The Role of the Circle of Willis in Carotid Occlusion: Assessment with Phase Contrast MR Angiography and Transcranial Duplex*

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Purpose: To study the collateral pathways recruited after occlusion of the internal carotid artery (ICA), and to evaluate its influence on the impairment of hemispheric blood flow supply and development of low flow infarcts.

Methods: 38 patients with ICA occlusion (18 asymptomatic; five transient ischaemic attacks; and 15 strokes) were included. Infarcts on cerebral MR scanning were categorised in order to differentiate patients with territorial infarcts or no lesion (group I; n = 22) from those with brain damage due to low flow (group II; n = 16). Patency and direction of flow in the communicating arteries were assessed by means of cine phase contrast MR angiography (PC-MRA). Flow velocity in the middle cerebral artery (MCA) was measured by means of transcranial Duplex (TCD).

Results: Cine PC-MRA revealed a reversed ophthalmic artery blood flow ipsilateral to the ICA occlusion in all except two patients in group I and one patient in group II (N.S.). Posterior to anterior flow in the ipsilateral posterior communicating artery (PCoA) was detected in 16 (73%) patients in group I and in 13 (81%) in group II (N.S.). In contrast, reversed blood flow in the ipsilateral A1 segment of the anterior cerebral artery, through a patent anterior communicating artery (ACoA), was identified in 19 (86%) patients of group I, vs. 7 (44%) of group II (p = 0.005). The relative risk of low-flow infarcts was significantly higher in those cases with non-functioning ACoA (odds ratio = 8.1; p < 0.05). TCD showed a lower peak systolic velocity (PSV) in the ipsilateral MCA than in the contralateral one (60 ± 9 cm/s vs. 90 ± 11 cm/s; p < 0.005). Those patients without crossed flow through the ACoA, showed an even lower PSV in the ipsilateral MCA (55 ± 7 cm/s vs. 64 ± 9 cm/s; p = 0.03).

Conclusions: These data suggest that even though ICA occlusion may occur without cerebral damage, collateral blood supply is not enough to maintain normal hemispheric perfusion. The ACoA may be a key collateral pathway as a non-functioning ACoA is associated with an increased risk of developing low-flow infarcts.

Key Words: Internal carotid artery occlusion; Magnetic resonance imaging; Transcranial Doppler; Circle of Willis; Pattern of brain infarction.

Introduction

The circle of Willis acts as a safety system which maintains cerebral perfusion despite the occlusion of any of its major inflow vessels. Unilateral occlusion of the internal carotid artery (ICA) may result in inadequate intracranial blood flow supply due to either contralateral ICA occlusion or abnormalities of the circle of Willis. This may cause low-flow induced ischaemic infarcts. However, although a large number of patients have already suffered a stroke by the time ICA occlusion is identified, others present with minor symptoms and an appreciable proportion are symptom-free. What then are the differential patterns of flow in the circle of Willis that preclude cerebral damage in these patients?

The pathophysiological mechanism of the cerebral ischaemic lesions can be inferred from the location and morphological appearance on computed tomography (CT) or magnetic resonance imaging (MRI). A thrombotic or embolic occlusion of a large basal cerebral artery or its branches leads to an infarct that is strictly related to its territory. Such infarcts have been called "territorial". Territorial infarcts are most often cortical or large striatocapsular infarcts. 1 Low flow infarcts are considered to be the result of a critically reduced perfusion pressure secondary to prolonged systemic hypotension or ICA occlusion. Such infarcts may occur between neighbouring territories in the frontoparasagittal or temporo-parieto-occipital watershed areas. More commonly, these infarcts are found...
in the terminal supply areas of the deep perforator arteries in the supraganglionic periventricular white matter, strictly sparing the grey substance. This study was undertaken to determine the collateral pathways recruited after ICA occlusion occurs, and to evaluate its effect on hemispheric blood flow supply and the development of low-flow infarcts.

Patients and Methods

A group of patients with ICA occlusion were classified on the basis of MRI, into two groups: low flow infarcts and territorial or no infarcts. Haemodynamic patterns of the Circle of Willis were compared using cine phase contrast MRA and TCD blood flow measurement of the Middle Cerebral Artery (MCA).

