Relationship Between Pattern of Intracranial Artery Abnormalities on Transcranial Doppler and Oxfordshire Community Stroke Project Clinical Classification of Ischemic Stroke

G.E. Mead, MD; J.M. Wardlaw, MD; M.S. Dennis, MD; S.C. Lewis, PhD; C.P. Warlow, MD

Background and Purpose—The Oxfordshire Community Stroke Project (OCSP) devised a simple classification for acute stroke based on clinical features only, which is of value in predicting prognosis. We investigated whether the pattern of intracranial vascular abnormalities is related to the clinical syndrome.

Methods—Patients with acute ischemic stroke were classified by a stroke physician as having total or partial anterior circulation infarct (TACI or PACI, respectively), lacunar infarct (LACI), or posterior circulation infarct (POCI). Color-coded power transcranial Doppler was done whenever possible. Intracranial arterial velocities were compared in the 4 subtypes of ischemic stroke after adjustment for age and time to transcranial Doppler.

Results—Middle cerebral artery velocity was abnormal (hyperemia, reduced velocity, occlusion, or focal stenosis) in 38 of 69 TACIs (55%), 50 of 171 PACIs (29%), and 20 of 236 LACIs or POCIs (8%) (P < 0.001). Velocity in the A1 segment of the anterior cerebral artery was reversed in 12 of 69 TACIs (17%), 20 of 171 PACIs (12%), and 8 of 236 LACIs or POCIs (3%) (P < 0.001). Basilar artery velocity was abnormal in 8 of 121 POCIs (7%) compared with 5 of 355 (1%) of the other subtypes (P = 0.005). Vertebral artery velocity was abnormal (reduced velocity, occlusion, stenosis) in 20 of 121 POCIs (17%) compared with 20 of 355 others (6%) (P = 0.01).

Conclusions—Intracranial arterial abnormalities were related to OCSP clinical subtype. Therefore, it is possible to stratify patients according to OCSP classification in trials of new treatments in which treatment effectiveness may depend on the underlying pattern of arterial pathology and before any arterial imaging is available. (Stroke. 2000;31:714-719.)

Key Words: cerebrovascular disorders • cohort studies • stroke, acute • ultrasonography, Doppler

The Oxfordshire Community Stroke Project (OCSP) devised a simple and quick classification for acute stroke based on symptoms and signs only, which is of some value in predicting prognosis and risk of recurrent stroke.1 There is also a relationship between the classification and pathophysiology of ischemic stroke. When an infarct is seen on CT or MRI, its site and size are appropriate to the clinical syndrome in approximately 75% of patients.2–5 Carotid disease and cardiac sources of emboli are more common in total and partial anterior circulation infarcts (TACIs and PACIs, respectively) than in lacunar (LACIs) and posterior circulation infarcts (POCIs).6,7

While numerous studies have examined intracranial arterial velocities in middle cerebral artery (MCA) stroke,8–13 only 2 (of 32 and 85 patients, respectively) have investigated the relationship between the OCSP classification and patterns of vascular occlusion.14,15 Both demonstrated that those with TACIs were more likely than the other subtypes to have occlusion of the MCA, and those with LACIs were unlikely to have intracranial large-vessel disease.15 If these findings could be confirmed in a larger group of patients, this would support the use of the OCSP classification in stratifying patients in trials of any new treatment in which the underlying pattern of arterial disease may make a difference in the effectiveness of treatment, particularly if there is not enough time for complex investigations.

We hypothesized that distinct patterns of vascular abnormality would be found in the 4 OCSP subtypes: TACIs would involve more disease of the MCA or anterior cerebral artery (ACA) than the other subtypes, PACIs would involve more disease of the MCA or ACA than LACIs or POCIs but less than TACIs, and POCIs would involve more disease of the posterior circulation (basilar artery [BA], vertebral artery [VA], or posterior cerebral artery [PCA]) than the other 3 subtypes.

