Regional Cerebral Blood Flow in Patients With Transient Ischemic Attacks Studied by Xenon-133 Inhalation and Emission Tomography

SISSEL VORSTRUP, M.D.,* RALF HEMMINGSSEN, M.D.,† LEIF HENRIKSEN, M.D.,*
HELLE LINDEWALD, M.D.,‡ HANS C. ENGELL, M.D.,§ AND NIELS A. LASSEN, M.D.¶

SUMMARY Cerebral blood flow CBF was studied in 14 patients with transient ischemic attacks TIA and arteriosclerotic neck vessel disease. CBF was measured by a rapidly rotating single photon emission computerized tomograph using Xenon-133 inhalation. This method yields images of 3 brain slices depicting CBF with a spatial resolution of 1.7 cm. Based primarily on the clinical evidence and on the angio graphical findings embolism was considered the pathogenetic factor in 10 cases, whereas chronic hemodynamic insufficiency rendered symptomatic by postural factors probably accounted for the symptoms in 4 patients.

Of the 14 patients, all studied days to weeks after the most recent TIA, four showed hypoperfused areas on the CBF-tomograms and with roughly the same location hypodense areas on CT-scanning, i.e. areas of complete infarction. However, an additional five patients showed reduction of CBF in areas with no abnormality on the CT-scan. The abnormal blood flow pattern was found to be unchanged after clinically successful reconstructive vascular surgery. This suggests the presence of irreversible ischemic tissue damage without gross emol lition (incomplete infarction).

It is concluded, that TIAs are often harmful events, as no less than 9 of the 14 patients studied had evidence of complete and/or incomplete infarction. Thorough examination and rational therapy should be instituted as soon as possible to prevent further ischemic lesions.

Stroke Vol 14, No 6, 1983

AS A TRANSIENT ISCHEMIC ATTACK TIA per definition leaves the patient without a neurological deficit, irreversible damage to brain tissue was formerly thought not to occur. However, neuropsychological studies of patients with TIA and arteriosclerotic neck vessel disease have revealed intellectual impairments in many of these patients, presenting either as lateralized deficits or as diffuse signs of cortical dysfunction.1,2 Despite the lack of psychometric tests prior to the first TIA these findings are thought to be the result of the repetitive ischemic episodes leading to deterioration of complex integrated cerebral function and eventually to "multiinfarct dementia," a term introduced primarily by Hachinski et al in 1974.3 Further, as prospective studies of CT-scannings in patients with TIAs have revealed infarcted areas in 35% of patients studied, it has become accepted that some TIAs are in fact minor strokes with definite areas of ischemic tissue necrosis, although the patients recover neurologically.4,5

Only few studies of CBF in patients with TIA have been undertaken, and in none of these were the findings compared to the results of CT-scanning.6,8 Single photon emission computerized tomography with inhalation of Xenon-133 has been shown to be a valid method for determination of alterations in CBF in stroke often revealing areas of low flow larger than the CT-scan defects, a discrepancy possibly reflecting partial neuronal loss (incomplete infarction).9,10 This method is safe, atraumatic and by its three-dimensional approach to CBF circumvents tissue layer superposition. Consequently we found it of interest to study a series of TIA patients with Xenon-133 inhalation tomography and CT-scan to elucidate the pathogenesis of the attacks and the state of intactness of the brain tissue.

Methods

Regional CBF was measured by the Xenon-133 inhalation method, using a rapidly rotating single photon emission tomograph* recently described in detail.11 The instrument has 64 sodium iodide crystals each measuring 12 cm in length, 1.3 cm in width, and 2.0 cm in depth. They are arranged in four banks that look at the brain from four sides. This geometric arrangement yields with the collimators used a high sensitivity of 18,000 counts per second/slice for a 20 cm wide circular phantom with an isotope concentration of 1 μCi Xenon-133/ml and with a 20% energy window.12 The lead collimators are focusing so that three slices of brain tissue are seen.

