Arterial Supply of the Feline Motor Cortex

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SUMMARY The arterial supply of the feline motor cortex is derived from both the anterior and middle cerebral arteries. The anterior cerebral artery supplies most of cortical area 6 (premotor cortex), the intrafundal cruciate and medial postcruciate cortex, (hindlimb motor cortex), and the midline and medial portions of the sensory areas 3a–7. The middle cerebral artery supplies the lateral prefrontal cortex, (lateral premotor cortex), precruciate and lateral sigmoid cortex, (forelimb motor cortex), and the remainder of the coronal and Sylvian cortical areas.

THE ARTERIAL SUPPLY of the feline brain has been studied with particular reference to the skull, the carotid rete system and as a model for cerebral infarction. However, details of the specific distribution of the anterior and middle cerebral arteries in reference to the motor cortex and associated cytoarchitectonic areas are not available. This information is of importance in investigations of this cortical region or clinical evaluation of deficits where blood supply is a consideration. The following studies were carried out to investigate the pattern of arterial supply to the pericruciate motor cortex of the cat. The carotid rete system was also investigated and some variations from the literature described.

Methods

Seventeen adult cats of either sex were utilized for dissection and study of the arterial system. The animals were anesthetized with an intraperitoneal injection of Nembutal (40 mg/kg), exsanguinated with transcardial saline infusion, and perfused with 10% buffered formalin using conventional methods.

The left common carotid was cannulated in seven animals and red liquid latex injected into the carotid arterial tree. In 10 animals the origins of the anterior and middle cerebral arteries were selectively cannulated and injected with different colored liquid latex. The latex used (Wards, red, blue) was too viscous to cross the capillary beds.

The dissections were performed with the aid of a stereodissecting microscope and the use of microdissection neurosurgical instruments. These enlargements and fine instruments were particularly helpful in the dissection of the rete system. The progressive stages of the dissection were photographed on ASA 25 Ektachrome film with a Nikon camera equipped with a 50 mm macro lens and 2x extension tube. The arterial pattern observed in each brain was sketched on a scale model of the cortex according to the measurements and relative locations of the sulci.

Results

The blood supply to the feline motor cortex is derived from both the middle and the anterior cerebro arterial arteries. The anterior and middle cerebral arteries branch from the cerebral carotid artery at the level of the optic chiasm. The middle cerebral artery courses dorsally along with the Sylvian fissure supplying cortical branches to the pyriform lobe and lateral hemisphere including the coronal, sigmoid, and orbital gyri. The anterior cerebral artery courses rostrally from the optic chiasm supplying small branches to the medial olfactory tract along the way. At the rostroventral extent of the olfactory lobe, the anterior cerebral artery follows a sigmoid course to become the artery of the corpus callosum. At this point, branches supply the fornicate and marginal gyri. In 7 of 17 dissections, the main trunk of the anterior cerebral artery coursed dorsally from the medial end aspect of the cruciate sulcus to the cingulate sulcus. Rostral to the genu an interhemispheric anastomotic anterior communicating artery was observed in one brain.

In 10 brains the anterior cerebral artery and the middle cerebral artery were differentially injected; e.g., anterior cerebral artery with red latex, middle cerebral artery with blue latex. Examination of these preparations revealed that the middle cerebral artery supplied the lateral prefrontal, prefrontal, precruciate and lateral sigmoid cortex. The lateral postcruciate gyrus was supplied by the middle cerebral in 5 of 10 preparations; anastomotic connections between middle cerebral artery and intrafundal branches of the anterior cerebral artery were observed in the other 5 preparations.

The anterior cerebral artery supplied the prefrontal, medial precruciate and medial one-third to two-thirds of the postcruciate gyrus; this pattern is summarized in figure 1.

