The Arterial Patterns Associated with Internal Carotid Disease and Cerebral Infarcts

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SUMMARY In 20 necropsies with 15 stenosed and 17 thrombotic occluded internal carotid arteries there were 46 cerebral infarcts larger than 1 cm diameter. Using postmortem arteriographic and pathological techniques the patterns of the neck and brain artery systems were correlated with the situation and extent of the brain infarcts.

Massive infarcts involving two major cerebral artery territories were associated with distal internal carotid artery occlusion and grossly ineffective cervical and circle of Willis anastomoses. Isolated middle cerebral artery territory infarcts were associated with internal carotid occlusion or stenosis and impairment of the circle of Willis anastomoses, perhaps with middle cerebral artery stenosis. The pattern of adequate size arteries determined if these infarcts were total, deep central, anterior, medium or posterior partial territory infarcts.

Boundary zone infarcts were associated with internal carotid artery disease and limitation of anterior or posterior circle of Willis anastomoses. These limitations determined which boundary zones were affected. Isolated anterior cerebral artery territory infarcts were associated with bilateral internal carotid disease and an anterior cerebral artery stenosis or small caliber anterior communicating artery. Isolated posterior cerebral artery territory infarcts were associated with internal carotid disease and a direct impairment of the ipsilateral posterior cerebral artery capability.

THE FREQUENT ASSOCIATION OF STENOTIC and occlusive internal carotid artery (ICA) disease with cerebral infarction is well recognized. 1-3 Although some reports have related the nature, severity and extent of the vascular and of the ischemic lesions, 4-6 further clarification of these relationships is desirable. In this paper an analysis is made of the arterial patterns associated with the various brain infarcts in a group of necropsy cases with ICA stenosis or occlusion and large cerebral infarcts. In all cases the entire carotid, vertebral and cerebral artery systems were initially studied using a post-mortem arteriographic method, 7,8 which readily demonstrates anastomotic pathways with adequate filling of arteries distal to occlusions. The arterial lumen is also fixed distended so that the degree of lumen stenosis observed with the complementary pathological techniques is more related to that existing in vivo than that seen after the contraction of standard fixation methods. Small focal infarcts less than 1 cm are not considered in this report.

Materials and Methods

In a series of 91 patients with suspected cerebrovascular disease which were specially studied at necropsy, there were 72 with brain infarcts of which 20 were cases with severe stenoses or occlusions by thrombosis in the ICA and with cerebral infarcts larger than 1 cm in diameter. The other cases with embolic arterial occlusion or with small focal infarcts are not considered here. The 20 cases comprised 17 males and 3 females aged 41 to 86 with a mean age of 69.6 years. All showed gross neurological deficit which had persisted until death for periods varying from 2 days to 17 years. Fourteen (70%) of the patients were hypertensive as judged by either clinically recorded diastolic blood pressures of at least 110 mm of mercury or at necropsy an obvious left ventricular hypertrophy without other heart disease and a heart weight of more than 350 g for women or 400 g for men. Ten (50%) of the patients including 4 of the hypertensives had necropsy evidence of ischemic heart disease.

In all necropsies the carotid and vertebral artery systems were simultaneously injected in situ by a multiple-nozzle air-piston syringe connected to separate cannulae in the innominate, left subclavian and left common carotid arteries (CCA) as previously described. 9 The Schlesinger type warm-setting gelatin-based radiopaque medium was pumped in at simulated blood pressure and provided filling of arteries more than 100 μm diameter. After x-rays of the head and neck were taken and solidification of the injection medium had occurred the scalp was reflected, the calvarium removed and the brain taken out in the usual way. The base of the skull, the cervical spine and the neck arteries in their attached soft tissue were then excised in toto and fixed before further examination. Using the post-mortem arteriograms as a guide the entire carotid, vertebral and cerebral artery systems were studied after fixation by transverse sections of the arteries at intervals of a few mm and the lumen diameter measured with a cylindrical feeler gauge calibrated in 0.5 mm intervals. Blocks sampling all stenotic or occlusive arterial segments were examined histologically. The fixed brain was sliced coronally at 5 mm intervals and appropriate samples of cerebral arteries as well as all infarcts were also examined histologically. In each case charts were prepared showing the nature and extent of the ischemic lesion in the brain parenchyma, of the stenotic or occlusive lesions in the carotid, vertebral, basilar and cerebral arteries and of the developed anastomotic pathways for each system. The WHO
atherosclerotic classification was used to describe the type of each atherosclerotic lesion and to assess the degree of lumen narrowing as moderate stenosis (more than half diameter lumen preserved), severe stenosis (less than half lumen diameter preserved) or occlusion.