The CBF study lasts four minutes during which a series of four consecutive one-minute periods is recorded. Xenon-133 is rebreathed throughout the first 1½ minute from a closed system yielding a maximum lung concentration of 10 mCi/l. With the concentration used, the counting rate is about 400,000 counts in the second one minute period in which the counting rate is at its maximum. At this counting rate, the resolution in the plane is about 1.7 cm expressed as Full Width of a point source measured at its Half Maximum counting rate (FWHM). The air curve monitored over the right

---

*From the Departments of *Neurology, †Psychiatry, ‡Neuroradiology, and §Vascular Surgery, University Hospital, Copenhagen, Denmark, and ¶the Department of Clinical Physiology, Bispebjerg Hospital, Copenhagen, Denmark.

Address correspondence to: Sissel Vorstrup, M.D., Department of Neurology, University Hospital, 9, Blegdamsvej, DK-2100 Copenhagen, Denmark.

Received August 31, 1982; revision accepted May 10, 1983.
TABLE 1 Summary of Clinical Data and Findings on Angiogram, CT-Scan and CBF Tomogram

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Age</th>
<th>Sex</th>
<th>Days since most recent TIA before CBF-study</th>
<th>Symptoms (no of attacks in parenthesis)</th>
<th>Duration</th>
<th>Angiogram</th>
</tr>
</thead>
<tbody>
<tr>
<td>Embolic cases</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>NS</td>
<td>44</td>
<td>M</td>
<td>rt hemiparesis/esthesia and aphasia (3)</td>
<td>3h</td>
<td>ascl of lft ICA</td>
</tr>
<tr>
<td>2</td>
<td>ED</td>
<td>54</td>
<td>F</td>
<td>paresis/esthesia of rt arm (20)</td>
<td>12h or less</td>
<td>ascl in lft ICA</td>
</tr>
<tr>
<td>3</td>
<td>CN</td>
<td>57</td>
<td>M</td>
<td>rt AF (2), lft hemiparesis (1)</td>
<td>10m</td>
<td>ascl of both ICA ulcerative on rt, ascl of both CCA, lft VA</td>
</tr>
<tr>
<td>4</td>
<td>EN</td>
<td>66</td>
<td>F</td>
<td>rt AF (10) weakness of lft hand (3)</td>
<td>2-3m</td>
<td>ascl in both ICA's</td>
</tr>
<tr>
<td>5</td>
<td>ES</td>
<td>58</td>
<td>F</td>
<td>lft AF (&gt; 100)</td>
<td>10m or less</td>
<td>severe stenosis of lft ICA</td>
</tr>
<tr>
<td>6</td>
<td>GJ</td>
<td>63</td>
<td>F</td>
<td>lft AF (&gt; 50)</td>
<td>1-2m</td>
<td>severe stenosis of lft ICA and ascl of rt ICA</td>
</tr>
<tr>
<td>7</td>
<td>KH</td>
<td>50</td>
<td>M</td>
<td>rt AF (&gt; 100) weakness of lft hand and arm</td>
<td>1-2m 3h</td>
<td>rt ICA occluded</td>
</tr>
<tr>
<td>8</td>
<td>MA</td>
<td>61</td>
<td>M</td>
<td>lft AF (20)</td>
<td>1-2m</td>
<td>lft ICA occluded, ascl of rt ICA</td>
</tr>
<tr>
<td>9</td>
<td>BS</td>
<td>47</td>
<td>F</td>
<td>lft hemiparesis and aphasia (1)</td>
<td>1/2h</td>
<td>ascl lesion in rt ICA</td>
</tr>
<tr>
<td>10</td>
<td>IK</td>
<td>68</td>
<td>F</td>
<td>paresis/esthesia of either rt arm or lower limb (6)</td>
<td>2-3m</td>
<td>severe stenosis of lft ICA, ascl in rt CCA and ICA</td>
</tr>
<tr>
<td>Hemodynamic cases</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>EP</td>
<td>65</td>
<td>F</td>
<td>rt AF (&gt; 100) provoked by head tilt backwards</td>
<td>1m</td>
<td>severe stenosis of innominate with subclavian steal from both rt ICA and rt VA</td>
</tr>
<tr>
<td>12</td>
<td>AN</td>
<td>65</td>
<td>F</td>
<td>dizziness in upright position provoked by headturning to lft (&gt; 100) weakness of rt hand (1)</td>
<td>1/2m</td>
<td>occluded lft subclavian artery with steal, ascl in rt CCA &amp; ICA</td>
</tr>
<tr>
<td>13</td>
<td>AM</td>
<td>66</td>
<td>M</td>
<td>rt AF (1). lft arm paresthesia dizziness and blurred vision related to postural changes (many)</td>
<td>1/2m</td>
<td>severe stenosis of both ICA and lft VA</td>
</tr>
<tr>
<td>14</td>
<td>KS</td>
<td>66</td>
<td>M</td>
<td>rt hemiparesis/esthesia and aphasia, dizziness, and blurred vision by head turning to lft</td>
<td>10m</td>
<td>rt ICA occluded, severe stenosis of lft ICA</td>
</tr>
</tbody>
</table>