Subjects and Methods

A stroke physician identified consecutive patients with acute stroke admitted to our hospital or seen in neurovascular outpatient clinics

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From the Neurosciences Trials Unit, Department of Clinical Neurosciences, University of Edinburgh, Western General Hospital, Edinburgh, UK. Correspondence to Gillian E. Mead, MD, Neurosciences Trials Unit, Department of Clinical Neurosciences, University of Edinburgh, Western General Hospital, Edinburgh EH4 2XU, UK. E-mail GEM@skull.dcn.ed.ac.uk

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from November 1994 to April 1998 and classified them as having total anterior circulation syndrome, partial anterior circulation syndrome, lacunar syndrome, or posterior circulation syndrome (blind to all imaging data). A consultant neuroradiologist reviewed the brain CT or MRI. Intracerebral hemorrhages were excluded. Recent infarction on imaging in the appropriate hemisphere was classified as cortical (including border zone), PCA territory, lacunar, or large subcortical.

Whenever possible, a consultant neuroradiologist or radiographer (always blind to the clinical and CT or MRI data) performed color and color-coded power transcranial Doppler (TCD) ultrasound. The severity of carotid stenosis was determined by standard velocity criteria, and the lesion appearance was determined on an Acuson 128XP 10V machine with a 7-MHz multifrequency probe. The intracranial vessels were examined by the transtemporal and subcortical approaches with the same machine but a 2-MHz multifrequency probe and by the transorbital approach with a 7-MHz probe. The following arteries were examined and the velocities recorded: A1 and A2 segments of the ACA, anterior communicating artery (ACoA), MCA from its origin to the bifurcation, posterior communicating artery (PCoA), P1 and P2 segments of PCA, BA, and VA. The TCD operator made a diagnosis of normal, reduced velocity, hyperemic, occluded, or focal stenosis at the time of examination, and the data were then entered into our local stroke registry. Abnormal velocity was defined as a mean velocity either below or above the published limits of normality for the MCA, ACA, PCA, or BA with the patient’s age taken into account or an interhemispheric difference of >25% for the MCA. Published tables of normal values indicate a steady decline in mean velocities in all basal intracranial arteries with age. In the VA there is greater natural variation between sides, and therefore the velocity was considered abnormal if the arteries appeared to be of similar size but the velocity in one was greatly reduced or absent (ie, absent diastolic flow or peak systolic velocity less than half of the other side). If 1 VA was very small, it was considered likely that the artery was congenitally hypoplastic. Occlusion was diagnosed if there was a good bone window and the other basal arteries were visible (ie, failure to visualize the artery was not simply attributable to technical factors). Focal stenosis was defined as a localized area of increased velocity (above abnormal limits) associated with turbulence. Hyperemia was defined as diffuse velocity increase (above normal limits) along the length of the artery that could be insolated. We included data from subjects with poor bone windows in our analysis and analyzed data from the arteries ipsilateral to the brain lesion. A subject with a poor bone window was defined as a patient in whom either a very limited arc of the sector Doppler display or poor detail of the intracranial ultrasound landmarks was visible.

The presence or absence of any abnormality in a particular artery was considered the dependent variable, with age, time, and ischemic stroke subtype (TACI, PACI, LACI, or POCI) as the independent variable, in a logistic regression analysis. Probability values were calculated from the change in deviance when the variable relating to artery abnormality was added into the model. We adjusted for time between stroke onset and TCD for 2 reasons: (1) because spontaneous recanalization of intracranial vessels can occur over time and (2) because the time between stroke onset and TCD was different for the different OCSP syndromes (patients with severe strokes were admitted and investigated more rapidly than patients with less severe strokes, who were more often seen as outpatients and therefore seen later). We adjusted for age because it can influence the absolute velocity measurements. We also investigated the association between stroke subtype and clinical classification and abnormal MCA velocity patterns (P<0.001). For TACIs, the proportion with MCA abnormalities was similar for those having TCD at 0 to 1 day after stroke onset (12/24, 50%), 2 days (11/16, 69%), 3 days (9/15, 60%), and >3 days (6/14, 43%).