Results

The Internal Carotid Artery Lesions

Bilateral Occlusion

In 4 cases, both ICA were occluded by thrombi, 7 of which were associated with atherosclerosis and 1 with a giant cell arteritis. Of these 8 thrombi, 6 filled the ICA to the ophthalmic artery (OA), 1 the entire ICA to the middle cerebral artery (MCA) and 1 the distal ICA only.

Unilateral Occlusion

One ICA was occluded by thrombus in 9 cases, in 3 of which the other ICA was severely stenosed, in 2 of them by sinus atherosclerosis and in 1 by cervical fibromuscular dysplasia. All 9 unilateral ICA occluding thrombi had underlying atherosclerosis which was severely stenotic in all except 1 in the distal ICA. Of the 9 thrombi, 1 occluded the entire CCA and ICA to the OA origin, 4 occluded the ICA to the OA, 2 occluded the entire ICA, anterior cerebral artery (ACA), MCA, posterior communicating artery (PCoA) and posterior cerebral artery (PCA) and 2 occluded only a short distal ICA segment.

Bilateral Stenosis

In 3 cases, both ICA were stenosed by atherosclerosis at their origins and of these 6 ICA, 4 were narrowed in their cavernous part as well.

Unilateral Stenosis

In 4 cases, there was atherosclerotic stenosis of one ICA, in 3 at the origin only, but with additional stenotic lesions in the distal part in the other.

Other Artery Lesions

There were atherosclerotic lesions some of which were severely stenotic in the external carotid arteries (ECA), in the circle of Willis, in the larger cerebral artery branches and in the vertebral and basilar arteries. There were also occlusions in smaller peripheral arteries related to the cerebral infarcts. In old infarcts these were organized lesions the original nature of which could not be determined. In recent infarcts the occlusions appeared to be due to thrombosis which had occurred during local circulation failure at the time of infarction although a possible embolic basis could not be excluded. Atheromatous emboli were not found in arteries related to any of the infarcts.

The Infarcts

In 13 of the 20 cases, there were 21 large infarcts associated with the occlusion by thrombi of 17 ICA. Of these infarcts, 6 were massive infarcts (1 partly bilateral) involving ACA territory (Te)-MCATe or MCATe-PCATe, 10 infarcts were in a MCATe only, 3 infarcts were in a boundary zone (BZ), 1 infarct was in an ACATe only and 1 infarct was in a PCATe only. In 12 of the 20 cases, there were another 25 large infarcts associated with the stenosis of 15 ICA. Of these infarcts 10 were in a MCATe only, 10 were in a BZ, 2 were in bilateral ACATe, 2 were in an ACATe only, and 1 was in a PCATe only. In 3 cases, stenosis and subsequent thrombosis of the same ICA had been associated with separate episodes of infarction.