F = female; M = male; rt = right; lft = left; ascl = arteriosclerosis; AF = amaurosis fugax; AA = anonymous artery; CCA = common carotid artery; ICA = internal carotid artery; ACA = anterior cerebral artery; MCA = middle cerebral artery; PCA = posterior cerebral artery; VA = vertebral artery; Subc = subcortical; Occ = occipital; Par = parietal; m = minutes; h = hours.

The sequence of four one-minute tomograms is used for calculating CBF by a deconvolution procedure. The calculations assume a fixed value of lambda \( \lambda \) of 0.85 ml/g in all areas. This \( \lambda \) value corresponds for patients with normal hemoglobin concentration to \( \lambda \) for grey matter, i.e. the high flow areas of the brain used to scale the flow in absolute units of ml/100g/min. No correction was made for Compton scatter. The endexspiratory CO\(_2\)-concentration, PaCO\(_2\), was measured in the third or fourth minute of the washout period by an infrared capnograph.

Mean hemispheric blood flow values were calculated from the middle slice 2 (OM + 5 cm). For evaluation of inter- and intrahemispheric differences the mean CBF was calculated from slice 2 in a square area of variable size, ranging from 3.5 cm\(^2\) (1.9 \( \times \) 1.9) to 14.1 cm\(^2\) (3.8 \( \times \) 3.8) placed either anteriorly in the frontal region close to the midline, in the lateral Syl-
CT-scan was performed with an EMI 1010 Head scanner.

Patients

Fourteen patients, 6 men and 8 women, mean age 59 years, range 44 to 68 years, were included in this study after informed consent had been obtained. All the patients had a clear history of recurring TIAs referred to either the carotid or vertebrobasilar system with symptoms subsiding in less than 24 hours. They were all examined by a neurologist, and none revealed abnormal findings at the time of examination 2 to 90 days after the most recent attack. All patients had aortocervicography performed, except one who was studied with carotid angiography. Arteriography revealed arteriosclerotic lesions in all patients, ranging from discrete plaques to severely stenosing lesions, the latter defined as a reduction of lumen to 1.5 mm or less. Three patients had unilateral internal carotid artery occlusion, and one had an occluded subclavian artery.

CT-scan showed abnormal findings in 6 cases. Four patients showed hypodense lesions, three of these in addition showed central and cortical atrophy. A further 2 patients, age 61 and 65 years, showed central and/or cortical atrophy. The remaining 8 patients had normal findings.