Thirty-eight consecutive patients with ICA occlusion were included in the study. The diagnosis of ICA occlusion was initially made by Duplex examination, performed by an experienced technician with a 5 MHz linear array colour flow probe (ATL Ultramark 9 DP, Seattle WA, U.S.A.). Thirty-two (84%) patients were men and six (16%) women (mean age 57, range 18–74 years). Eighteen (47%) patients were asymptomatic and 20 (53%) presented with ipsilateral neurological symptoms (five transient ischaemic attacks and 15 stroke).

All the patients underwent MR-MRA imaging on a 1.5 Tesla magnet (Signa, General Electric, Milwaukee, U.S.A.). Brain images on spin-echo (SE) T1 and fast spin echo (FSE) T2 weighted on sagittal and axial planes were firstly obtained (SE 550/15 and FSE 4000/15–20, 256 × 192, FOV 22 cm, thickness 5 mm). The angiographic sequences were bi-dimensional phase contrast (2D PC), used as a localiser in coronal and sagittal planes over each carotid artery distribution (21°/23/6.5, flow velocity encoding (VENC): 35 cm/s, FOV 28–24 cm, 256 × 192). Three-dimensional time of flight (3D TOF) was performed in double volume on the circle of Willis and basilar artery (20°/45/6.9, FOV 22 cm, 512 × 256, saturation pulse (SAT) superior, 1 NEX). Bi-dimensional time of flight (2D TOF), with anterior neck surface coil, was used for carotid bifurcation (60°/45/8.7; 50 slices, thickness 1.5 mm, FOV 16 cm, 256 × 192, SAT superior, 2 NEX). On both acquisitions the maximum intensity projection (MIP) were used to assess morphology and abnormalities. Post-processing subvolumes were generated to isolate each carotid artery and create 20 MIP images at 10° increments. 3D TOF single slices were carefully read to assess the level and patency of the communicating arteries.

Flow direction in the communicating arteries were determined by means of cine PC-MRA. Direction of flow in the ophthalmic (OphA) and posterior communicating (PCoA) arteries was assessed using flow encoding in the anterior-posterior axis. Direction of flow in the anterior communicating (ACoA) and A1 segment of the anterior cerebral (ACA) arteries was determined by means of right to left flow encoding (20°/23/min, single slab thickness 20 mm, FOV 22 cm, 256 × 128, VENC 35–45 cm/s, 2 NEX).

The patterns of infarcts visible in T2-weighted cerebral views were categorised in order to differentiate patients with territorial or no lesion (group I, n = 22) from those with brain damage due to low-flow (group II, n = 16). Cortical and large striato-capsular (> 3 cm in diameter) in the ipsilateral MCA territory were typified as territorial infarcts. They were included in a single group together with those patients in whom no cerebral lesion could be identified. Patients were selected for inclusion in group II based on the following diagnostic criteria: watershed lesions; ischaemic areas in periventricular white matter; and large infarcts affecting two territories simultaneously.

Flow velocity in the middle cerebral artery (MCA) was measured by means of transcranial Duplex (TCD). A 2.25 MHz phased-array colour flow probe was used. Insonation of the MCA was performed through the temporal approach at a depth ranging from 4.5 to 5.5 cm. Once the colour signal was obtained and the artery correctly aligned, the Doppler tracings of both MCA were registered for further analysis.

Statistical analysis included comparison between both groups for the different patterns of flow in the ipsilateral OphA, PCoA and ACoA by the chi-square test. Comparison between groups for peak systolic velocity at the MCA were obtained using the Student's t-test or the non parametric Mann-Whitney U-test. Statistically significant difference was considered when p < 0.05 in bilateral comparisons.

Results

2D TOF acquisition technique confirmed the Duplex diagnosis of ICA occlusion in all but two patients (one had a critical ICA stenosis and the other < 60%). Both cases were excluded from the study. Bilateral ICA occlusion was identified in another patient. Cine PC-MRA in this patient revealed a posterior to anterior pattern of flow in both posterior communicating arteries. In the final analysis this patient was considered as a single case of non-functioning anterior
communicating artery, probably due to contralateral ICA occlusion.