Flow direction in the ipsilateral A1 segment of the ACA was reversed in 12 of 69 TACIs (17%), 20 of 171 PACIs (12%), and 8 of 236 LACIs or POCIs (3%) (P<0.001 after adjustment for age and time between stroke onset and TCD). Although we did not hypothesize that there might be differences between the subtypes in the pattern of flow in the ACoA, it is interesting to note that flow in the ACoA was more commonly toward the side of the lesion in TACIs (12%) compared with 10% of PACIs, 4% of LACIs, and 1% of POCIs. This may reflect the association between reversed A1 flow and ACoA flow toward the side of the lesion: in the 25 patients in whom ACoA flow was toward the lesion (and the A1 was seen), A1 flow reversal was seen in 22 (88%), whereas all 11 patients with ACoA flow away from the side of the lesion had correct A1 flow (P<0.001, χ²).

Abnormal velocity in the basilar artery was seen in 8 of 121 POCIs (7%) compared with 5 of 355 (1%) of the others (P=0.002 after adjustment for age and time). Abnormal velocity in the ipsilateral vertebral artery (reduced, occluded, or stenosed) was seen in 20 POCIs (17%) compared with 20
of 355 others (6%) (P < 0.001 after adjustment for age and time). Abnormal velocity was seen in the P1 segment of the PCA in 1 PACI and 1 LACI.

There were 18 recent cortical infarcts (without a visible lacunar infarct) on CT or MRI in the patients clinically classified with LACIs. Therefore, the likely cause of the patients' stroke was the cortical infarct even though the patient was classified clinically with a LACI. Seven (39%) of these subjects had abnormalities in MCA velocity, a proportion similar to that for those with TACIs and PACIs.

We also investigated potential associations between cervical internal carotid artery (ICA) disease and patterns of intracranial artery abnormalities. Of the 102 patients with >70% ipsilateral ICA stenosis or occlusion, 56 (55%) had MCA abnormalities compared with 54 of 374 (14%) of those without ipsilateral ICA stenosis or occlusion (P < 0.001, χ²). Of the 43 patients with flow reversal in the A1 segment of the ACA, 22 (51%) had ipsilateral ICA occlusion and 11 (26%) had 70% to 99% ipsilateral ICA stenosis. Ipsilateral 70% ICA stenosis or occlusion was found in 25 of 69 TACIs (36%), 52 of 171 PACIs (30%), 15 of 115 LACIs (13%), and 10 of 121 POCIs (8%) (P < 0.001, χ², for comparison of PACIs and TACIs combined with LACIs and POCIs combined.)

Abnormal MCA velocity was also associated with abnormalities in the carotid siphon. All of the 5 patients (100%) who had

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**TABLE 1. Number of Patients in Whom TCD Was Attempted and in Whom a Good or Poor Bone Window Was Available**

<table>
<thead>
<tr>
<th></th>
<th>TACI</th>
<th>PACI</th>
<th>LACI</th>
<th>POCI</th>
<th>All</th>
<th>P*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total No.</td>
<td>118</td>
<td>528</td>
<td>322</td>
<td>253</td>
<td>1221</td>
<td></td>
</tr>
<tr>
<td>TCD attempted</td>
<td>87 (74%)</td>
<td>266 (50%)</td>
<td>150 (47%)</td>
<td>152 (60%)</td>
<td>655 (54%)</td>
<td></td>
</tr>
<tr>
<td>Good or poor bone window</td>
<td>69 (58%)</td>
<td>171 (32%)</td>
<td>115 (36%)</td>
<td>121 (48%)</td>
<td>476 (39%)</td>
<td></td>
</tr>
</tbody>
</table>