Results

In normal man the tomographic flow map is symmetrical with a cortical flow level of 60–80 ml/100 g/min as evaluated from the Sylvian/insular regions, in which a high flow is always seen. The same flow level is found mesially in the frontal and occipital (striate) regions. Hemispheric mean flow in slice 2 averages 53 ± 6 ml/100 g/min (1 SD) and in slice 3 55 ± 7 ml/100 g/min (1 SD). The clinical results are shown in table 1 and 2. The hemispheric mean flow values in these patients with TIA did not differ significantly from the normal values.

Based on clinical story — in particular on an analysis of symptom provoking factors — and on the angiograms the patients were divided into embolic cases (10 patients) and hemodynamic cases (4 patients).

Embolic Cases Without Evidence of Irreversible Ischemic Lesions (5)

All 5 patients had lateralized symptoms arising from the carotid system: amaurosis fugax (AF) and/or hemiparesis/hemiparesis in some combined with aphasia. As seen from table 1, the patients — cases 1–5 had normal CT scans and also normal CBF tomograms. In all five the angiograms showed arteriosclerotic lesions in the internal carotid arteries ICA in one case also of the common carotid artery — on the side corresponding to the clinical symptoms in several cases also the contralateral carotid arteries — on the side corresponding to the clinical symptoms in several cases also the contralateral carotid arteries — on the side corresponding to the clinical symptoms in several cases also the contralateral carotid arteries.
### TABLE 2  Flow Values in Slice 2 and PaCO₂

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>Hemispheric flow</th>
<th>Regional CBF</th>
<th>Cerebellar mean flow PaCO₂</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean Left Right</td>
<td>Anterior Left Right</td>
<td>Median Left Right</td>
</tr>
<tr>
<td>1 NS</td>
<td>68 67 68</td>
<td>66 68</td>
<td>82 76</td>
</tr>
<tr>
<td>2 ED</td>
<td>62 62 61</td>
<td>63 65</td>
<td>65 70</td>
</tr>
<tr>
<td>3 CN</td>
<td>54 54 54</td>
<td>49 53</td>
<td>50 49</td>
</tr>
<tr>
<td>4 EN</td>
<td>60 60 59</td>
<td>58 60</td>
<td>64 66</td>
</tr>
<tr>
<td>5 ES</td>
<td>66 65 66</td>
<td>59 65</td>
<td>72 75</td>
</tr>
<tr>
<td>6 GI</td>
<td>79 77 80</td>
<td>72 80</td>
<td>79 92</td>
</tr>
<tr>
<td>7 KH</td>
<td>62 63 60</td>
<td>63 61</td>
<td>70 68</td>
</tr>
<tr>
<td>8 MA</td>
<td>70 69 71</td>
<td>60 69</td>
<td>72 74</td>
</tr>
<tr>
<td>9 BS</td>
<td>67 69 65</td>
<td>79 78</td>
<td>69 60</td>
</tr>
<tr>
<td>10 IK</td>
<td>53 52 54</td>
<td>50 53</td>
<td>57 62</td>
</tr>
<tr>
<td>11 EP</td>
<td>65 66 63</td>
<td>65 67</td>
<td>63 60</td>
</tr>
<tr>
<td>12 AN</td>
<td>56 56 55</td>
<td>62 66</td>
<td>70 72</td>
</tr>
<tr>
<td>13 AM</td>
<td>45 45 45</td>
<td>43 47</td>
<td>44 47</td>
</tr>
<tr>
<td>14 KS</td>
<td>41 41 41</td>
<td>37 40</td>
<td>41 41</td>
</tr>
</tbody>
</table>

Mean value ± 1 SD 60±10 60±10 61±10 60±12 5.5±0.7

Flow in ml/100 g/min and PaCO₂ as per cent (v/v).

en two days after the last attack demonstrates the adequacy of in-between-attack blood supply to the left carotid territory through the severe stenosis and through collateral channels as well.