Cerebral MR scanning (T2-weighted images) showed cortical infarcts, or large (> 3 cm in diameter) striatocapsular infarcts ipsilateral to the ICA occlusion in seven and three patients of group I, respectively. In another 12 patients, no cerebral ischaemic lesions were detected. Sixteen patients were selected for inclusion in group II. Frontoparasygittal or temporo-parieto-occipital watershed lesions were identified in five patients of this group (Fig. 1). Hyperintense areas in periventricular white matter were detected in eight cases. Another three patients had large infarcts affecting two territories simultaneously.

Collapsed MIP 2D projection images revealed hypoplasia or absence of one or both PCoA in eight (21%) patients; unilateral in five and bilateral in three. Unilateral absence of the A1 segment of the ACA was identified in three (8%) cases, all of them ipsilateral to the ICA occlusion. In another two (5%) patients unilateral hypoplasia of the initial P1 segment of the PCA was detected. There was no significant difference between either group regarding these abnormalities.

Cine PC-MRA showed reversed OphA blood flow ipsilateral to the ICA occlusion in all but two patients in group I and one in group II (chi-square, NS). Posterior to anterior flow in the ipsilateral PCoA was observed in 16 (73%) patients in group I and in 13 (81%) in group II (chi-square, NS). In contrast, only one patient in group I and four in group II showed this pattern of flow in the contralateral PCoA. Reversed blood flow in the ipsilateral A1 segment of the ACA, through a patent anterior communicating artery (ACoA), was identified in 19 (86%) patients in group I, as opposed to seven (44%) in group II (chi-square

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(1) OphA: reversed flow in the ophthalmic artery.
(2) PCoA: posterior to anterior flow in the posterior communicating artery.
(3) ACoA: crossed flow through the anterior communicating artery.
* Chi-square test.

p = 0.005 (Table 1; Figs. 2 and 3). Subsequently, the relative risk of low-flow infarct was significantly higher in those cases with non-functioning ACoA (odds ratio = 8.1; p < 0.05).

Duplex imaging of the MCA was achieved in all except five (13%) patients, mainly due to a poor transtemporal window. The TCD examination showed a lower PSV in the MCA ipsilateral to the ICA occlusion than in those fed by a patent ICA (60 ± 9 cm/s vs. 86 ± 11 cm/s; t-test p = 0.005). No significant difference was detected in the flow velocity of the MCA between groups I and II. Nevertheless, those patients without a crossed flow through the ACoA, showed an even lower PSV in the ipsilateral MCA when compared to controls with patent ACoA (56 ± 7 cm/s vs. 64 ± 9 cm/s; Mann-Whitney U-test p = 0.03) (Fig. 4).

**Discussion**

The old belief that the progression of a tight stenosis of the ICA towards total occlusion often occurs asymptptomatically, and that this protects against further neurological events has been completely discredited. The
prevalence of stroke in patients with ICA occlusion fluctuates between 43% and 52%. About 90% of these patients have ipsilateral infarcts on CT scan. Moreover, subsequent annual stroke rates are reported to be between 2% and 8%; similar to those of patients with TIAs. Twenty-six (68%) patients in this series had cerebral infarcts ipsilateral to the ICA occlusion.

The high incidence of low-flow infarcts ipsilateral to the ICA occlusion has been previously reported. Such haemodynamic infarcts have two well defined topographic patterns on MR scans. Low-flow infarcts in the cortical border zones (true watershed lesions) are rare. They have been found bilaterally in thromboangiitis obliterans and prolonged systemic hypotension, whereas a unilateral location is usually related to ICA occlusion. The more common subcortical type of low-flow infarct; the terminal supply-area or subcortical watershed infarcts, are located in the distal part of territories perfused by the deep perforators, in the corona radiata and centrum semiovale adjacent to the lateral ventricles. Two or more infarctions in one cerebral hemisphere also have been found to be highly associated with severe ICA stenosis or occlusion. A haemodynamic mechanism might be considered in this subgroup of patients, especially in those cases where there are simultaneous lesions in the MCA and PCA territories. In this study, frontoparasagittal or temporo-parieto-occipital watershed lesions were identified in five patients; hyperintense areas in periventricular white matter were detected in eight cases; and another three patients had large infarcts affecting two territories simultaneously.

