소 양상에는 차이가 있어 A군은 폐쇄후 8시간까지 혈류가 감소했으나 그후 점차 상승하여 폐쇄후 16시간에는 대조치로 다시 상승하였으며, B군에서는 계속 감소된 상태였다. 중대뇌동맥 변연부에서도 같은 양상을 보였다.

5. 이는 뇌경색이 10%이하였던 B군에서 반대측 혈류감소가 더욱 심하고 16시간까지 계속 감소상태가 유지되었던것으로 B군에서는 잘 발달된 측부순환과 함께 기능해리현상이 폐쇄측의 허혈을 일부 보상해주는 가능성을 시사하였다.