"Low Perfusion Hyperemia" Following Middle Cerebral Arterial Occlusion In Cats of Different Age Groups

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SUMMARY Thirty-one cats were divided by age into 3 groups, young (Y), middle (M) and old (O). Continuous recordings of focal cerebral blood volume (CBV) and frequent measurements of mean transit time of blood (t) were made from the Sylvian opercula after ischemia was produced by transorbital clipping of the middle cerebral artery at its origin (MCA occlusion). Control recordings were made simultaneously from the corresponding area of the contralateral cerebral hemisphere.

MCA occlusion temporarily stopped cerebral blood flow (CBF) in the area supplied by the ipsilateral MCA, as indicated by a rapidly decreasing CBV and complete disappearance of hemodilution curves. Within 30 sec, CBF resumed with a dilatation of the vascular bed and reappearance of hemodilution curves through newly developed collateral channels. Despite a low CBF, below half the control, CBV recovered, overshooting the control level. The appearance of hyperemia in the ischemic area was statistically significant. Such "low perfusion hyperemia" was slower in appearance and of more diverse magnitude in group O than in group Y. This suggested that aging may lead to a decrease in rapidity of the vascular response to ischemia and impair the integrity of collateral vessels.

THE PARADOXICAL appearance of focal hyperemia in the infarcted brain of patients with occlusive cerebrovascular disease and in the ischemic region of experimental animals has been reported. Angiographic findings such as early venous filling and capillary blush correlated with regionally increased blood flow, and of red venous blood on the ischemic brain surface, lends support to the existence of hyperemia.

Recently, we have developed a photoelectric method for continuously measuring focal cerebral blood volume (CBV) in situ. By this method, it is also possible to measure the mean transit time of blood through tissue (t), and hence to separate focal cerebral blood flow into 2 flow parameters of CBV and t. Using our method, we studied the age-dependent appearance of transient hyperemia in the core areas of ischemia. The hyperemia, termed by us "low perfusion hyperemia," is discussed in relation to the luxury perfusion syndrome, its pathogenesis, its age-dependent appearance, and subsequent ischemic cerebral edema.

Methods

Thirty-one cats of both sexes weighing 2.0-4.2 kg were divided by age into young (Y = 9), middle (M = 14) and old (O = 8), according to the scoring system shown in table 1. All the cats were anesthetized with 50 mg/kg body weight of alpha-chloralose and 500 mg/kg body weight of urethane, and immobilized with atracurium chloride.

Tracheal intubation was performed and respiration was controlled with a Harvard respirator (model 662). The femoral artery and femoral vein were catheterized and a femoral cannula was inserted for repeated manual injections (serial injector, Hirasawa Co., Tokyo) of 0.5 ml of saline to produce hemodilution curves in the tissue of both hemispheres. Transorbital occlusion of a middle cerebral artery (MCA) at its origin was effected with a miniature Mayfield clip during continuous recording of the focal CBV from the core area (Sylvian opercula) of the ischemic region (this record is referred to below as CBV; i = ipsilateral), and simultaneously from the corresponding area in the opposite hemisphere (CBV; c = contralateral). In 2 cases (Nos. 13(O) and 15(M)) an additional recording was made from the peripheral area of the ischemic region (parasagittal gyrus). The records were scaled for relative changes of CBV assuming Eb = 1.2, as described in our previous paper (equation 6). For the value of L, the thickness of the tissue layer measured at autopsy was used. Concomi-
TABLE 1 Scheme for the Age Scoring of Cats

<table>
<thead>
<tr>
<th>Item</th>
<th>Score</th>
<th>Points to check</th>
</tr>
</thead>
<tbody>
<tr>
<td>Teeth</td>
<td>0-5</td>
<td>deciduous (milk) (0)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>striped (1-2)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>tartar coated (3)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>worn (4)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>toothless or edentated (5)</td>
</tr>
<tr>
<td>Skull</td>
<td>0, 1, 2</td>
<td>porosity and thickness</td>
</tr>
<tr>
<td>Skin</td>
<td>0, 0.5, 1</td>
<td>tightness upon needle insertion</td>
</tr>
<tr>
<td>Hair</td>
<td>0, 0.5, 1</td>
<td>loss of hair</td>
</tr>
<tr>
<td>Body weight</td>
<td>0, 0.5, 1</td>
<td>below 2.8 kg = 0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>above 3.3 kg = 1</td>
</tr>
<tr>
<td>Foot pad</td>
<td>0, 0.5, 1</td>
<td>cornification</td>
</tr>
</tbody>
</table>

Total score: 0-4 = young, 4.5-6.5 = middle, 7-10 = old age group.