Five of the 6 massive ACATe-MCATe and MCATe-PCATe infarcts had developed within a week of death. All 4 ACATe-MCATe infarcts included the head of the caudate nucleus but the area involved on the medial aspect of the hemisphere was variable. Of the 20 infarcts in the MCATe, 6 involved all or almost all of that territory — the total infarcts of Zülch — and 5 of them were recent. There were 14 partial MCATe infarcts of which the medium sized partial MCATe infarct and 3 of the 4 anterior partial MCATe infarcts were also recent but both the posterior partial MCATe infarcts and 5 of the 7 deep central MCATe were old. Most of the 13 BZ infarctions had occurred an appreciable time before death and only 2 BZ infarcts were less than 2 months old. In all the cases with BZ infarcts there were arterial territory infarcts also and except in one case, BZ infarction had occurred prior to arterial territory infarction. In 5 cases there were 8 infarcts involving the anterior inferior, anterior superior or posterior superior parts of the MCA territory in ACATe-BZs, in 3 cases there were 3 infarcts involving the posterior inferior BZ between the MCA and PCATe and in 2 cases there were infarcts involving the ACATe-MCATe-PCATe BZs. Of the 8 cases with BZ infarcts 5 had ischemic heart disease and 3 were hypertensives, 2 of which had received hypotensive therapy but no episodes of hypotension had been recognized in any of the 8 patients. The isolated ACATe infarcts involved varying parts of the distal artery territory but not the head of the caudate nucleus. In 2 of the cases bilateral asymmetrical infarction had occurred at the same time. Only 2 of these 5 infarcts were recent. The isolated PCATe infarcts developed 6 weeks and 2 days before death and occupied the greater part of that territory.

Arterial Patterns in Massive Anterior-middle and Middle-posterior Cerebral Artery Territory Infarcts

In all 6 cases with massive infarcts involving two large cerebral artery territories there was a thrombus occluding the ipsilateral ICA distally with ineffective cervical and circle of Willis anastomoses. The arterial patterns outlined in table 1 show that of the 4 ACATe-PCATe infarct cases, 1 also had an occluded MCA trunk but in the other 3 cases there were no stenoses and no occlusive thrombi or emboli in the main MCA branches (fig. 1A). Stenoses or occlusions of the ipsilateral ACA were not found in any of the 4 cases but in the 2 cases with widely patent contralateral ICAs, the anastomosis across the anterior part of the circle of Willis was limited by a tiny anterior communicating artery (ACoA). In the other 2 cases without limitation of the anterior circle of Willis anastomotic channels, the contralateral ICA was occluded. In none of these 4

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cases did the vertebro-basilar artery (VBA) system appear able to contribute significantly to the circle of Willis because of either small PCoA or some degree of VBA stenosis.

The 2 cases with massive MCATe-PCATe infarcts had occlusion of the ipsilateral ICA, MCA and PCA with occlusion in the posterior circle of Willis anastomosis and a contralateral ICA stenosis as well. The