The pathogenesis of these lesions has not been completely explained, but inadequate compensatory flow due to either contralateral ICA lesions or congenital abnormalities of the circle of Willis have been implicated. However, non invasive assessment of basal cerebral arteries has only become possible recently. The presence of adequate collateral pathways through the ophthalmic and anterior and posterior communicating arteries accounts for the ability of some patients to tolerate carotid occlusion without neurological deficit. Unfortunately, a complete "textbook" circle is present in only 21% of patients. Hypoplasia or absence of one or both posterior communicating arteries occurs in approximately 25-30% of individuals, leading to a greater or lesser degree of isolation of the carotid and vertebral circulations. Eight patients (21%) in this series had the aforementioned abnormalities of the PCoA; unilateral in five and bilateral in three. Collateral blood flow between the two cerebral hemispheres is mainly via the ACoA. Anomalies in this territory, including hypoplasia or absence of ACoA or the A1 segment of the ACA occur in about 26% of patients. In this situation, collateral inflow towards ACA and MCA...

Fig. 2. Phase contrast MR angiograms in a case of right internal carotid artery (ICA) occlusion. Flow is encoded right to left to assess patency of anterior communicating artery (ACoA). The appearance of both A1 segments of the anterior cerebral artery in dark demonstrates compensatory flow from the left ICA through the ACoA. (MCA = middle cerebral artery; PCA = posterior cerebral artery).
usually comes from reversed flow in the PCoA ipsilateral to the ICA occlusion. In this study, a non-functioning ACoA was detected in 12 cases (32%), including three patients (8%) with hypoplasia of the A1 segment. Nevertheless, three of these patients presented with contralateral ICA stenosis >90% including one case of bilateral occlusion. Consequently, critical stenosis of the contralateral ICA could partially explain this high prevalence of hemodynamic impairment of the ACoA. Hypoplasia of the initial (P1) segment of the PCA with maintenance of flow via an enlarged PCoA occurs in approximately

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**Fig. 3.** Phase contrast MR angiograms in a case of right internal carotid artery (ICA) occlusion and severe left ICA stenosis. (a) Right-left flow encoding. Opposite flow in the A1 segments of both anterior cerebral arteries (right in white, left in dark) demonstrates the absence of compensatory flow across the anterior communicating artery. (b) Anterior-posterior flow encoding in the same patient shows reversed flow in the ophthalmic artery (OphA) and posterior to anterior flow in both posterior communicating arteries (PCoA).
15–20% of cases. This abnormality was detected in only two (5%) patients of this series.

Both TOF and PC MRA techniques can be used to assess the flow dynamics within the circle of Willis. For the TOF techniques, flow direction can be determined by the careful positioning of presaturation bands over the carotid or basilar arteries during the acquisition of a 2D or 3D angiogram. Any blood flowing downstream of these presaturation bands will disappear from the MR angiogram. This method is quick and simple to perform and does not require electrocardiogram gating. Its accuracy for detecting intracranial circulation, as compared to angiography, has recently been studied by Furst et al. Sensitivities in detecting ACoA, PCoA and A1 segment of the ACA were 95%, 97% and 100% respectively. Nevertheless, the method showed a lower sensitivity (67%) in identifying transorbital flow.

PC flow imaging permits the generation of 2D or 3D angiograms using phase-difference processing and encoding for a threshold velocity. Flow-encoding gradients can be selected for any axis, generating phase maps that display flow in the following directions: anterior-posterior (AP), right-left (RL), or superior-inferior (SI). Flow in the direction of the flow-encoding gradient is represented in the image by bright (white) pixels and flow in the opposite direction is represented by dark pixels. The cine PC technique is a modification of 2D PC MRA in which the acquisition of the images is synchronised with the cardiac cycle by means of cardiac or peripheral gating. Cine PC provides images in which the vascular structures can be displayed with time resolution, overcoming the temporal averaging problems caused by pulsatility.

