Ischemic Cortical Lesions After Permanent Occlusion of Individual Middle Cerebral Artery Branches in Rats

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Our study describes the anatomy of the middle cerebral artery (MCA) in 65 Sprague-Dawley rats and the spatial distribution of ischemic cortical lesions caused by occluding major MCA branches. The rats characteristically had at least two major MCA branches, frontal and parietal. Many rats had additional branches supplying the pyriform and temporal cortices. Permanent occlusion of the frontal or parietal branches combined with 30 minutes of bilateral carotid artery occlusion produced visible Evans blue dye uptake by ischemic cortical areas after 24 hours. No lesions distal to the occlusion were apparent in 38% and 43% of rats with frontal and parietal branch occlusions, respectively; small lesions contiguous with the occlusion site were observed in 38% and 32% of the rats. Only 6% of the frontal and 7% of the parietal branch occlusions produced isolated distal infarcts as expected if these branches were end-arteries. Blood flow was reversed in arteries distal to the occlusion. We conclude that extensive collateral connections of the frontal and parietal MCA branches with other arterial systems protect the anterior and posterior cortical regions. In contrast, occlusions of the pyriform branch of the MCA invariably caused infarcts in the frontopyriform region. In about one third of the rats, frontal or parietal branch occlusions produced lesions involving much of the proximal MCA territory; the frontopyriform region was most consistently affected. Combined, these data suggest that the pyriform MCA branch is an end-artery and that the cortical region it supplies is prone to ischemic damage resulting from any reduction of blood flow through the main MCA trunk. Seventy-five percent of rats with parietal branch occlusions suffered infarcts in the frontopyriform region. We propose that these paradoxical lesions represent an intrahemispheric steal phenomenon, that is, blood is diverted by the posterior cerebral artery feeding collaterals to the ischemic parietal cortex, reducing blood flow in the main MCA trunk. (Stroke 1988;19:870–877)
Rubino and Young

MCA Branch Occlusion in Rats

A 46%  B 26%  C 17%

D 5%  E 5%  F 2%

FIGURE 1. Major middle cerebral artery (MCA) branching patterns (A, standard) are shown schematically with frequency of occurrence in 65 rats. In all but one rat, MCA bifurcated into frontal and parietal branches above rhinal fissure. Frontal and pyriform branches were present in a minority of rats.

decrease bronchial secretions. A 2-cm vertical skin incision was made midway between the ear and eye. The temporalis muscle was divided vertically and severed from its mandibular connections. This approach exposed the skull where the frontal bone joins the temporal bone (coronal suture). Under x 25 magnification, a 5 x 5 mm area of frontal bone was thinned with a dental drill and removed with a 25-gauge needle and fine forceps, exposing the MCA. After opening the dura with a bent 25-gauge needle, the chosen branch of the MCA was dissected free of associated pia and arachnoid.

A 3-cm midline incision over the submandibular glands exposed the carotid arteries. The glands and the underlying sternohyoid muscle were vertically divided, revealing the trachea and common carotid artery 6 mm lateral to the trachea. A length of 2-0 silk was slipped around each carotid artery after separating it from the vagus nerve. At this point, the previously exposed branch of the MCA was occluded with bipolar radiofrequency current applied through jeweller’s forceps. Saline irrigation during coagulation reduced adherence of the branch of the MCA to the forceps. The temporalis muscle was laid over the craniectomy site, and the skin was closed with surgical clips. The silk suture surrounding each carotid artery was drawn into thin polyethylene tubing, clamping the artery and preventing blood flow, then removed after 30 minutes of occlusion.

Identification of Lesion Sites and Types

Evans blue (2 ml, 4% saline solution) was administered through a femoral vein immediately before or shortly after occlusion of the MCA branch. We found in previous studies that the volume of dye produces dense stains of the infarct site visible on the cortical surface; it has no apparent detrimental effects on survival or recovery of the rats from MCAo. Twenty-four hours after MCAo, the rats were given an overdose of pentobarbital and perfused with 5% formalin fixative. The brains were removed and immersed in 5% formalin.

Intravenous Evans blue binds rapidly to serum albumin. Its presence in brain tissue after ischemia indicates breakdown of the blood–brain barrier (BBB), allowing passage of albumin into the brain. Because BBB breakdown may be delayed, we chose to assess the brains 24 hours after MCAo, well beyond the time when BBB breakdown begins to occur. Very low blood flow may also reduce tissue staining because the dye is not brought to the tissue. To reduce this possibility, we injected Evans blue before or shortly after MCAo so that the brains were perfused with dye-containing blood for a long time, ensuring reliable staining of most areas of the brain that have suffered ischemic damage.