### Table 1: The Arterial Patterns Associated with 46 Large Cerebral Infarcts in 20 Cases with Internal Carotid Disease

<table>
<thead>
<tr>
<th>Infarcts*</th>
<th>Ipsilateral ICA†</th>
<th>Ipsilateral cervical anastomosis ↓</th>
<th>Contra-lateral ICA†</th>
<th>Anterior circle of Willis anastomosis ↓</th>
<th>Posterior circle of Willis anastomosis ↓</th>
<th>Infarcted territory arteries ↓</th>
<th>Vertebral-basilar system ↓</th>
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<tr>
<td>ACATe-MCATe</td>
<td>2 O O $ O $</td>
<td>O O − −</td>
<td>O O + +</td>
<td>− + −</td>
<td>+ + −</td>
<td>P O + +</td>
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<tr>
<td></td>
<td>2 O O</td>
<td>P P − −</td>
<td>O O + +</td>
<td>− − +</td>
<td>SS P + −</td>
<td>− − +</td>
<td></td>
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<tr>
<td>MCAte-PCATe</td>
<td>2 O O</td>
<td>− SS</td>
<td>SS MS SS</td>
<td>− − −</td>
<td>− − −</td>
<td>− − −</td>
<td></td>
</tr>
<tr>
<td>Total MCAte</td>
<td>2 O O</td>
<td>− SS</td>
<td>− − − −</td>
<td>− − −</td>
<td>− − −</td>
<td>− − −</td>
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<tr>
<td>Anterior partial MCAte</td>
<td>2 SS $ SS $</td>
<td>+ +</td>
<td>SS SS</td>
<td>+ + −</td>
<td>− + −</td>
<td>P P + +</td>
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<tr>
<td></td>
<td>2 SS MS</td>
<td>+ +</td>
<td>MS MS</td>
<td>− − −</td>
<td>P MS + −</td>
<td>− − −</td>
<td></td>
</tr>
<tr>
<td>Deep central MCAte</td>
<td>2 O** O</td>
<td>+ +</td>
<td>P SS</td>
<td>+ + −</td>
<td>MS P + −</td>
<td>+ + −</td>
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<td></td>
<td>1 SS</td>
<td>+</td>
<td>SS</td>
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<td>1 MS</td>
<td>− (ECA SS†)</td>
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<td>+ −</td>
<td>MS −</td>
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<tr>
<td>Posterior partial MCAte</td>
<td>2 O†† O</td>
<td>+ +</td>
<td>SS SS</td>
<td>+ + −</td>
<td>− + −</td>
<td>P P + +</td>
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<tr>
<td>Medium partial MCAte</td>
<td>1 MS</td>
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<td>P</td>
<td>− − −</td>
<td>P −</td>
<td>+ − −</td>
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<tr>
<td>ACATe-MCAte BZ</td>
<td>2 MS MS</td>
<td>+ +</td>
<td>P P</td>
<td>− − −</td>
<td>P P + +</td>
<td>+ + −</td>
<td></td>
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<td></td>
<td>1 SS</td>
<td>+</td>
<td>P</td>
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<td>1 SS</td>
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<td>SS</td>
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<td>2 MS MS MS</td>
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<td>1 O</td>
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<td>SS</td>
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<td>1 SS</td>
<td>+</td>
<td>O</td>
<td>+ − −</td>
<td>SS +</td>
<td>+ − −</td>
<td></td>
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<tr>
<td>MCAte-PCATe BZ</td>
<td>1 MS</td>
<td>+</td>
<td>MS</td>
<td>+ + −</td>
<td>SS −</td>
<td>− − −</td>
<td></td>
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<td></td>
<td>1 SS</td>
<td>+</td>
<td>SS</td>
<td>+ + −</td>
<td>MS −</td>
<td>− − −</td>
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<td></td>
<td>1 SS</td>
<td>+</td>
<td>P</td>
<td>− − −</td>
<td>P −</td>
<td>+ + −</td>
<td></td>
</tr>
<tr>
<td>ACATe-MCAte-PCATe BZ</td>
<td>2 O O</td>
<td>+ +</td>
<td>SS SS</td>
<td>+ + +</td>
<td>P P − +</td>
<td>− − −</td>
<td></td>
</tr>
<tr>
<td>Isolated ACATe</td>
<td>2 MS‡‡ MS</td>
<td>+ +</td>
<td>MS MS</td>
<td>+ + +</td>
<td>SS SS +</td>
<td>− − −</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2 MS SS</td>
<td>+ +</td>
<td>MS MS</td>
<td>− − −</td>
<td>SS SS −</td>
<td>− − −</td>
<td></td>
</tr>
<tr>
<td>Isolated PCATe</td>
<td>2 O</td>
<td>+</td>
<td>O</td>
<td>+ − −</td>
<td>Small −</td>
<td>− − −</td>
<td></td>
</tr>
</tbody>
</table>

*ACATe, anterior cerebral artery territory; MCAte, middle cerebral artery territory; PCATe, posterior cerebral artery territory; BZ, boundary zones. †ICA, internal carotid artery; ECA, external carotid artery; O, occlusion by thrombus; SS, severe stenosis; MS, moderate stenosis; P, patent lumen. $+$, adequate channel; $−$, limited channel. $|$ see figure 1A. ‡see figure 1B. $§$see figure 2A. **see figure 2B. ††see figure 3A. ‡‡see figure 3B.
FIGURE 1. A – Case 64 with 4 day old massive RACATe-MCA
infarct resulting from occlusion distal RICA with old
cervical LICA occlusion (another older infarct is omitted). B –
Case 26 with 2 day old total RMCATe infarct and severe steno-
sis sinus RICA with limitation of anterior and posterior circle of
Willis anastomoses.