Tant and frequent measurements of the mean transit time (I) of blood through the tissue were made by detecting hemodilution curves with the same photosensing units in 17 cases (Nos. 1(M), 2(M), 3(Y), 4(O), 5(M), 8(Y), 9(M), 14(M), 16(Y), 18(M), 20(Y), 21(O), 22(O), 23(M), 25(Y), 29(Y), and 30(O)). Values of I obtained by the area-over-height method were further used for the calculation of local cerebral blood flow (CBF), as \( 60 \cdot \text{CBV}/t \). For the calculation, it was assumed that the control value of CBV, was 6.3 vol%, and that the influence of aggregate formation by red blood cells (RBC aggregation) as we have described below was nil.

The limitations of our photoelectric method should be mentioned. As described elsewhere, the method can be used to determine CBV when flow changes are in the physiological range or above. CBV values would be subject to some error with significant flow decreases such as in experiments involving arterial occlusion. This occurs because the optical density of the tissue becomes influenced not only by the blood content but also by the flow-dependent formation of reversible RBC aggregation. Under hypoxic conditions, the changes in the optical density of the tissue caused by the 2 factors are opposite in direction: increased CBV in the tissue will increase the optical density, while the formation of RBC aggregates will decrease it. The CBV values described below may thus be underestimated due to RBC aggregation, since blood flow decreased greatly. No correction is introduced here because our conclusions for the present purpose are not materially affected. The factor of RBC aggregation does become of crucial importance when attempts are made to estimate quantitatively the reduction of the cerebral vascular bed after arterial occlusion by the present method.

In 5 cats, the orbit was closed in a water-tight manner with dental cement after clipping the artery, and the intracranial pressure (ICP) was continuously recorded using a strain-gauge transducer connected to a flexible thin-walled polyethylene catheter inserted epidurally and positioned in the vicinity of the parieto-temporal region. In the remaining 26 animals, the orbit was left open. The total duration of the operative procedure averaged about 3 h. The experiment was terminated by unclipping the MCA, upon which various degrees of reactive hyperemia were elicited. Autopsy was performed in all cats at the end of the experiment for macroscopic estimation of the brain swelling, and for measurement of the depth of the lamp, i.e., the thickness of the measured tissue layer.

Results

Details of Typical Cases

Figure 2 illustrates a typical protocol for changes in CBV, and CBV, recorded simultaneously before and during MCA clipping (No. 14(M)). The occlusion of MCA produced a rapid decrease in CBV, which was followed by a gradual restoration and overshoot above the pre-occlusive level. The maximum increase in CBV, was attained at 1 h after occlusion. The degree of hyperemia was +1.6 vol% above the pre-occlusive level. A hemodilution curve was produced by saline injection into the ipsilateral carotid artery before MCA occlusion, and appeared as a downward deflection on the CBV record, but it disappeared immediately after the occlusion (record not shown in the figure). However, it began to reappear with time. The blood flow calculated from CBV, and I, under the hypemic condition was 38.2 ml/100g/min, while the corresponding value for the opposite hemisphere was 99.4 ml/100g/min. After transient hyperemia with low perfusion, CBV, began to decrease progressively, with accompanying prolongation and eventual disappearance of the hemodilution curves. The mean arterial blood pressure (MABP) increased in this...
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FIGURE 2. Discrete examples of continuous recordings of \( CBV \) and \( CBV_e \) before and during left MCA occlusion in a middle age cat. Note the sudden decrease in \( CBV \) at clipping and the low perfusion hyperemia at 1 h after occlusion. Hemodilution curves appear as downward deflections: the times of saline injection into the carotid artery and mean transit times calculated from the curves are indicated.

animal from 110 mm Hg in the control condition to 131 mm Hg in the hyperemic condition. It then increased further to 160 mm Hg, probably as a result of the Cushing effect. \( CBV_e \) for the healthy hemisphere remained relatively unchanged until a sudden decrease with disappearance of hemodilution curves. The EEG then became flat and the animal died of cerebral herniation at 10 h after the occlusion.