The anterior cerebral artery supplies the entire extent of the intrafundal cruciate cortex, typically from 2 major branches which leave the parent artery (fig. 2A). The first branch (more rostral) enters the sulcus and adheres to the superficial precruciate intrafundal cortex. Two to three mm into the sulcus this small artery branches extensively and supplies the major extent of the precruciate intrafundal cortex. The branches which course rostrally up and out of the sulcus Anastomose with the middle cerebral artery on the extrasulcal precruciate bank. In 3 of 11 animals the artery sent a single branch across the sulcus to supply a portion of the superficial postcruciate intrafundal cortex or anastomose with vessels from the second branch.

The second branch enters the deep portion of the
fundus and adheres to the postcruciate cortex. After a short distance (2-3 mm), a branch is given off which crosses the sulcus and supplies the deep portion of the precruciate intrafundi cortex, or anastomoses with vessels from the first branch. The deep postcruciate intrafundal artery arborizes extensively and supplies the postcruciate cortex. The extrasulcal bank of the medial postcruciate gyrus is supplied by branches which course from the intrafundal arteries. The branches on the medial postcruciate anastomose with splenial branches of the anterior cerebral artery and laterally with the middle cerebral artery.

The blood supply with reference to the cytoarchitectural zones drawn according to Hassler and Muhs-Clement is summarized in figures 1 for the midline and 2B for the intrafundal cortex.

The hindlimb motor cortex is known to be localized to the medial postcruciate and intrafundal cruciate cortex, which is supplied by the anterior cerebral artery. The forelimb motor cortex has been localized to the lateral sigmoid cortex, which is supplied by the middle cerebral artery. These patterns are summarized in figure 3.

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**Figure 1.** Dorsomedial view of Left Frontal Hemisphere, dotted lines mark cytoarchitectural regions.

**Figure 2.** A) Dorsomedial view of Left Frontal Cortex with Cruciate Sulcus opened and the Anterior Cerebral arterial pattern sketched. B) Left Cruciate Sulcus opened, with cytoarchitectural regions and arterial patterns marked with reference to Hassler and Muhs-Clement and observations of the arterial pattern in 10 dissections.

**Figure 3.** Summary of arterial pattern to motor cortex with forelimb and hindlimb regions marked according to stimulation experiments.1, 23
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Discussion

Variations from reports in the literature are as follows:

1) A common trunk for the internal carotid, ascending pharyngeal and occipital arteries was not found as described by Hüllemann. Instead, an initial common trunk giving rise to the occipital and ascending pharyngeal artery arose 3-5 mm caudal to the origin of the internal carotid artery.

2) Inconsistency exists concerning the nomenclature for the following vessels. The internal ophthalmic artery according to Sisson and Grossman, originates from the external rete and follows the optic nerve intracranially to anastomose with its fellow artery. It continues anteriorly as the single anterior meningeal artery and may receive an anastomosis from the median chiasmatic artery. In one cat, the median chiasmatic artery branched to join the internal ophthalmic and an anterior cerebral artery. Davis and Story, Kamijyo and Garcia, and Daniel et al.* referred to the internal ophthalmic as the internal ethmoid while Martinez referred to it as the interreticular artery. Davis and Story, in half their animals, found a small artery from the cerebral carotid which they called the internal ophthalmic artery. We observed this artery in one of 11 brains.

3) In one animal, the anterior cerebral artery branched to form the artery of the corpus callosum for the contralateral hemisphere.

For technical and economic reasons the cat is a popular animal for studies involving the nervous system. It is known that there are substantial differences between the feline and primate with regard to the fissural patterns and functional organization of the motor-sensory cortex. Middle cerebral artery occlusion in cats produces patterns of motor deficits which are surprisingly similar to those observed following this procedure in primates, e.g. limb weakness with the forelimb deficits being the most severe. The knowledge of the blood supply to the specific functional regions of the primate motor-sensory cortex is available, and the neurological findings are compatible with the observation that the hindlimb motor-sensory cortex is located in the superior-medial precentral gyrus which is supplied by the anterior cerebral artery. The more lateral forelimb motor cortical region is supplied by the middle cerebral artery.