Arterial Patterns in Middle Cerebral Artery Territory
Infarcts

In the 6 cases with total MCATe infarcts (Table 1) there was either bilateral ICA occlusion (2 cases) or severe ipsilateral ICA disease and an inadequate anterior circle of Willis anastomosis (4 cases). In 5 of the cases ipsilateral ICA occlusion had apparently precipitated infarction. In 2 of these cases the contralateral ICA was already occluded thereby preventing anastomotic utilization of the large anterior circle of Willis communication. In the other 3 of these cases the contralateral ICA sinus was stenosed, 1 also had MCA trunk stenosis and all 3 had a limited anterior circle of Willis anastomosis. In the remaining case (fig. 1B) the ipsilateral ICA sinus and ECA origin were both severely stenosed and although the contralateral ICA was widely patent the anterior circle of Willis anastomosis was limited in caliber.

In the 4 cases with anterior partial MCATe infarcts (table 1) there was bilateral ICA stenosis and an impaired anterior circle of Willis anastomosis if the contralateral ICA stenosis was but moderate (fig. 2A).

Of the 7 deep central partial MCATe infarcts (table 1), 1 infarct (fig. 2B) was associated with ipsilateral ICA occlusion and some MCA stenosis and the other 6 with bilateral ICA disease of variable severity. Four of these infarcts were associated with but moderate steno-
sis of the ipsilateral ICA but there was also ipsilateral MCA stenosis in 1 case with 3 infarcts and in another case where there was in addition severe stenosis of the ipsilateral ECA. There was no inadequacy of the anterior circle of Willis anastomosis in the cases with deep central MCATe infarcts.

Each of the 2 posterior partial MCATe infarcts (Table 1) was associated with ipsilateral ICA occlusion, contralateral ICA stenosis and a limited posterior circle of Willis anastomosis although no VBA impairment was apparent (fig. 3A). The medium sized partial MCATe infarct (table 1) was associated with moderate stenosis only of the ipsilateral ICA sinus and although there was no other artery narrowing both the anterior and the posterior circle of Willis anastomoses were of limited caliber.

The position and size of the 20 infarcts in the MCATe in these 15 cases appeared to be determined by the severity of the stenotic or occlusive carotid disease on both sides and the degree and extent of impairment of the circle of Willis anastomoses resulting from pathological changes in and from differing anatomical configurations of the arteries of the circle.

Arterial Patterns in Boundary Zone Infarcts

Of the 8 infarcts involving ACATe-MCATe BZs (table 1) 3 were associated with unilateral ICA disease and a limitation of the anterior circle of Willis and 5 with bilateral ICA disease in 4 of which there was an adequate lumen capacity in the anterior circle of Willis anastomosis although in 2 of these there was ACA or
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FIGURE 3. A - Case 60 with 8 year old posterior partial LMCA infarct and LICA occlusion (another recent infarct is omitted). B - Case 36 with 3 week old isolated bilateral ACA infarcts, bilateral moderate stenosis ICA and severe stenosis of larger RACA (other older infarcts are omitted).

MCA stenosis also. Of the 3 MCATe-PCATe BZ infarcts, 2 were associated with bilateral ICA disease and some MCA or PCA stenosis and 1 with unilateral ICA disease and limited circle of Willis anastomoses. The 2 cases with ACATe-MCATe-PCATe BZ infarcts had severe bilateral ICA and VBA disease.