**TABLE 2. Characteristics of Patients Undergoing TCD Compared With Those in Whom TCD Was Not Attempted**

<table>
<thead>
<tr>
<th></th>
<th>TCD Attempted</th>
<th>TCD Not Attempted</th>
<th>Odds Ratio (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Survived &gt;14 days after stroke</td>
<td>637 (97%)</td>
<td>528 (93%)</td>
<td>2.5 (1.4, 4.5)</td>
</tr>
<tr>
<td>Inpatient</td>
<td>497 (78%)</td>
<td>206 (36%)</td>
<td>5.5 (4.3, 7.0)</td>
</tr>
<tr>
<td>Carotid Doppler done</td>
<td>649 (99%)</td>
<td>477 (84%)</td>
<td>20.2 (8.8, 47)</td>
</tr>
<tr>
<td>Carotid Doppler showed &gt;70% ipsilateral stenosis or occlusion</td>
<td>136/649 (21%)</td>
<td>41/477 (9%)</td>
<td>2.8 (1.9, 4.1)</td>
</tr>
</tbody>
</table>

**Survival and Inpatient Status**

- **Survived >14 days after stroke**: 637 (97%) TCD Attempted, 528 (93%) TCD Not Attempted, Odds Ratio (95% CI) 2.5 (1.4, 4.5)
- **Inpatient**: 497 (78%) TCD Attempted, 206 (36%) TCD Not Attempted, Odds Ratio (95% CI) 5.5 (4.3, 7.0)

**Carotid Doppler**

- **Carotid Doppler done**: 649 (99%) TCD Attempted, 477 (84%) TCD Not Attempted, Odds Ratio (95% CI) 20.2 (8.8, 47)
- **Carotid Doppler showed >70% ipsilateral stenosis or occlusion**: 136/649 (21%) TCD Attempted, 41/477 (9%) TCD Not Attempted, Odds Ratio (95% CI) 2.8 (1.9, 4.1)

**Time between stroke onset and clinical assessment, d**

- **Median**: 3 TCD Attempted, 11 TCD Not Attempted, P < 0.001, Wilcoxon 2-sample test
- **Interquartile range**: 1–9 TCD Attempted, 3–27 TCD Not Attempted
- **Minimum and maximum**: 0, 191 TCD Attempted, 0, 225 TCD Not Attempted

**Total**: 655 (100%) TCD Attempted, 566 (100%) TCD Not Attempted

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Values are number (percentage) unless indicated otherwise.

From χ² for sex and from Kruskal-Wallis test for age and time from stroke onset.
siphon stenosis also had abnormal MCA velocity (4 reduced velocity and 1 hyperemic), compared with 16 (15 reduced velocity and 1 occluded) of 21 (76%) with occluded siphons and 87 (21%) of the patients with no siphon disease. Most patients with an occluded siphon also had a proximal ICA occlusion. Only 1 of the 5 patients who had siphon stenosis also had an occluded ICA, compared with 20 of 21 (95%) with occluded siphons and 29 (6%) of the remaining patients.

Discussion
We found the anticipated relationships between the OCSP subtypes of acute ischemic stroke and patterns of intracranial vascular disease
First, MCA abnormalities were found in approximately 50% of the TACIs and 25% of the PACIs and were very uncommon in LACIs or POCIs. This is consistent with the type and severity of neurological symptoms and signs in the different subtypes and the site and extent of cerebral infarction on CT.4 The differences between the 4 subtypes were significant even after correction for the differences in the time between stroke onset and TCD, with TACIs being scanned earlier than the others. The most common MCA abnormality was reduced ipsilateral MCA velocity, which probably occurred either as a result of major branch or main stem occlusion or during the initial reopening phase of main stem MCA occlusion after embolus migration and hence distal vessel occlusion.20 The proportion of patients with MCA occlusion was similar to previous studies of MCA infarcts (in which between 33% and 67% had MCA occlusion), although, in these earlier studies, patients had TCD within a few hours of stroke onset.11–13 We found MCA stenosis in a small number of patients, which may be due to primary atherosclerotic stenosis or more likely a recanalizing embolic occlusion.8 Abnormally high MCA velocities were also seen, probably as a result of reactive postischemic hyperemia.20 We found associations between the presence of MCA abnormalities and disease of the ICA at the bifurcation or in the siphon, which would be