In all rats studied, the MCA had at least two major branches, the frontal and parietal branches. These two branches bifurcate from the main MCA trunk (Figure 1). This Y configuration of the MCA yields three clearly demarcated regions, the frontopryiform, anterior, and posterior regions. We categorized the lesions into three types, primary, secondary, and tertiary. Primary lesions were defined as blue-stained areas immediately distal to and around the occlusion site, usually along the occluded artery; we excluded primary lesions that measured <4 mm² since the radiofrequency current applied to the artery may have directly damaged the
cortex. Secondary lesions were defined as blue-stained areas distal to the occlusion that extended at least 4 mm beyond the occlusion; secondary lesions resembled end-artery lesions. Tertiary lesions were defined as blue-stained areas not contacting the occlusion site and located distant from the occluded artery; tertiary lesions of all sizes were included in the analyses. Lesions that were contiguous with primary lesions and spread into one adjacent region were defined as extensive, a combination of primary and tertiary lesions. Lesions that involved all three regions were defined as catastrophic, a combination of secondary and multiple tertiary lesions.

**Experimental Groups and Data Analyses**

A total of 65 rats were studied. The frontal MCA branch was occluded approximately 2 mm from the bifurcation in 16 rats; the parietal branch was likewise occluded in 28 rats; the pyriform branch was occluded in three rats. In five rats, only the carotid arteries were occluded. Rats that did not survive for 24 hours or that suffered significant hemorrhage during the procedure were excluded from the analyses. We used $\chi^2$ analyses to compare the distributions of primary, secondary, tertiary, extensive, and catastrophic lesions produced by each branch occlusion group; $p<0.05$ indicated significance.

The surface areas of lesions were calculated from their radii when they were circular or from the major and minor axes when they were elliptical. Lesion borders were sufficiently distinct in all rats to allow measurements with a precision of 0.5 mm. Both hemispheres were assessed. In addition to examining the cortical surface, coronal sections were made of the brain to ascertain the depth of staining of the brain.

**Results**

**MCA Branching Patterns**

The main trunk of the MCA originated in the circle of Willis, proceeded dorsally, and crossed a prominent vein on the inferior lateral aspect of the brain. This vein ran in the rhinal fissure, which extends from the front of the pyriform cortex to the occipital lobe; we called this vein the rhinal vein. Two small symmetric branches, running anteriorly and posteriorly, originated from the MCA 1–3 mm below the rhinal vein and accompanied the vein in the rhinal fissure; we called these branches the anterior and posterior rhinal branches. The MCA trunk usually bifurcated 2–6 mm above the rhinal vein, forming two major branches that we called the frontal and parietal branches. In addition, two other branches often split from the main MCA trunk. One supplied the frontopyriform cortex, and we called it the pyriform branch; the other supplied the temporal cortex, and we called it the temporal branch. The four branches above the rhinal vein formed three common and three rarer branching patterns. Figure 1 illustrates the different patterns and their incidences.

In 30 of 65 rats (46.2%), the MCA had a standard Y configuration (Figure 1, A). After bifurcation from the main trunk, the frontal branch proceeded superiorly and frontally and often divided several times while approaching the midline. The parietal branch proceeded occipitally, paralleling the midline and giving off multiple branches that covered much of the cortical surface behind the MCA trunk and above the rhinal vein. The parietal branch divided more frequently than the frontal branch. Some of its branches extended inferiorly; in two rats, these branches anastomosed with the posterior rhinal MCA branch. The frontal and parietal branches approached the anterior cerebral (ACA) and posterior cerebral (PCA) arteries, respectively.

A second pattern resembled the standard pattern except for the presence of a pyriform branch, which makes a right-angle split from the main MCA trunk above the rhinal vein (Figure 1, B). The pyriform branch usually originated several millimeters proximal to the frontal-parietal bifurcation. In rats in which the pyriform branch originated closer to the frontal-parietal bifurcation, the pyriform branch left the main MCA trunk at a sharper angle of $\sim 50^\circ$. The pyriform branch seldom divided further. The area supplied by the pyriform branch appeared isolated from other major surface vessels. This pattern was seen in 17 of 65 rats (26.1%).