Recent development of new duplex transducers has allowed the direct imaging of the basal cerebral arteries. Transcranial Duplex (TCD) has overcome the initial drawbacks of "blind" Doppler methods. MRA and TCD share similar haemodynamic principles, providing dynamic and flow-encoded imaging. Nevertheless, MRA has partially solved the main pitfalls of TCD: operator dependence; inadequate examinations due to a poor transtemporal window and indirect assessment of communicating arteries which require compression manoeuvres.

In this study, cine PC MRA was used to assess flow direction in the intracranial pathways recruited after occlusion of the ICA. Reversed flow in the ipsilateral OphA was found in most of the patients of both groups. Posterior to anterior flow was the predominant pattern in the ipsilateral PCoA. However, no statistically significant difference between either group was detected regarding these collateral pathways. In contrast, crossed flow through a patent ACoA was significantly more frequent in the group of patients with territorial infarcts or no lesions (Group I) than in those with haemodynamic infarcts (Group II). Those patients with a non-functioning ACoA showed an increased relative risk of low-flow infarcts. Similar observations have recently been reported. Furst et al. using MRA techniques, found that low-flow infarcts occurred more frequently in patients with ICA occlusion and in those with posterior to anterior collateral flow rather than with collateral flow via ACoA. Ringelstein et al. evaluated the circle of Willis in 64 patients with ICA occlusion by means of TCD and compression manoeuvres. While only 11% of the cases with cross-filling via aACoA had low-flow infarcts, the proportion rose to 28% in patients with an exclusively posterior to anterior flow pattern and as this increased to 44% if compensatory flow relied on the OphA alone. These data suggest that the presence of compensatory crossed flow through a patent ACoA confers some protection against low-flow infarcts.

In this study, Doppler measurements of PSV in the MCA were used as an index of hemispheric perfusion. MRA methods for flow quantification (bolus tracking and phase-encoded techniques) have been successfully used to determine volume flow rates and flow velocity in the ICA. However they have some drawbacks when compared to Doppler measurements. Velocities measured by Doppler are the average velocity within the sample volume which may be as small as 1×1×3 mm, depending upon the transducer used. In MRI, the velocity is averaged over

Fig. 4. Peak systolic velocity in the middle cerebral artery (MCA) ipsilateral to: (A) patent internal carotid artery (ICA); (B) occluded ICA with crossed flow through the anterior communicating artery (ACoA); and (C) occluded ICA with non-functioning ACoA. * p < 0.005 (Student's t-test); † p = 0.03 (Mann-Whitney U-test).
the size of the voxel which is typically 1 × 1 × 5 mm. Moreover, the MRI measurement is averaged over a longer time period. In ultrasound methods, measurements are made about every 1 ms, while in MRI, measurements are made every 30 ms or more.22

Cerebral blood flow studies using inhaled 133-Xe, positron emission tomography, single photon emission computed tomography, and TCD in patients with occlusive carotid disease have demonstrated reduced cerebral blood flow, reduced perfusion reserve, increased oxygen extraction, and impaired vasomotor reactivity in the hemisphere ipsilateral to an ICA occlusion.23-27 In a recent study, cerebral vasomotor reactivity (CVMR) was determined by means of TCD measurement of blood flow velocity in the MCA during hypo- and hypercapnia, in a group of patients with ICA occlusion. CVMR was significantly higher in those cases of anterior cross-filling via the ACoA than with ICA occlusion. CVMR was significantly lower in the hemisphere ipsilateral to an ICA occlusion. Determination of blood flow direction using velocity-phase image display with 3D phase contrast MR angiography. AJNR 1992; 13: 1435-1438.

In conclusion, this study suggests that even though ICA occlusion may occur without cerebral damage, collateral blood supply is insufficient to normalise hemispheric perfusion. The ACoA may play a key role as a collateral pathway as a non-functioning ACoA is associated with an increased risk of developing low flow infarcts.

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