The specific correlations between the arterial supply and the functional organization of the feline motor-sensory cortex were not available before and have been revealed in the present report. These findings are compatible with the neurological findings from the arterial occlusion studies. The anterior cerebral artery was observed to supply the intrafundal cruciate cortex and the medial postcruciate gyrus. This region is known to be involved in motor functions of the hindlimb. The middle cerebral artery supplies the more lateral sigmoid cortex which is specifically involved in motor functions of the forelimb. Occlusion of this vessel correspondingly results in the most severe deficits of the forelimb.

Therefore, the combination of knowledge of:

a) the neurological findings following arterial occlusion,
b) the functional organization of the feline motor-sensory cortex,
c) the correlated arterial supply to the feline motor cortex, indicates that the cat is a comparatively good animal model for middle cerebral artery occlusion studies with reference to the motor cortex, with special considerations in mind.

References

Brain Extracellular Ion Composition and EEG Activity Following 10 Minutes Ischemia in Normo- and Hyperglycemic Rats

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SUMMARY Hyperglycemia severely impairs the outcome from cerebral ischemia. In order to sort out whether impaired brain ion homeostasis contributes extracellular [K⁺], [Ca²⁺], and [H⁺] concentrations, [K⁺]ₑ, [Ca²⁺]ₑ, and [H⁺]ₑ of brain cortex, as well as the EEG, were monitored during and after 10 minutes of complete cerebral ischemia in normo- and hyperglycemic rats. In both groups, the EEG-activity disappeared in 10-20 seconds of ischemia, at a time when [K⁺]ₑ, [Ca²⁺]ₑ, and [H⁺]ₑ started to increase. After about 1.5 min, [K⁺]ₑ showed an abrupt increase and [Ca²⁺]ₑ a steep decrease in the normoglycemic group. In the hyperglycemic group the same event took place after about 3 min of ischemia. pH decreased to 6.6 and 6.1 in the normoglycemic and hyperglycemic group, respectively. Following the ischemic episode, [K⁺]ₑ reached pre-ischemic level after 4 min, [Ca²⁺]ₑ after 13 min, and [H⁺]ₑ after 30 min in both groups. Recovery of the EEG, however, was clearly different in the 2 groups. EEG-activity reappeared later in the hyperglycemic group and showed after one hour a pattern of burst-suppression activity while the normoglycemic group showed asynchronous activity resembling the control pattern.

It is concluded that high glucose content in brain prior to ischemia — and hence lower brain pH during ischemia — does not interfere with the return of normal extracellular ion composition after cerebral ischemia, whereas the return and pattern of EEG activity is severely affected.

Stroke, Vol 12, No 2, March-April 1981

IT HAS BEEN SHOWN that glucose administration prior to cerebral ischemia seriously aggravates the clinical outcome of rats. Also, the return of EEG activity after ischemia in hyperglycemic rats is significantly delayed. The reason for the detrimental effect of the high glucose level in blood, and hence in brain, has remained unknown, but since brain glucose during ischemia is converted to lactic acid, it was suggested that the augmented lactic acidosis in the hyperglycemic rats was the culprit. How the lactic acidosis affects brain function in the post-ischemic period and what cellular mechanism it eventually will damage is presently unknown.

To find out whether the delayed EEG appearance is caused by a delayed normalization of ion gradients across cell membranes in brain, we measured [K⁺]ₑ and [Ca²⁺]ₑ in brain cortex during and after ischemia. We also determined the severity of brain acidosis by simultaneous measurement of extracellular pH during ischemia and ability of the brain to normalize pH after ischemia.

Material and Method

Preparation of Animals

The experiments were performed on male Wistar rats, weighing approximately 350 g. All rats were fasted overnight.

The rats were initially anesthetized with ether and intubated with a steel cannula. Following relaxation with suxamethonium, they were mechanically ventilated with 1% halothane and 35% O₂ in air. Polyethylene catheters were placed in the femoral artery and vein. The ventilation was adjusted to maintain arterial Pco₂ between 32-40 mm Hg. Rectal temperature was kept at 37°C by a heating lamp. One group of rats received an i.p. injection of 50% solution

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doi: 10.1161/01.STR.12.2.233
Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1981 American Heart Association, Inc. All rights reserved.
Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/12/2/233

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