TABLE 3. Intracranial Flow Abnormalities in the 4 OCSP Subtypes of Ischemic Stroke

<table>
<thead>
<tr>
<th></th>
<th>TACI (n=69)</th>
<th>PACI (n=171)</th>
<th>LACI (n=115)</th>
<th>POCI (n=121)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A1 segment of ACA</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Reversed flow</td>
<td>12 (17%)</td>
<td>20 (12%)</td>
<td>4 (3%)</td>
<td>4 (3%)</td>
</tr>
<tr>
<td>Correct direction</td>
<td>34 (49%)</td>
<td>85 (50%)</td>
<td>73 (63%)</td>
<td>88 (73%)</td>
</tr>
<tr>
<td>A2 segment of ACA</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Visible</td>
<td>22 (32%)</td>
<td>85 (50%)</td>
<td>50 (43%)</td>
<td>55 (45%)</td>
</tr>
<tr>
<td>ACoA</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Flow toward lesion</td>
<td>8 (12%)</td>
<td>17 (10%)</td>
<td>5 (4%)</td>
<td>1 (1%)</td>
</tr>
<tr>
<td>Flow away from lesion</td>
<td>2 (3%)</td>
<td>3 (2%)</td>
<td>4 (3%)</td>
<td>2 (2%)</td>
</tr>
<tr>
<td>Balanced flow</td>
<td>7 (10%)</td>
<td>27 (16%)</td>
<td>25 (22%)</td>
<td>27 (22%)</td>
</tr>
<tr>
<td>MCA</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>26 (38%)</td>
<td>111 (65%)</td>
<td>95 (83%)</td>
<td>107 (88%)</td>
</tr>
<tr>
<td>Abnormal*</td>
<td>38 (55%)</td>
<td>50 (29%)</td>
<td>13 (11%)</td>
<td>7 (7%)</td>
</tr>
<tr>
<td>P1 segment of PCA</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Reversed flow</td>
<td>0</td>
<td>1 (1%)</td>
<td>1 (1%)</td>
<td>0</td>
</tr>
<tr>
<td>Correct direction</td>
<td>44 (64%)</td>
<td>123 (72%)</td>
<td>88 (77%)</td>
<td>99 (82%)</td>
</tr>
<tr>
<td>P2 segment of PCA</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Visible</td>
<td>46 (67%)</td>
<td>144 (84%)</td>
<td>102 (89%)</td>
<td>104 (86%)</td>
</tr>
<tr>
<td>PCoA</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anterior posterior flow</td>
<td>6 (9%)</td>
<td>21 (12%)</td>
<td>19 (17%)</td>
<td>27 (22%)</td>
</tr>
<tr>
<td>Posterior anterior flow</td>
<td>4 (6%)</td>
<td>15 (9%)</td>
<td>8 (7%)</td>
<td>1 (1%)</td>
</tr>
<tr>
<td>BA</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>29 (42%)</td>
<td>83 (49%)</td>
<td>58 (50%)</td>
<td>55 (45%)</td>
</tr>
<tr>
<td>Abnormal</td>
<td>1 (1%)</td>
<td>3 (2%)</td>
<td>1 (1%)</td>
<td>8 (7%)</td>
</tr>
<tr>
<td>VA</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>43 (62%)</td>
<td>110 (64%)</td>
<td>80 (70%)</td>
<td>67 (55%)</td>
</tr>
<tr>
<td>Abnormal</td>
<td>3 (4%)</td>
<td>12 (7%)</td>
<td>5 (5%)</td>
<td>20 (17%)</td>
</tr>
</tbody>
</table>

Data from the vessel ipsilateral to the site of the brain lesion are given. Two POCIs and 1 LACI with bilateral lesions are excluded. For each vessel, the numbers do not add up to the column totals because the number of vessels that were not seen are excluded.

