Computed Tomographic–Angiographic Findings Within the First Five Hours of Cerebral Infarction

S.H. Horowitz, MD; J.L. Zito, MD; R. Donnarumma, RN, MA; M. Patel, MD; and J. Alvir, DrPH

Background and Purpose: Modern management of acute stroke necessitates early diagnosis. To this end, we sought to delineate the radiographic features of focal hemispheric infarction within 5 hours of ictus.

Methods: Fifty patients, ages 54–79, with ischemic strokes productive of at least hemiparesis underwent computed tomographic scanning and cerebral angiography (n=38) or carotid ultrasound (n=12). Radiographic lesions were characterized for location, size, and pathophysiology.

Results: Acute abnormalities, hypodensity, and mass effect were seen in 56% of scans and confirmed on a second scan 5–7 days later. Intracranial angiographic abnormalities occurred in 61% of patients: arterial occlusions in 45% and delayed arterial filling in 16%. Hemorrhagic infarctions occurred in 26% of second scans and were associated with mass effect (100%) and arterial occlusions (89%). Infarcts with hemorrhagic transformation were larger on both scans than those without (p=0.001). Of four patients with infarctions in watershed territories on the scans, two had middle cerebral artery occlusions on angiography, thereby questioning the specificity of such scan lesions to low-flow states.

Conclusions: We conclude that cerebral infarctions are often visible on early scans, but their locations may not be etiologically determinative. The infarcts associated with intracranial arterial occlusions (45%) were of thromboembolic origin, but, given current controversies as to the pathophysiology of lacunar and watershed infarctions, we cannot ascertain the etiology in the remainder. These findings are relevant to the new stroke therapies that require administration in the first hours after infarction. (Stroke 1991;22:1245–1253)

Modern approaches to patients with cerebral infarction emphasize early diagnosis and management. Sophisticated computed tomographic (CT) scanners, angiography, and Doppler/ultrasound of the carotid and intracranial circulations increase the possibility of radiological confirmation of a suspected cerebral infarct within the first few hours after ictus and aid in its localization and pathophysiological characterization. However, delays in implementation limit the value of angiography and ultrasound due to frequent early recanalization of thrombi or clot lysis. In order to delineate early focal ischemic events and correlate the findings of current clinical, CT, and angiographic or carotid ultrasound techniques, we studied 50 patients within 5 hours of onset of infarction. The radiographic findings are reported here and the clinical–radiographic correlations will be reported (S. Horowitz, J. Zito, R. Donnarumma, M. Patel, J. Alvir, unpublished observations).

Subjects and Methods

Between November 1, 1987, and January 1, 1990, 50 consecutive patients with acute onset of focal cerebral dysfunction, who gave informed consent, presented with hemispheric infarction (right=20, left=30) of 3 hours or less duration, accurate timing of onset, and motor deficit (hemiparesis). The exclusion criteria consisted of inaccurate determination of onset (patients who awakened with deficits were excluded); previously known cerebral disease; non-ischemic etiology, that is, hemorrhage, tumor, or...
infection; other life-threatening illnesses; coma; clinical improvement within the first 5 hours, considered to be transient ischemic attacks; focal cerebral dysfunction without hemiparesis; and brainstem involvement. There were 33 males and 17 females, ages 54–79 (mean 67.9) years.

Noncontrast CT scans were performed on all patients using a GE 9800 scanner (Milwaukee, Wis.) with 10-mm cuts displayed on a 512x512 matrix (2-second scan time) from 45 minutes to 4 hours after onset of ictus, and were repeated at 5–7 days. The scans were visually assessed for areas of decreased attenuation (hypodensity); mass effect, as evidenced by sulcal effacement, ventricular compression, or contralateral shift of midline structures; hemorrhagic infarction, defined as small, multifocal hyperdensities within a larger hypodense area; localization of lesions to the vascular territories of the three major cerebral arteries and branches; obscuration of the lentiform nucleus; hyperdensity of the middle cerebral artery (MCA); and antecedent lesions indicative of previously silent infarcts. Lesion size was estimated as small (<1/2 lobe = 1), medium (1/2–1 lobe = 2), and large (>1 lobe = 3). Only lesions situated in areas anatomically compatible with the acute clinical deficits were considered. Infarcts were characterized as "territorial" if they occurred in a discrete intracranial arterial distribution and "watershed" if they occurred between two arterial territories. The angiographic results contributed to this differentiation.