In all the cases with infarcts affecting the ACATe-MCATe BZ there was ipsilateral ICA disease together with either a limitation of the anterior circle of Willis or the presence of contralateral ICA disease as well. In all the cases with infarcts affecting the MCATe-PCATe BZ there was ipsilateral or bilateral ICA disease together with a limitation of the PCA capacity as a result of either severe VBA disease, or limitation of the posterior circle of Willis anastomosis or the major PCA channel originating from the ipsilateral diseased ICA.

Arterial Patterns in Posterior Cerebral Artery Territory Infarcts

In only 2 cases was there an isolated PCATe infarct not associated with a contiguous MCATe infarct (table 1). One right PCATe infarct was a complication of a separate 2 day old right MCATe infarct associated with stenosis of the right ICA and resulted from right PCA kinking which was well demonstrated by the in situ postmortem arteriograms although not apparent in the brain after removal. The other isolated right PCATe infarct had followed a right ICA thrombosis when the major channel of the right PCA was from the ICA through a large PCoA. In both these cases with PCATe infarcts there was an underlying ICA impairment and a direct impairment of PCA capability.

Discussion

The postmortem arteriographic technique used provides a filling of the entire cervical and cephalic arterial lumina with the medium bypassing arterial stenoses or occlusions and demonstrating available anastomotic channels. These anastomoses have three distinct pathways, those between the ECA and ICA, those around the circle of Willis and those distally in the leptomeninges between the major cerebral arteries. Although the caliber of an artery is not a measure of the perfusion rate through it, the observed unavailability or limitations of the cervical and Willisian anastomotic channels in the present cases with ICA disease, do correlate with the localization and the extent of the large infarcts found in the brain. Such a correlation could not be demonstrated, however, with the less striking variations in the more peripheral leptomeningeal anastomoses which generally comprise quite small arteries less than 0.5 mm in diameter.3

A degree of ICA stenosis has been commonly reported in older subjects some of whom have no ischemic cerebral disease15-19 and as in the present large infarct cases the stenosis most often involves the artery at the sinus.1 When one carotid sinus is stenosed, there is a greater probability15 that the other is also stenosed and in a third of the present cases the stenosis was bilateral. Fisher20 reported fewer bilateral ICA stenoses but his cases included many with but small focal infarcts. A thrombus occluding a severely stenosed sinus will inevitably propagate distally21 and in the present cases, all occluding thrombi originating in the carotid sinus extended at least to the OA and some beyond although shorter thrombi have been reported in a third2 to about half3 to about half the cases with proximal ICA occlusion. In the distal ICA, atherosclerosis is common22 but severe stenosis is much less frequent than in the carotid sinus.23,24 Although thrombi appear
to develop in less severely stenosed lumina in the distal ICA. Few thrombi originate there as in the present cases and all reports of frequent occlusion of the distal ICA include many cases of embolus impacted there.\textsuperscript{4, 10, 23} The cases of this series with obvious embolic occlusion of major arteries have not been considered here but in the present cases of large infarcts associated with non-occlusive ICA disease it is possible that embolism from proximal atherosclerotic lesions may have occurred and the emboli had subsequently become displaced from the sites of impaction. Indeed, in the cases with ICA stenosis such possible embolism could have been a factor precipitating infarction — except with the BZ infarcts. Nevertheless, in the present cases of proximal ICA disease the location and extent of the large infarcts do appear directly related to the patterns of the cervical and Willisian anastomoses whether unrecognized embolism may or may not have contributed to the development of the infarction.

Blackwood et al\textsuperscript{7} found that large, rapidly fatal cerebral infarcts were commonly associated with carotid disease in the neck and Castaing et al\textsuperscript{16} noted frequent ICA occlusions beyond the bifurcation with such infarcts. Only 1 (17%) of the present 6 patients with distal ICA occlusion and massive ACATe-MCATe or MCATe-PCATe infarcts lived more than a week but 12 (60%) of the 20 infarcts involving the MCATe only, had developed more than a week before death and 3 (60%) of the isolated ACAT infarcts were older than 3 weeks. Further, all 13 BZ infarcts were at least 2 weeks old and 6 (46%) developed 2 years or more before death.