A third pattern resembled the second, with the addition of a temporal branch (Figure 1, C). The temporal branch was often a mirror image of the pyriform branch, originating from the main MCA trunk at right angles and proceeding along the temporal lobe. Eleven of 65 rats (16.9%) showed this pattern. The temporal branch anastomosed with the posterior rhinal branch in two rats.
Several other branching patterns were seen infrequently. A rare branching pattern had a temporal branch stemming from the main MCA trunk without a pyriform branch (4.6%) (Figure 1, D). We observed three rats (4.6%) in which the main MCA trunk followed the path of the parietal branch but gave off several major frontal branches and few temporal branches (Figure 1, E). We encountered one aberrant rat (1.5%) in which the frontal and parietal branches split immediately after leaving the circle of Willis (Figure 1, F).

**Primary and Secondary Lesions**

To investigate the cortical territories supplied by individual MCA branches, we selectively occluded the frontal branch 2 mm above the bifurcation in 16 rats; the parietal branch was likewise occluded in 28 rats. Approximately equal proportions of the major branching patterns were chosen for occlusion in the two groups. The pyriform branch was selectively occluded in three rats. Due to the low incidence of temporal branches, we were not able to assess the effects of selective temporal branch occlusions.

Blue-stained cortical areas resulting from branch occlusions ranged from light to deep blue. Darker stains were associated with necrosis and tissue softening. Even the lighter-stained areas could be clearly distinguished from surrounding unstained tissues. Evans blue usually penetrated the cortex to depths of 2–4 mm. The stained areas were conic, with deep apexes. Subcortical structures were never stained, and the corpus callosum frequently bounded stained regions. No staining was seen in the contralateral hemisphere in any rat.

Frontal branch occlusions did not produce primary or secondary lesions in six of 16 rats (37.5%) (Figure 2). In six of 16 rats (37.5%), small blue-stained areas were centered around the occlusion site and proceeded 3 mm along the frontal branch distal to the occlusion. The mean ± SD surface area of primary lesions was 9.5 ± 5.1 mm². A large wedge-shaped secondary infarct (surface area 21.6 mm²) was seen distal to the occlusion in only one of 16 rats (6.3%).

Occlusion of the parietal branch likewise caused limited and inconsistent staining of cortical areas distal to the occlusion (Figure 2). In 12 of 28 rats (42.9%), there were no primary or secondary lesions in the cortex distal to the occlusion. In nine of 28 rats (32.1%), parietal branch occlusion produced small circular or elliptical primary lesions that extended along the artery for 2–3 mm distal to the occlusion site. In two of 28 rats (7.1%), we observed large wedge-shaped secondary lesions that began at the occlusion site and ran distally for 6 mm along the parietal branch. The mean ± SD surface area of the primary lesions averaged 9.7 ± 3.5 mm², while secondary lesions averaged 20.0 ± 1.7 mm².

All three rats with pyriform branch occlusions had secondary infarcts. The mean ± SD surface area was 12.5 ± 3.2 mm², and all lesions were within the boundaries of the frontopyriform region.

**Tertiary Lesions and Patterns of Evans Blue Staining**

Branch occlusions frequently produced blue-stained areas outside the region supplied by the branch. Tertiary lesions in one or more regions were observed in nine of 16 rats (56.3%) with frontal branch occlusion and in 24 of 28 rats (85.7%) with parietal branch occlusion. Figures 3 and 4 compare the incidences of regional ischemic damage resulting from frontal and parietal MCA branch occlusions. The frequencies of lesions including and excluding extensive and catastrophic infarcts are given. Frontal branch occlusion produced lesions more frequently in the anterior region than in the posterior region. Parietal branch occlusion produced a higher incidence of lesions in the posterior region than in the anterior region. Considering only the anterior and posterior regions, distributions of blue staining produced by frontal and parietal branch occlusion were significantly different (p < 0.05; χ²).

Frontal branch occlusion produced an 18.8% incidence of catastrophic lesions, 18.8% incidence of extensive lesions, and 25% incidence of tertiary frontopyriform lesions. Parietal branch occlusion caused a 17.9% incidence of catastrophic lesions, 17.9% incidence of extensive lesions, and 75% incidence of tertiary frontopyriform lesions. Approximately 36% of the rats with tertiary frontopyriform infarcts resulting from parietal branch occlusions did not show staining in other regions.
Frontal branch occlusions produced only half the incidence of tertiary frontopyriform lesions compared with anterior primary and secondary lesions while parietal branch occlusions resulted in nearly twice the incidence of tertiary lesions in the frontopyriform region compared with primary and secondary posterior lesions. Frontal branch occlusions produced less than half the incidence of lesions in the frontal region compared with parietal branch occlusion. $\chi^2$ analysis of the frequency of frontopyriform lesions indicated significant differences between frontal and parietal branch occlusion ($p<0.05$).