Cerebral angiography was performed in 38 patients immediately after the initial scan and completed within 5 hours of ictus. The clinically appropriate extracranial carotid and intracranial vasculature was studied via transfemoral catheterization with a 15% solution of meglumine iothalamate (Conray R). In 12 patients, the extracranial arteries were assessed for atherosclerotic plaques and ulceration, kinking, intraluminal filling defects, turbulence of blood flow, and stenosis. Stenosis was graded on a scale of 0–4 with grade 1 as <30%; grade 2, 31–74%; grade 3, 75–99%; and grade 4, total occlusion. Grades 3 and 4 were considered hemodynamically significant.

The extracranial arteries were assessed for atherosclerotic plaques and ulceration, kinking, intraluminal filling defects, turbulence of blood flow, and stenosis. Stenosis was graded on a scale of 0–4 with grade 1 as <30%; grade 2, 31–74%; grade 3, 75–99%; and grade 4, total occlusion. Grades 3 and 4 were considered hemodynamically significant.

The extracranial arteries were designated according to the classification of Salamon and Huang and assessed for the presence of stenosis or intraluminal filling defects, occlusion, delayed flow, or collateral circulation. Oclusions or stenoses of the MCA were classified as follows: type I, proximal to the origin of the lenticulostriate branches; type II, distal to the lenticulostriate origins; type III, distal to the divisional bifurcation involving either the superior (type IIIa) or inferior (type IIIb) divisions; or type IV, individual branch involvement.

Intracranial arterial flow abnormalities were characterized as complete occlusion, occlusion with retrograde filling via a patent collateral circulation, and slow filling with or without arterial stenosis. Slow or delayed filling was in evidence when there was persistence of contrast material in an artery into the venous phase (>5.5 seconds after the onset of vascular opacification).

Carotid ultrasound was performed on the remaining 12 patients within 5 hours of ictus. A similar grading scale assessed carotid stenosis.

The data were analyzed with the chi-square test for differences in proportions and using the t test for differences in means.

We wish to note a methodological caveat in our data analysis. These patients participated in a randomized double-blind study of GM1 ganglioside (Fidia, SpA, Abano Terme, Italy) in patients with early infarcts. The clinical examination, first CT scan, and angiogram were performed before drug administration. We included the second scan for purposes of corroboration and further definition of the initial findings. In so doing, we made the assumption that neither GM1 nor placebo affected the second CT scan in a manner or to a degree that would compromise these purposes. We believe that the findings of this scan reflect the pathophysiological changes that underlie the radiographic features of an infarct more accurately than do the findings of the first scan, especially in the absence of new infarctions or anticoagulant therapy in any patient in the 5–7-day period between the initial ictus and the second scan. To exclude the second scan is unsatisfactory in that it precludes a discussion of hemorrhagic infarction and limits our ability to make accurate CT-angiographic correlations in this paper and clinical-radiographic correlations elsewhere (S. Horowitz, J. Zito, R. Donnarumma, M. Patel, J. Alvir, unpublished observations). At this time, the randomized code has not been broken and we are unaware of who received GM1 or placebo.

Results

Evidence of acute infarction occurred on the first CT scan in 28 patients (56%) and on the second scan in 37 patients (74%). In 11 patients, the first scan was normal and the second abnormal; in 11, both scans were normal; and in two, the first scan was abnormal and the second normal.

Table 1 shows the radiographic features, size, and location of lesions on both CT scans. Typical lacunar infarctions (circumscribed hypodensities in the basal ganglia–internal capsular area <1.5 cm in diameter) occurred in two patients. In another, a lacunar infarction was seen along with hypodensity and mass effect in the MCA cortical distribution. Infarct size was the same on both scans in 16 patients. In five patients, lesion size increased from small to medium; in two, from small to large; and in three, from medium to large. Of 11 patients with normal first scans and abnormal second scans, nine developed small lesions, one a medium lesion, and one a large lesion.

Mass effect was present on the first CT scan in 19 patients and on the second scan in 24 patients. On the first scan, mass effect occurred without hypodensity in four patients (8%); all had sulcal effacement (Figure 1). The lesions were small in two patients and
TABLE I. Summary of Computed Tomographic Scan Results in 50 Patients