Embolic Cases With Evidence Suggestive of Incomplete Infarction (3)

Of three patients (cases 6 to 8) in this group, all with lateralized symptoms from the carotid system two had normal findings on the CT-scan while case 8 showed a diffuse cortical atrophy. These patients studied 2 to 16 days after the most recent TIA showed focal reductions on the CBF tomogram persisting after reconstructive vascular surgery. Only in case 7 did the area of focal reduction of CBF correspond to the clinical symptoms. In the remaining 2 cases, however, the focal reductions were ipsilateral to the side from which the patients had suffered multiple AF.

**Case 7**

A 50-year-old man had suffered multiple right sided AF as well as two brief attacks of weakness of the left hand and arm. The attacks were not provoked by change of posture. Angiography showed first a narrow right ICA stenosis. But 8 weeks later reangiography revealed an occlusion of the right ICA. After this no more TIAs occurred. CBF in the right sided MCA-territory was 11% reduced posteriorly, right sided CBF 48 ml/100 g/min, left sided CBF 54 ml/100 g/min, see fig. 1a and 1b.

On the assumption that the asymmetric flow pattern was due to a low perfusion pressure, a superficial temporal-middle cerebral artery shunt was successfully performed and the patient remained without neurological symptoms. The asymmetrical flow pattern, however, persisted. In retrospect the fact that the patient became asymptomatic after total occlusion of the internal carotid artery speaks against a hemodynamic pathogenesis. Also the persisting asymmetric CBF following a technically successful by-pass points to the existence of chronic irreversible lesions in the right hemisphere, not manifest on the CT-scan (incomplete infarction).

Embolic Cases With Evidence of Complete Infarction (2)

Two patients (cases 9 and 10) had suffered symptoms from the carotid system and had evidence of infarct on CT-scan. The first patient had an infarct localized to the symptomatic hemisphere and the CBF tomogram showed low flow in the same region. The second patient had multiple bilateral small lacunar infarcts and the CBF tomogram showed only a moderate degree of asymmetry (lowest flow posterior in symptomatic hemisphere).

**Case 9**

A 47-year-old ambidext woman had three months previously suffered one single posturally unprovoked TIA in form of a half an hour lasting left sided hemiparesis with aphasia. Arteriography showed a non-stenosing arteriosclerotic lesion in the right internal carotid artery. CT-scan showed subcortical infarction in the right hemisphere. CBF tomography showed a larger area of low flow on right side in slice 2 and 3. In slice 2 right sided CBF was 60 ml/100 g/min, left sided 69 ml/100 g/min in the middle cerebral artery territory, see fig. 2a and 2b. This patient, we assume, has a relatively small ischemic infarct and probably areas of incomplete infarction as well on the basis of distal embolism causing her TIA.
FIGURE 7. TIA case 7: CBF tomography in slice 2 and 3 shows an asymmetrical flow pattern with reduced flow posteriorly in the right hemisphere.

Case 10

A 68-year-old woman treated for arterial hypertension during years had suffered 6 posturally unrelated attacks, each lasting 2–3 minutes, of paresis and paresthesia in either right arm or leg. Arteriography showed a severely stenosing left internal carotid artery and arteriosclerotic changes on right side. CT-scan showed symmetrical diffuse cortical atrophy and central atrophy, as well as several small "lacunar" infarcts bilaterally. CBF tomography showed a somewhat low flow level of 54 ml/100 g/min with distinctly low values in the watershed area posteriorly, most marked on the left side where, however, CT showed no lesion, see fig. 3a and 3b. This patient, we assume, had both small lacunar infarcts (due to in situ thrombosis of small perforating arteries with hypertensive wall changes?) and TIA due to repeated embolism from the arteriosclerotic lesion in the left internal carotid artery. The absence of orthostatic eliciting factors speaks against a hemodynamic pathogenesis.

Hemodynamic Cases Without Evidence of Irreversible Ischemic Lesions (2)

Two of the hemodynamic cases, cases 11 and 12, had normal CT-scans and normal CBF tomograms following successful vascular reconstruction.