Retrograde Blood Flow in Occluded Vessels

Blood cell movements inside the arteries were observed under high magnification with a dissecting microscope. Before occlusion, blood flow was always anterograde in the MCA branches. After occlusion, pale thrombi were visible in the occluded segment, usually occupying a 1-mm length of vessel. Within seconds of stable thrombus formation and complete occlusion in the frontal or parietal MCA branches, retrograde blood flow was typically present in the artery distal to the occlusion.

Discussion

The goals of our study were to define the anatomy of the MCA in Sprague-Dawley rats, to determine the cortical territories of the major MCA branches by occluding individual branches, and to ascertain whether individual branch occlusions combined with temporary bilateral carotid ligations cause reproducible lesions in selected cortical territories. Our results indicate that Sprague-Dawley rats have consistent and classifiable MCA branching patterns. Occlusions of individual MCA branches produced small and inconsistent Evans blue staining in regions distal to the occlusion, despite bilateral carotid occlusion. Branch occlusions sometimes produced extensive lesions covering much of the MCA territory, staining of cortex proximal to the occlusion site, and paradoxical staining of territories ostensibly unrelated to the occluded vessel.

Use of Major MCA Branches to Demarcate Cortical Regions

Rat brains have relatively smooth surfaces without visible sulci; consequently, the cortical areas cannot be defined by sulci as they are in higher mammals. Investigators usually identify cortical areas in rats by subcortical structures or by stereotactic coordinates referenced to skull sutures. Unfortunately, subcortical structures cannot be visualized from the surface, and stereotactic approaches cannot be conveniently applied to a brain after removal from the skull. There is a need for a method of identifying cortical regions in rats based on brain surface landmarks. Our study indicates that the standard MCA branching pattern can provide consistent landmarks for identification of cortical areas. Use of arterial branches for this purpose is analogous to identifying cortical areas by sulci since the branches of the MCA in humans and higher mammals run in major sulci.

The MCA in the rat usually has two to four major branches emanating from the main trunk. In order of frequency, they are the parietal, the frontal, the pyriform, and the temporal branches. All rats had at least one parietal and one frontal branch. Only 43% of the rats had a prominent pyriform branch, and 22% had a prominent temporal branch. We therefore propose using the frontal and parietal branches to demarcate three distinct cortical areas: the frontopyriform, anterior, and posterior regions. The frontopyriform region is bounded by the rhinal fissure, the frontal branch, and the main MCA trunk; this region includes the pyriform cortex and part of the frontal cortex. The anterior region is bounded by the frontal branch, the parietal branch, and the midline; it includes much of the frontal cortex, lateral aspects of the cingulate cortex, and part of the parietal cortex. The posterior region encompasses the area inferior to the parietal branch, behind the main MCA trunk and above the rhinal fissure, including part of the parietal cortex, the occipital cortex, and the superior aspects of the temporal cortex. Together, these three regions encompass 60–70% of the cortical surface of each hemisphere.

Note that we call these cortical areas regions as opposed to territories. The latter carries connota-
tions of individual blood supply. The cortexes included in each region, at least superficially, are supplied by two or more MCA branches. The superior aspects of the frontopiriform region are supplied from the surface by one or more frontal branches; the inferior aspects are often supplied by a pyriform branch of the MCA. The anterior region, located between the frontal and parietal branches, receives blood supply from both of these MCA branches. The posterior region is generally supplied by the parietal branch and occasionally by a temporal branch. We had originally hoped to define the territories of the different MCA branches by selective occlusion. The extensive collateral blood supply via retrograde flow, however, renders the concept of arterial territory meaningless, at least for the frontal and parietal branches of the MCA in rats.

Collateral Protection of MCA Territory

Coyle et al.14 correlated cortical damage from MCAo with areas of Evans blue staining. They found a sharp transition between stained and normal tissue in lesions. The borders of the stained area were hyperemic and corresponded to the edges of the histologic lesion. Our stained areas also had sharp borders. However, Coyle et al.14 observed that smaller lesions attributed to surgical damage often had areas of staining larger than areas of actual histologic damage and concluded that the blue-stained areas sometimes overestimated the actual histologic lesion. Since we used more dye than most authors (six times the amount that Coyle et al.14 used), unstained areas probably have no significant ischemic damage, and areas of even mild ischemic damage are likely to be stained by Evans blue.