<table>
<thead>
<tr>
<th>Radiographic features</th>
<th>First CT scan</th>
<th>Second CT scan</th>
</tr>
</thead>
<tbody>
<tr>
<td>No acute abnormalities</td>
<td>22 (44%)</td>
<td>13 (26%)</td>
</tr>
<tr>
<td>Hypodensity alone</td>
<td>7 (14%)</td>
<td>12 (24%)</td>
</tr>
<tr>
<td>Hypodensity with mass effect</td>
<td>15 (30%)</td>
<td>11 (22%)</td>
</tr>
<tr>
<td>Mass effect alone</td>
<td>4 (8%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>Hemorrhagic infarction with mass effect</td>
<td>0 (0%)</td>
<td>13 (26%)</td>
</tr>
<tr>
<td>Lentiform nucleus obscuration alone</td>
<td>2 (4%)</td>
<td>1 (2%)</td>
</tr>
<tr>
<td>Antecedent lesions</td>
<td>17 (34%)</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Lesion size</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Small</td>
<td>13 (26%)</td>
<td>14 (28%)</td>
</tr>
<tr>
<td>Medium</td>
<td>12 (24%)</td>
<td>14 (28%)</td>
</tr>
<tr>
<td>Large</td>
<td>3 (6%)</td>
<td>9 (18%)</td>
</tr>
<tr>
<td></td>
<td>28 (37%)</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Localization of infarcts by specific intracranial artery distribution</th>
<th>First CT scan</th>
<th>Second CT scan</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior cerebral artery</td>
<td>0 (1%)</td>
<td></td>
</tr>
<tr>
<td>Middle cerebral artery</td>
<td>22 (29%)</td>
<td></td>
</tr>
<tr>
<td>Cortical</td>
<td>15 (16%)</td>
<td></td>
</tr>
<tr>
<td>Mixed cortical−subcortical</td>
<td>6 (9%)</td>
<td></td>
</tr>
<tr>
<td>Subcortical</td>
<td>1 (4%)</td>
<td></td>
</tr>
<tr>
<td>Posterior cerebral artery</td>
<td>1 (1%)</td>
<td></td>
</tr>
<tr>
<td>Lacunar infarcts</td>
<td>2 (2%)</td>
<td></td>
</tr>
<tr>
<td>Watershed infarcts</td>
<td>3 (4%)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>28 (35%)</td>
<td></td>
</tr>
</tbody>
</table>

CT, computed tomographic.

*One in association with an anterior choroidal artery infarct.

The findings of the internal carotid artery (ICA) on angiogram and ultrasound are documented in Table 2. Of the 12 patients with angiographic hemodynamically significant ipsilateral ICA stenoses, six had complete MCA occlusions, three had MCA occlusions with partial retrograde filling, two (grade 3) had delayed flow, and one (grade 3) had normal intracranial flow. Tandem lesions of an ICA plaque and an intracranial arterial occlusion occurred in three patients with lesser stenoses (≤grade 2) on angiography. Common carotid or external carotid artery abnormalities were rare (<5%).

Nine of 28 (32%) patients with positive first CT scans and eight of 22 (36%) patients with negative first scans had grade 3 or 4 ICA stenosis. Thirteen of 37 patients (35%) with positive second scans and four of 13 patients (31%) with negative second scans had such ICA stenosis (p=NS).

Of the 38 patients undergoing angiography, 20 (53%) had abnormal first CT scans and 23 (61%) had abnormal intracranial angiograms (Table 3). There was a significant association between the presence of acute CT changes and the occurrence of occlusive or hemodynamic intracranial angiographic abnormalities (χ²=6.10, df=2, p=0.047) and between the size...
FIGURE 1. Initial (left panels) and repeat (right panels) computed tomographic scans of three patients (A, B, and C) with acute infarctions, demonstrating sulcal effacement (black arrows), ventricular compression (curved white arrow), and hypodensity (white arrows). Left hemisphere is on reader's right. Time from onset of ictus to initial scan: patient A, 2 hours and 15 minutes; patient B, 3 hours and 15 minutes; patient C, 1 hour and 30 minutes. In patient B, mass effect almost completely obliterated the Sylvian fissure on both scans (also seen on other sections). In patient C, a contralateral shift of the septum pellucidum is visible.

of the infarct and these same arterial abnormalities ($\chi^2=17.83$, df=6, $p=0.007$).

Acute changes on the second scan occurred in 26 patients (68%) and also correlated with the intracranial angiographic abnormalities ($\chi^2=7.29$, df=2, $p=0.027$). Of importance is the association of type I and type II angiographic findings with larger CT lesions ($\chi^2=28.15$, df=6, $p=0.0001$). In the 12 pa-
TABLE 2. Hemodynamic Findings of the Internal Carotid Artery on Angiogram and Ultrasound