A continuous recording at a slower chart speed is shown in figure 3, where ICP was recorded in conjunction with \( CBV \) (No. 24(Y)). After an initial decrease, \( CBV \) began to recover and then increased up to the maximum point of hyperemia. It should be noted that the occurrence of hyperemia was much earlier than in the above animal (No. 14(M)) from the middle age group. The ICP record at this time showed a broadly parallel change with \( CBV \). However, after the transient hyperemia, the time courses of \( CBV \) and ICP began to diverge: ICP continued to increase and then leveled off, while \( CBV \) shifted to a decrease. SABP was slightly increased with the increase in ICP. Autopsy was performed at 4 h after occlusion. The ischemic hemisphere was found to be swollen and pale with compressed vessels in the flattened gyri. The water content of the ischemic tissue estimated by the conventional drying method was 83% and 77% for 2 specimens taken from the ischemic area where \( CBV \) records were made, and 77% and 75% for control specimens taken from the corresponding region of the healthy control hemisphere.

The paradoxical appearance of a markedly dilated MCA and the well developed collateral channels on

FIGURE 3. Correlation between ICP and \( CBV_i \) in a young cat. Both ICP and \( CBV_i \) increased in parallel up to the low perfusion hyperemia. The subsequent progressive decrease in \( CBV_i \), in spite of a persistent increase in ICP, may indicate compression of the vascular bed and capillary squeezing by "brain swelling" (see Discussion).
FIGURE 4. Anterior view of formalin treated hemispheres of an old cat which died several hours after left MCA occlusion. Note the paradoxical dilation of the left MCA and well developed collateral channels from the anterior cerebral artery on the parasagittal gyrus.

the parasagittal gyrus of an old cat (No. 31(O)) which died several hours after MCA occlusion, are illustrated in figure 4. Since the main trunk of the MCA remained occluded, arterial blood appeared to have flowed in reverse. In this animal, the brain swelling was not marked. In general, brain swelling and vasodilation in the ischemic hemisphere appeared to be incompatible in the closed skull.

Occurrence of Hyperemia

Figure 5 summarizes the results of 29 experiments for CBV,. Two of the 31 animals were excluded from the figure since in one cat (No. 31(O)) CBV, records could not be obtained because of technical failure, and in the other (No. 25(Y)) the increase in CBV, was so large that it showed statistical difference from the remainder of the values. In the latter animal hemorrhage was found at autopsy in the recording area of the cortex. As shown in figure 5, the time courses of CBV, after MCA occlusion varied greatly. However, close inspection of individual records revealed a broad triphasic change consisting of an initial decrease, an increase and then a second decrease. The initial decrease was statistically significant ($p < 0.001$). A subsequent increase in CBV,, or hyperemia, occurred in 22 out of the 29 animals. The mean increase in CBV, above the pre-occlusive level was $0.66 \pm 0.92 \text{ vol\%}$, which was statistically significant ($p < 0.01$). Its mean appearance time was $45.8 \pm 50.7$ min after the occlusion. When the data were analyzed according to the 3 age groups, a marked age influence on the hyperemia and its appearance time was found as shown in table 2. Hyperemia occurred in all 8 animals from the young group ($p < 0.001$), in 10 out of 14 cats from the middle age group ($p = 0.05$), but in only 4 out of 7 cats from the old age group. Its appearance time was faster in the young age group ($12.9 \text{ min as mean}$) and slower in the old age group ($85.8 \text{ min as mean}$). This difference was statistically significant ($p < 0.05$). A triphasic pattern was also observed in the 2 records of CBV, from the peripheral area of the ischemia, although the changes were of
Table 2: Effect of Aging on Hyperemia and Its Appearance Time Following MCA Occlusion

<table>
<thead>
<tr>
<th></th>
<th>Young age group (8)</th>
<th>Middle age group (14)</th>
<th>Old age group (7)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CBV&lt;sub&gt;H&lt;/sub&gt; increase (mean ± SD)</td>
<td>1.02 ± 0.48 vol%*</td>
<td>0.44 ± 0.87 vol%†</td>
<td>0.67 ± 1.18 vol%</td>
</tr>
<tr>
<td>Appearance time (mean ± SD)</td>
<td>12.9 ± 11.6 min</td>
<td>44.6 ± 46.9 min</td>
<td>85.8 ± 69.6 min</td>
</tr>
</tbody>
</table>

* *p < 0.001.
† tp = 0.06.

lower magnitude than those concomitantly recorded from the core areas.