The pattern of staining in our experiments indicates that MCA branch occlusions produce limited and inconsistent ischemic damage in the cortex distal to the occlusion. Figures 3 and 4 show that 37% of the frontal and 43% of the parietal branch occlusions produced no ischemic damage in the cortex beyond the occlusion. In 32% of occlusions, Evans blue uptake was limited to areas immediately surrounding the occlusion. These findings indicate that an effective collateral blood supply protects the tissue distal to the occlusion. Since the frontal and parietal MCA regions adjoin territories supplied by the ACA and PCA, the sources of the collateral blood flow are likely to be these two arteries.

Several additional observations argue that the anterior and posterior regions are extensively supplied by arterial sources other than the MCA. First, we observed retrograde blood flow in the artery distal to the occlusion. Second, Meyer et al.15 reported rapid restoration of blood supply to the MCA territory after occlusion of that artery in monkeys. Those authors concluded that the pressure differential on either side of a collateral junction was the major factor determining direction of blood flow through collaterals. Third, several investigators have reported anatomic evidence of extensive collateral connections between MCA branches and other cerebral arteries in species ranging from humans to rats.16-18 For example, Coyle and Jokelainen19 visualized MCA-ACA surface collaterals with vasodilators and latex. We conclude that the frontal and parietal MCA branches are part of a hemispheric arterial network composed of the cerebral arteries and multiple collateral vessels joining the cerebral arterial branches.

Branch occlusions sometimes produced isolated secondary lesions that resembled the infarct expected from occlusion of end-arteries. This type of result occurred in 6% of frontal branch occlusions, 7% of parietal branch occlusions, and 100% of pyriform branch occlusions. We suspect that occurrences of these types of lesions in the anterior and posterior regions represent occasional congenital failures of adequate anastomoses between the ACA and PCA to MCA branches in these regions. In the case of the secondary pyriform lesions, we conclude that the frontopiriform region is largely or perhaps even solely supplied by the MCA.

Oclusion of the main MCA trunk below the rhinal fissure produces consistent infarcts in the cortex. Many investigators1-5 have used this standard MCAo model to study ischemia. Occlusions of the main MCA trunk cause infarcts centered on the frontopiriform region.5 These infarcts are limited to proximal regions of the MCA territory and usually do not extend more than 1 mm beyond the bifurcation of the frontal and parietal branches. We suggest that the infarct in the standard MCAo model occurs in part because retrograde collateral blood flow does not reach the main MCA trunk for two reasons. First, collateral vessels have diameters smaller than the main MCA trunk. Retrograde perfusion pressure will consequently decrease in the proximal regions of the MCA. Second, retrograde blood flow is drained from the MCA by arterioles as it progresses proximally. Furthermore, the frontopiriform region is not protected by retrograde collateral blood flow. Thus, occlusion of the main MCA trunk should produce infarcts centered on the frontopiriform region and limited to the proximal regions of the MCA territory.

Effectiveness of Collateral Protection

An effective collateral vascular supply to the MCA territory carries several implications. The most important is that the concept of the MCA territory needs to be reconsidered. If, for example, occlusion of an MCA branch does not consistently produce ischemia in the cortex apparently supplied by that branch, that cortical region should not be considered solely a territory of the MCA. Our results illustrate clearly the difficulties of assigning arterial territories. The concept of arterial territory is only applicable to end-arteries, such as the pyriform MCA branch.

The small circular and elliptical primary lesions that occurred in 38% of frontal and 32% of parietal...
branch occlusions probably result from the compromise of local end-arterioles around the occlusion site. We suggest that these primary lesions result from thrombus formation at the occlusion site, preventing both anterograde and retrograde blood flow to the arterioles around the occlusion site. The primary lesions were situated around the occluded MCA branch. Some lesions were elliptical, with major axes aligned with the occluded MCA, and others had major axes perpendicular to the occluded MCA running alongside small arterial branches. Intra-arterial thrombi were often observed at the occlusion site. Such thrombi typically occupied an approximately 1-mm length of artery and appeared pale.

The collateral network clearly did not protect the cortex in the cases of catastrophic or extensive lesions. Parietal branch occlusion produced a 17.9% incidence of catastrophic lesions involving much of the MCA territory and a 17.9% incidence of extensive lesions involving adjacent cortex fed by another branch of the MCA. The frontal-parietal bifurcation is a special point in the MCA system where three sources of blood meet. We suggest that spasm of the main MCA trunk, thrombi extension into the bifurcation, and inadequate collateral blood supply are responsible for the catastrophic and extensive lesions observed in approximately one third of the rats.