Case 11 had a threadlike stenosis of the innominate artery with retrograde flow both in the right vertebral artery and in the right internal carotid artery. She suffered symptoms from the carotid system: Amaurosis fugax of the right eye always correlated to postural changes of her head (backward head-tilt). The CT-scan was normal. CBF tomography showed preoperatively a focal reduction corresponding to the angiographical lesions with flow being reduced posteriorly in the right hemisphere. Following shunt operation the tomographic flow maps were normal and the patient asymptomatic.

Case 12

A 65-year-old woman had throughout 5 years noticed dizziness provoked by turning her head backwards and to the left. The symptoms never occurred in relation to exercise of the left arm, which however was painful after some time of exercise. CT-scan showed a diffuse central and cortical atrophy, but no infarcts. CBF tomography showed a normal hemispheric flow except in the primary visual cortical areas where CBF was somewhat lower, almost as low as in the cerebellar area, where flow averaged 50 ml/100 g/min, see fig. 4a and 4b. Angiography showed an occluded left subclavian artery with retrograde flow in the otherwise normal vertebral artery. The right vertebral artery was without arteriosclerotic lesions. Moderately stenosing lesions were found in both internal carotid arteries. This patient, we assume, had a hemodynamically significant but fairly mild chronic flow impediment in the vertebro-basilar territory rendered symptomatic by postural changes compromising blood flow in the single functioning vertebral artery. Following shunt surgery CBF increased in the vertebrobasilar territory and the patient became symptomfree.

Hemodynamic Cases With Evidence of Complete Infarction (2)

These two patients (cases 14 and 15) had very severe stenosing or occluding lesions bilateral in the internal carotid arteries without important lesions of the vertebro-basilar systems. CT showed old infarcts. The CBF tomograms (fig. 5a and 5b) pointed to a symmetrical reduction of blood flow in the hemispheres with normal flow in the primary visual cortex and in the cerebellum. In both patients, the TIAs were elicited by orthostatic stress. The symptoms were remarkable as dizziness and/or blurred vision was noted by both patients, in one of them combined with hemiparesis. Lacking angiological evidence pointing to the v-b sys-
tem, it thus appears that the symptoms of dizziness and blurred vision customarily ascribed to the v-b system may arise from the carotid system.

Discussion

Patients with TIA present several difficult problems. First one has to distinguish the syndrome from other transient neurological and/or ocular manifestations. In our material the clearcut history given by the patients on repeated interviews, their age and multiple arteriosclerotic lesions in the neck arteries is considered adequate evidence for making this distinction. It should be noted that our material is quite selected: the patients were referred to us from other hospitals for reconstructive vascular surgery on the basis of the above mentioned evidence, not because of an acute TIA episode. This probably enhances the diagnostic accuracy relative to an unselected group of patients admitted because of an acute episode.

Next comes the problem of the pathogenesis of the TIA. In this context the angiogram is of some help. Arteriosclerotic lesions without hemodynamically significant stenoses point to an embolic genesis. But, the finding of narrowstenosis or even complete occlusion of one or more arteries does not tell that the TIA is hemodynamic. It depends on the adequacy of the collaterals. Having documented severely occluding arterial disease relevant to the symptoms we relied on clinical evidence of clearcut posturally provoking factors for diagnosing a given case as a hemodynamic TIA. Four patients satisfied this criterion. All four presumed hemodynamic cases had a reduced CBF in the appropriate part of the brain in the in-between-attack interval, i.e. more than 24 hours after the last attack — this being the timing of the CBF studies in all our patients.

The simple orthostatic stresses provoking the TIAs in some patients decreases the systemic pressure moderately. The intact cerebral circulation is quite resistant to reductions in the perfusion pressure. Hence it follows that in such TIA cases there must be some brain areas having a low perfusion pressure and a reduced flow already in the resting recumbent state, i.e. an abnormality of CBF that ought to be readily detectable. Our TIA patients with orthostatically provoked symptoms did indeed all show such CBF changes. But because areas of infarction (complete or incomplete) also show such changes, it will be necessary to use a stress test to disclose the hemodynamic insufficiency. Such a test based on observing the CBF response to variations of systemic blood pressure, of arterial pCO₂ or to certain drugs such as a acetazolamide (Diamox) or theophylline are currently being developed.