The second decrease in CBV<sub>H</sub> which completed the triphasic pattern occurred in all animals where observations were continued. However, the mode of decrease was found to be complicated. Some cats showed a slow and slight decrease followed by serpentine fluctuations around the baseline level until completion of the experiment; others revealed a downhill course to severe ischemia. The final values of CBV<sub>H</sub> thus indicated considerable variation in the degree and extent of ischemia.

Changes in CBV<sub>C</sub>

Figure 6 summarizes the time course of CBV<sub>C</sub> following MCA occlusion. There was little change in mean CBV<sub>C</sub> in spite of the ischemic insult to the opposite hemisphere. In one animal (No. 14(M)), CBV<sub>C</sub> showed a catastrophic fall to disastrous ischemia.

Changes in I<sub>t</sub> and CBF<sub>I</sub>

Discrete time courses of the reciprocal of mean transit time (1/I<sub>t</sub>) and CBF<sub>I</sub> after MCA occlusion in 17 animals where measurements of I<sub>t</sub> were made, are shown in figure 7. The time scale in each is adjusted to give coincidence at the time of appearance of low perfusion hyperemia. As demonstrated by the mean change, MCA occlusion produced a decrease in CBV<sub>H</sub> of -1.11 ± 0.49 vol% (p < 0.001) and in 1/I<sub>t</sub> from 0.25 ± 0.04 s<sup>-1</sup> to 0.01 ± 0.02 s<sup>-1</sup> (p < 0.005). CBF<sub>I</sub> was thus calculated to decrease from 92.8 ± 15.7 ml/100g brain tissue/min to 4.7 ± 8.6 ml/100g brain tissue/min (p < 0.001). The minimum level was zero in 13 of the 17 animals, and a mean of 14 ml/100g brain tissue/min in the remaining cats (5(M), 15(18(M)), 16(25(Y)) and 29(23(M)). However, within 30 sec or so, the CBF<sub>I</sub> level tended to recover in all. Although CBV<sub>H</sub> exceeded the control level up to hyperemia, the changes in CBF<sub>I</sub> were not necessarily dependent on those in CBV<sub>H</sub> since 1/I<sub>t</sub> was overwhelmingly low. In some animals I<sub>t</sub> was again prolonged after the reappearance of hemodilution curves, contributing to discrepant changes in CBF<sub>I</sub> and CBV<sub>H</sub>. At the maximum point of hyperemia of +0.72 ± 1.03 vol% (p < 0.02) which occurred at 46.9 ± 57.2 min in the 17 selected animals, 1/I<sub>t</sub> was still as low as 0.10 ± 0.05, and CBF<sub>I</sub> was therefore 40.9 ± 18.6 ml/100g brain tissue/min or 44.0% of the control value. The low values showed statistically significant difference from the control level (p < 0.001).

Figure 6. Summary of observed CBV<sub>C</sub> changes after contralateral MCA occlusion. The time scale is logarithmic.
The values of $1/t_i$ and $CBF_i$ in different age groups for the control, minimum point, and low perfusion hyperemia are shown in Table 3. There were no marked age differences in these flow parameters except for vascular responses to ischemia indicated by dissociated appearance times of low perfusion hyperemia, which seemed to be more exaggerated than those in the others.

**MABP and ICP**

In spite of variability in individual animals, the mean values of MABP showed only negligible changes before and during MCA occlusion. They were 109.4 ± 30.6 mm Hg at the control stage, 108.8 ± 32.3 mm Hg immediately after and 110.9 ± 30.2 mm Hg at the maximum of the hyperemic condition.

The change in ICP was, in general, similar to that reported by Hayakawa and Waltz: a transient decrease immediately after occlusion, followed by a gradual increase. The initial decrease in ICP persisted for 30 sec to 1 min and was seen in all 5 animals where ICP recording was carried out. All 5 ICP records revealed an increase following this initial decrease, although the degree and extent of the increased ICP showed great variability.