**Intrahemispheric Steal**

Parietal branch occlusion combined with temporary bilateral carotid ligation produced an unexpectedly high incidence (75%) of infarcts in the frontopyriform region. We propose that the paradoxical frontopyriform infarcts result from an intrahemispheric steal of blood flow. In our experiments, temporary bilateral occlusion of the carotid arteries should reduce blood flowing into the circle of Willis. Carotid occlusion alone does not produce frontopyriform infarcts. After MCA parietal branch occlusion, the posterior region is protected by collaterals, most probably from the ipsilateral (left) PCA. Because the left PCA is located between the basilar artery and the left MCA, it may steal the blood that otherwise would supply the MCA.

Such a steal mechanism should lower blood flow to the ipsilateral (left) anterior region as well as to the frontopyriform region. Although there was a 35.7% incidence of catastrophic and extensive lesions after parietal branch occlusions, tertiary anterior infarcts did not occur as frequently as tertiary frontopyriform infarcts: 77% of the tertiary frontopyriform infarcts we encountered were not associated with lesions in the adjacent anterior region. Note, however, that the anterior region is protected by ACA-MCA collaterals. Moreover, since the right and left ACA fuse to form a common ACA trunk in rats, the left anterior region is likely to be supplied by blood from the right side through the common ACA trunk and collaterals. We therefore suggest that blood flow through the MCA to the anterior region is reduced by the steal, but that collateral blood flow from the ACA prevents this decrease from reaching critical levels. Alternatively, bilateral carotid artery occlusions can produce posterior-to-anterior blood flow gradients in the leptomeningeal circulation, which may contribute to the higher incidence of frontopyriform infarcts.

The extensive collateralization of blood vessels in the head and neck provides many opportunities for steal phenomena. Two types of intracranial steal phenomena have been extensively documented in the literature: interhemispheric and intracerebral. The interhemispheric steal phenomenon was described by Tulleken and van Dieren, who directly measured the retrograde perfusion pressure in an occluded MCA during contralateral carotid artery occlusion in cats. They found that contralateral carotid occlusion decreased retrograde perfusion pressure in the MCA collaterals, concluding that unilateral carotid occlusion produces an interhemispheric steal across the circle of Willis.

Intracerebral steal purportedly occurs when vasodilators (papaverine or CO2) are administered after occlusion of the main MCA trunk. Blood flow in the ischemic area decreases while that in the surrounding tissues increases. This has been attributed to loss of autoregulation in ischemic arterioles that do not respond to the vasodilator. Blood vessels outside the ischemic region should dilate, shunting blood from the ischemic region to the surrounding tissues. Later studies, however, challenged this steal mechanism and attributed the decreased blood flow in the ischemic region simply to a decrease in blood pressure caused by the vasodilators. When blood pressure was maintained, blood flow to the ischemic territory increased with vasodilator administration.

The intrahemispheric steal mechanism that we have proposed differs in several important respects from both interhemispheric and intracerebral steal. In our experiments, we occluded both carotid arteries while unilaterally occluding a branch of the MCA. Evans blue staining did not occur in the contralateral (right) hemisphere in any of our rats. Thus, if interhemispheric steal occurred in our experimental situation, it was insufficient to produce an infarct in the hemisphere contralateral to the MCAo. Intrahemispheric steal differs from intracerebral steal in that blood flow is "stolen" by an ischemic region from a perfused region through the circle of Willis.

**Conclusions**

Occlusions of the frontal and parietal MCA branches produce limited and inconsistent lesions in the cortex distal to the occlusion, strongly supporting previous work showing that Sprague-Dawley rats have an extensive collateral network protecting the anterior and posterior MCA regions. The frontopyriform region supplied by the MCA,
however, is not protected by collaterals. This region invariably showed infarcts when the pyriform branch of the MCA was occluded. Finally, our study uncovered an intrahemispheric steal phenomenon not reported in the literature. Occlusion of the parietal MCA branch combined with bilateral carotid artery occlusion caused a high incidence of infarcts in the ipsilateral frontopiryform region. We attributed this to steal of blood flow by the ipsilateral PCA, which provides collaterals to the posterior region. Blood flow consequently falls in the MCA branches to the anterior and posterior regions. The anterior region, however, is protected by collateral blood flow from the ACA. The frontopiryform region is not protected and therefore becomes ischemic. This may explain why occlusion of the main MCA trunk produces infarcts centered in the pyriform cortex.

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