*Abnormal includes reduced velocity, hyperemia, occlusion, or focal stenosis.
consistent with either an embolic source or propagation of thrombus from the carotid to the MCA or simply poor collateral flow to an otherwise patent MCA.

Second, we found that reversed flow in the A1 segment of ACA was more common in TACIs and PACIs than LACIs or POCIs. This may be due to the development of collateral flow but may simply be related to the presence of severe carotid stenosis, which was more common in TACIs and PACIs than LACIs or POCIs. We found that AC0A flow was more commonly toward the side of the lesion in TACIs than the other subtypes, but this may reflect the association with reversed AI flow, which was also more common in TACIs.

Third, more subjects with POCIs than the other subtypes had abnormalities in the BA or ipsilateral VA, but it is interesting to note that most subjects with POCIs had normal BAAs or VAs. This suggests that early spontaneous recanalization may have taken place, that the site of vascular occlusion may have been in the smaller branches of the BA, VA, or PCA, or that there was a proximal embolic source (e.g., heart or aortic arch).

Finally, very few subjects with LACIs had any detectable intracranial artery abnormalities. This supports the traditional view that LACIs are due to small-vessel disease. Interestingly, half of the subjects with LACIs with MCA abnormalities had cortical infarcts rather than LACIs on brain imaging, supporting a clinical classification usefully modified (but not replaced) by brain imaging.21

What are the limitations of our study? First, approximately 50% of the patients did not undergo TCD. We attempted TCD as often as possible but were constrained by availability of equipment and by time. Outpatients were less likely to be examined because of the long time lapse from stroke onset, which meant that the likelihood of finding an intracranial vascular occlusion was minimal. In outpatients, we were more interested in collateral pathways in patients with severe carotid stenosis or occlusion, (hence patients with stenosis or occlusion more often had TCD) than in intracranial acute occlusions. The second problem was that many patients had TCD a few days after the onset of the stroke (and therefore vessel recanalization may have occurred by the time of TCD), which may explain why approximately 50% of the TACI subjects and 75% of PACI subjects did not have any MCA abnormality. The OCSP clinical classification is based on the maximum neurological deficit since the stroke, not the deficit at the time of examination, since by then the patient may have improved. Hence time delay will not have influenced the clinical categorization of patients but may have affected the frequency of finding a vessel occlusion on TCD. However, we took time into account in our logistic regression model and still found significant differences between the 4 OCSP subtypes. Furthermore, there was no evidence that MCA abnormalities in TACIs were more common at days 0 to 1 compared with after day 3 from stroke onset.

What are the implications of this study? It is very plausible that some interventions may be beneficial in some types of ischemic stroke and not others and that the pattern of intracranial disease may be relevant to the effectiveness of stroke treatments or stroke prevention strategies. For example, thrombolysis may be more useful in patients with a large-vessel occlusion rather than intracranial small-vessel disease. In the acute phase of stroke, CT and MRI are often normal, angiography is too time-consuming and risky, and TCD is not always instantly available or available at all. CT or MR angiography may be possible alternatives but add to the duration and complexity of baseline imaging investigations. Given that there is a relationship between the OCSP classification and the patterns of vessel occlusion, it would be reasonable to stratify patients with the OCSP used as a rough guide to the site and probability of intracranial vessel occlusion. Further studies investigating the validity of the classification in predicting the site of vascular occlusion within a few hours of ischemic stroke onset would be useful. Of course, it may be that the clinical classification itself would then be less valid because of fluctuating clinical signs, but the predicted pattern of intracranial arterial abnormalities might provide useful, robust information to guide patient management.

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References
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