**Discussion**

The data reported above clearly support the concept of a paradoxical appearance of hyperemia in the cerebral regions made ischemic by permanent MCA occlusion. Any artifact can be dismissed on the basis of the stable record of $CBV_e$ obtained from the

**Table 3**

<table>
<thead>
<tr>
<th></th>
<th>Young age group (6)</th>
<th>Middle age group (14)</th>
<th>6.3</th>
<th>Old age group (7)</th>
</tr>
</thead>
<tbody>
<tr>
<td>$CBV_i$ (vol%)</td>
<td>Control*</td>
<td>6.3</td>
<td>6.3</td>
<td>6.3</td>
</tr>
<tr>
<td>Minimum</td>
<td>5.2 ± 0.5</td>
<td>5.2 ± 0.5</td>
<td>5.1 ± 0.5</td>
<td></td>
</tr>
<tr>
<td>Maximum</td>
<td>7.5 ± 0.4</td>
<td>6.9 ± 0.9</td>
<td>8.5 ± 1.4</td>
<td></td>
</tr>
<tr>
<td>(Appearance time)</td>
<td>(9.3 ± 6.0 min)</td>
<td>(41.7 ± 30.7 min)</td>
<td>(112.5 ± 90.8 min)</td>
<td></td>
</tr>
<tr>
<td>$1/t_i$ (s⁻¹)</td>
<td>Control</td>
<td>0.25 ± 0.06</td>
<td>0.24 ± 0.03</td>
<td>0.24 ± 0.03</td>
</tr>
<tr>
<td>Minimum</td>
<td>0.01 ± 0.02</td>
<td>0.03 ± 0.03</td>
<td>0.0</td>
<td></td>
</tr>
<tr>
<td>At maximum $CBV_i$</td>
<td>0.08 ± 0.04</td>
<td>0.13 ± 0.06</td>
<td>0.08 ± 0.02</td>
<td></td>
</tr>
<tr>
<td>$CBF_i$ (ml/100g/min)</td>
<td>Control</td>
<td>95.3 ± 21.5</td>
<td>91.9 ± 10.5</td>
<td>90.8 ± 11.9</td>
</tr>
<tr>
<td>Minimum</td>
<td>3.2 ± 6.4</td>
<td>8.3 ± 10.5</td>
<td>0.0</td>
<td></td>
</tr>
<tr>
<td>At maximum $CBV_i$</td>
<td>35.0 ± 17.7</td>
<td>51.0 ± 19.2</td>
<td>30.5 ± 3.8</td>
<td></td>
</tr>
</tbody>
</table>

*The control values were obtained from reference 10.*
Healthy control hemisphere of the same cat. Waltz observed hyperemia or supranormal flow in ischemic cerebral cortex after MCA occlusion in cats. He attributed the hyperemia to rises in SABP affecting dysautoregulated vessels in the ischemic region. This was not the case with our results since SABP as a mean remained unchanged before and during the MCA occlusion. According to Waltz, hyperemia is equivalent to increased flow above control. However, hyperemia in the strict sense represents an excess of blood in the tissue. Such hyperemia cannot be recognized by flow studies alone, but requires measurement of blood volume. When compared with the luxury perfusion syndrome (LPS) reported by Lassen, hyperemia is found to differ as follows: 1) its early appearance at a few minutes to several hours after occlusion, as opposed to the reported occurrence of LPS at later than one and a half days, 2) its appearance even in the core areas of ischemia, as opposed to the observation of LPS mainly in perifocal areas, 3) the low flow (less than half of control), as opposed to supranormal flow in LPS, and 4) the accompaniment of dark deoxygenated coloration in the venous blood, as opposed to a frequent association of red venous blood in LPS. Based on these differences, hyperemia is tentatively termed by us "low perfusion hyperemia."