In the remaining 10 patients, the acute onset of

Figure 2a. + 2b. TIA case 9: CBF tomography in slice 2 shows a reduced flow in the right MCA-territory. CT scan shows an infarct in the right hemisphere.

Figure 3a. + 3b. TIA case 10: CBF tomography in slice 2 shows a low flow in the watershed area of the left hemisphere. CT scan shows several lacunar infaracts.
symptoms unrelated to postural changes combined with relevant arteriosclerotic lesions was taken as evidence of embolic pathogenesis. Five of these patients showed a focal reduction of CBF on the tomographic flow map in two associated with focal CT lesions. The three without such lesions are of particular interest.

These patients had either a severely stenosing or occluded internal carotid artery ipsilateral to the side from which they showed a focal reduction of CBF and had suffered multiple episodes of amaurosis fugax. The finding of a longstanding focal reduction of CBF in these patients with severe stenosing arterial lesions and with no CT lesion we at first interpreted as evidence of a clinically non-significant hemodynamic impediment. However, repeated CBF measurements following clinical successful reconstructive vascular surgery showed an unchanged flow pattern. Had a chronic hemodynamic factor been present, the arterial reconstruction would at least in some of the cases have been expected to abolish the flow asymmetry. Therefore the persistent low focal CBF in patients with embolic pathogenesis points to an irreversible ischemic tissue damage. Since the CT scan is negative no area of frank infarction has occurred. Hence a state of elective ischemic necrosis of the parenchyma without emolliation — incomplete infarction — was suspected, a state well described pathoanatomically, e.g. by Scholz.22

A brief account of the pathoanatomical changes will serve to correlate this pathology to the clinical picture in such patients. Ischemic lesions not resulting in infarction show injuries of the most vulnerable tissue component, the neuronal tissue, whereas the supportive tissue, the glial cells and vessels are spared. The histologic appearance of the end stage lesion ranges from a slight reduction of nerve cells not easily recognized to a total focal substitution of the neuronal tissue by astrocytic cells. The macroscopic findings corresponding to small incomplete infarcted areas are sparse and consist of focal atrophy, some degree of hardening of the brain tissue and discolouration of grey matter.22 These pathoanatomical changes may well correspond to a negative CT scan combined with a diminution of flow as measured by CBF tomography. We presume that after several months some of such patients may
develop a focal cortical atrophy recognizable on the CT scan reflecting the underlying selective parenchymatous necrosis (incomplete infarction).

It should be emphasized that patients with completed stroke with definite tissue necrosis may have areas of incomplete tissue infarction surrounding the hypodense lesion seen on the CT scan. In a previous study of CBF by computed emission tomography in 10 unselected stroke patients Lassen et al found a discrepancy in that the low flow areas on the CBF tomograms tended to be larger than the hypodense areas. A similar discrepancy was noted in several of the patients in the present series.

On basis of these findings, we conclude that TIAs will more often than hitherto suspected result in ischemic tissue damage, in form of incomplete or complete infarction. The frequent finding of mild dementia in such patients constitutes the clinical correlate. Many TIAs representing essentially a form of minor stroke, a continuum is formed by patients with TIA and dementia and the classical syndromes of multiinfarct dementia.

References
Regional cerebral blood flow in patients with transient ischemic attacks studied by Xenon-133 inhalation and emission tomography.
S Vorstrup, R Hemmingsen, L Henriksen, H Lindewald, H C Engell and N A Lassen

Stroke. 1983;14:903-910
doi: 10.1161/01.STR.14.6.903
Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1983 American Heart Association, Inc. All rights reserved.
Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/14/6/903