The circle of Willis is not merely a distributor station\textsuperscript{18} and Torvik and Jørgensen\textsuperscript{8} concluded that very large infarcts are the result of a total failure of the circle of Willis anastomosis. The arterial patterns in the present cases with massive ACATe-MCATe or MCATe-PCATe infarcts demonstrate inadequate cervical anastomoses and show that the latter may be due to anatomical features or to pathological changes within the circle or to severe contralateral ICA disease when the circle of Willis channels are adequate.

Torvik and Jørgensen\textsuperscript{8} also concluded that total MCATe infarcts were the result of ICA occlusion with blockage of the circle of Willis while partial MCATe infarcts usually resulted from ICA occlusion with a patent circle of Willis. In the present cases total MCATe infarcts were associated usually with bilateral severe ICA disease or with ipsilateral severe ICA disease and an impairment of the anterior circle of Willis anastomoses. Anterior partial MCATe infarcts were associated with less severe bilateral ICA disease but with an inadequate anterior circle of Willis anastomosis. Deep central MCATe infarcts were usually associated with bilateral ICA disease and a generally adequate anterior circle anastomosis although MCA disease was often a feature. The posterior partial MCATe infarcts were associated with severe bilateral ICA disease and an impairment of the posterior circle of Willis anastomosis. The medium partial MCATe infarct was associated with ipsilateral ICA stenosis and some limitation of the anterior and posterior circle anastomoses.

BZ infarcts have been described as involving frontier or border zones,\textsuperscript{6, 22, 29} last field,\textsuperscript{30} outside the supply-territory\textsuperscript{31} or — with a confusing reversal of the hydrological analogy — as watershed areas.\textsuperscript{30} In the present cases only those infarcts which appeared to overlap adjacent artery territories\textsuperscript{31} have been regarded as BZ infarcts. The ACATe-MCATe BZ infarcts were present in cases either with bilateral ICA disease or with ipsilateral ICA disease and a limitation of the anterior circle of Willis anastomosis. The MCATe-PCATe BZ infarcts were present in cases with ipsilateral ICA disease and a limitation of the PCA capacity either from VBA disease, or from an inadequate posterior circle of Willis anastomosis or when the main PCA channel came from the diseased ICA. In some cases the diseased ICA was not severely stenosed but there was no clinical evidence suggestive of an episode of severe hypotension in any of these patients with BZ infarcts. The present analysis does not support the conclusion of Romanul and Abramowicz\textsuperscript{2} that BZ infarcts are larger when due to ICA occlusion than when due to ICA stenosis although some of the infarcts they described as BZ infarcts are similar to peripheral partial MCATe infarcts in the present cases which did develop after previous BZ infarction when a stenosed ICA became occluded by thrombosis.

ACA stenosis may be readily demonstrable angiographically\textsuperscript{33, 34} but in pathological studies severe disease in the ACA\textsuperscript{3, 22, 23} has not been a constant feature in cases with ACATe infarcts. In the present cases with isolated ACATe infarcts there was bilateral ICA disease although stenosis was not always severe and there was also a limitation of the territorial perfusion channel due either to stenosis in the ACA itself or to a small caliber ACoA apparently reducing the effectiveness of the anterior circle of Willis anastomosis. The isolated PCATe infarcts in the present cases with ICA disease were by contrast precipitated by an acute impairment of the PCA itself.

Although Yates and Hutchinson\textsuperscript{3} reported a high incidence of VBA disease in their pathological study and occlusion or severe stenosis was found in the VBA in 13 (65%) of the present cases with 24 (52%) of the 46 infarcts, such VBA lesions appeared to contribute significantly to the development of but a few large cerebral hemisphere infarcts.

Acknowledgments

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