The pathogenesis of such low perfusion hyperemia may be explained as follows. The first step upon MCA clipping is a full stoppage of blood flow, followed by 3-dimensional formation of RBC aggregates (rouleau networks of red blood cells) in the vasculature peripheral to the site of occlusion of the artery and in the pertaining veins. Such RBC aggregation causes changes in the local blood viscosity (the non-Newtonian property of blood), which would later give rise to unexpected and peculiar blood flow through the vasculature in the ischemic region. The next step is an accumulation of acid metabolites in the ischemic cerebral tissue under hypoxic conditions, which causes the vessel walls to become extremely flaccid. The time lag between the stoppage of flow and commencement of the vasoparalysis has been estimated by one of the present authors (NT) as approximately one minute. Meanwhile, blood supply to the ischemic region may begin from the neighboring areas via collateral communicating channels. However, owing to the extreme flaccidity of the arterial walls or complete vasoparalysis, and the relatively high venous outflow resistance with RBC aggregated blood, a ballooning of the vascular bed and therefore pooling of blood in the ischemic region tends to result. The intraluminal pressure of the occluded vasculature will then be gradually increased as the vascular walls become tensioned by the ballooning together with the further development of collateral channels. When the intraluminal pressure reaches a certain level exceeding the shear stress to disperse RBC aggregates in the venous system, blood flow will resume through the ischemic tissue. However, flow resistance would remain shifted to the venous side due to the compressed venous drainage system with increased ICP in addition to the enormous vasodilation in the arterial tree. The increase in blood content with the ballooning of the vascular bed and perhaps recruitment of capillaries, would extend up to low perfusion hyperemia. The mechanisms involved in the development of collateral circulation are not yet clearly understood. One widely accepted hypothesis is that a sudden decrease in arterial pressure due to occlusion and subsequent development of a hydrodynamic pressure gradient may provide the collateral circulation with a driving force, and that a concomitant increase in acid metabolites diffusing out from the ischemic tissue may serve to open collateral channels by exerting a vasodilatory action. An increase in blood flow in the perifocal area of ischemia has been reported by many investigators. However, the present CBV records from the perifocal area were somewhat discrepant with this finding, showing less marked hyperemia than that from the core area of the ischemic lesion. It appears likely that vasodilatation in the perifocal area may occur mainly in the leptomeningeal vessels but less in the parenchymal vasculature. A similar discrepancy was reported by Blair and Waltz, who observed dissociated changes in flow in the superficial cortical microvasculature and in deep regions of the brain, using the autoradiographic technique. In these circumstances, regional flow may be apparently increased, while the regional blood content in the tissue (CBV) may not be greatly increased. There seems as yet to be no adequate explanation for the persistent opening of collateral channels continuing after vasoactive substances from the acid metabolites would have been expected to be washed away through increased blood flow. It remains uncertain why and how the "remote demand and supply" mechanism for the collateral circulation works in the perifocal area when the regular route of blood supply to a local area of the cerebral tissue has been interrupted.

The efficiency of development of the collateral circulation appears to be broadly age-dependent, as judged from the relative rapidity of appearance of low perfusion hyperemia in the present experiments. A decrease of CMRO, with age as well as a reduced cerebrovascular reactivity in the elderly could play a role in delaying its appearance in old animals. However, when the age-dependence is analyzed according to the extent of hyperemia, the CBV values of the young age group tend to be clustered in a narrow range while those of the old age group are widely scattered (as shown by the large standard deviation). Such diversity of results in the old age group is demonstrated in figure 5, where one CBV trace (13(O)) remained low with little tendency to recover while another (22(O)) revealed enormous hyperemia. Aging may impair the integrity of collateral vessels. The final step in the low perfusion hyperemia is its disappearance. At this phase, variations in ICP accompanying a decrease in CBV may provide a key to the hemodynamic change in the brain. A persistent increase in ICP mirroring the progressive decrease in CBV (as shown in fig. 3) indicates that compression of
the vascular bed or capillary squeezing by swelling of the local tissue due to ischemic cerebral edema has occurred, leading the tissue into severe ischemia, no-reflow and irreversible infarction. Such strong swelling may be driven by an increase in osmotic potential in the ischemic cerebral tissue, which was found by us to be as high as 600 mm Hg in vitro.22

Low perfusion hyperemia of transitory nature was thus found to appear spontaneously in the ischemic cerebral region of cats following MCA occlusion. The concept of low perfusion hyperemia may provide a cornerstone in the temporal classification of the pathophysiology of cerebral ischemia.

References

"Low perfusion hyperemia" following middle cerebral arterial occlusion in cats of different age groups.
M Tomita, F Gotoh, T Amano, N Tanahashi and K Tanaka

Stroke. 1980;11:629-636
doi: 10.1161/01.STR.11.6.629

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