<table>
<thead>
<tr>
<th>Degree of stenosis</th>
<th>Angiogram</th>
<th>Ultrasound</th>
<th>Totals</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ipsilateral to side of lesion</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>None or &lt; grade 2</td>
<td>26 (3)</td>
<td>7 (1)</td>
<td>33 (4)</td>
</tr>
<tr>
<td>Grade 3*</td>
<td>5† (3)</td>
<td>3 (2)</td>
<td>8 (5)</td>
</tr>
<tr>
<td>Grade 4*</td>
<td>7 (2)</td>
<td>2</td>
<td>9 (2)</td>
</tr>
<tr>
<td>Total</td>
<td>38 (8)</td>
<td>12 (3)</td>
<td>50 (11)</td>
</tr>
<tr>
<td>Contralateral to side of lesion</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>None or &lt; grade 2</td>
<td>9 (1)</td>
<td>11 (2)</td>
<td>20 (3)</td>
</tr>
<tr>
<td>Grade 3*</td>
<td>2† (1)</td>
<td>1</td>
<td>3 (1)</td>
</tr>
<tr>
<td>Grade 4*</td>
<td>1</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>12 (2)</td>
<td>12 (2)</td>
<td>24 (4)</td>
</tr>
</tbody>
</table>

Numbers in parentheses indicate arteries with atherosclerotic plaque formation. In the case of grade 4 occlusions, plaques were seen proximal to occlusions.

*Hemodynamically significant.
†One patient had bilateral grade 3 stenosis.

Patients with normal second CT scans, one had a type IV occlusion, three had slow-flow states, and the remaining eight (67%) had normal angiograms.

Intracranial occlusive disease was associated with a high frequency of CT lesions (Table 3). Thirteen of the 17 patients (76%) with MCA occlusions had abnormal first CT scans and 15 (88%) had abnormal second CT scans. The two patients with normal second scans had type IV occlusions. Patients with flow abnormalities as the sole angiographic finding were less likely to have acute CT changes (two of six on the first scan, three of six on the second scan). Fifteen patients (39%) had normal intracranial vasculature: in 10 the first CT scan was normal, two had lacunar infarctions, one had lentiform nucleus obscuration, and two had infarcts in the MCA distribution. Two patients with normal first scans developed MCA infarcts visible on the second scan, bringing to four the number of patients with normal angiograms and territorial infarcts on CT.

In four patients (8%), infarcts were localized exclusively to watershed or border zone areas, three on both CT scans and one only on the second scan. Three were cortical temporoparieto-occipital and one was subcortical. In one patient with a temporoparieto-occipital infarct, angiography revealed a type III MCA occlusion. The patient with a subcortical infarct had a type I MCA occlusion. In the two remaining patients, one had a grade IV ICA occlusion on ultrasound and one had a grade I ICA stenosis with type III MCA slow flow on angiography. Thus, only the latter patient, without evidence of intracranial occlusive disease, had a true watershed infarct. The patient with grade IV ICA occlusion on carotid ultrasound may have had a true watershed infarct, but the status of his intracranial vasculature is unknown. The other two patients had definite thromboembolic disease that mimicked watershed infarction on CT scan.

Hemorrhagic infarction was not visible on any first scan, but was present on second scans in 13 patients (26%), accompanied by mass effect (no confluent hematomas were seen). First-scan infarcts were evident in 12 patients (92%) who developed hemorrhagic infarction, as compared with 16 patients (43%) in whom hemorrhagic infarction did not evolve ($p=0.002$, t test). Visualized lesions were...
larger on both scans in patients demonstrating hemorrhagic infarction (first scan, $p=0.001$; second scan, $p=0.001$; $t$ tests). Of patients without visible infarcts on the first scan, only one of 22 (5%) developed hemorrhagic infarction, whereas five of 13 patients (38%) with small infarcts, four of 12 (33%) with medium infarcts, and three of three (100%) with large infarcts on the first scan developed hemorrhagic infarction subsequently. On the second scan, one of 14 patients (7%) with small infarcts, five of 14 (36%) with medium infarcts, and seven of nine (78%) with large infarcts exhibited hemorrhagic infarction. Nine patients with hemorrhagic infarction underwent angiography; eight (89%) demonstrated occlusive arterial lesions, as compared with nine of the remaining 29 patients (32%) without hemorrhagic infarction ($p=0.013$, $t$ test).

Hyperdensity of the MCA was seen on the first CT scan in seven patients and in one on the second scan as well. In three cases, it occurred on the affected side; in two, it was seen on the unaffected side on both scans; and in two, it was present bilaterally on both scans. In an eighth patient, bilateral hyperdense MCAs were seen only on the second scan. In four patients, calcification of the basilar artery was visible, implying that the hyperdense MCAs were due to atherosclerosis. Two patients (4%) had hyperdense MCAs only on the first scan, only on the appropriate side, and without basilar artery calcification, thereby suggesting a thrombus or embolus within the artery.

Angiography was performed on five of eight patients demonstrating hyperdense MCAs on CT scan. In the two with hyperdense MCAs on the affected side only on the first scan, there was a complete ICA occlusion (grade 4) in one and a type II MCA occlusion in the other. In one patient with a hyperdense MCA on the unaffected side and in two patients with bilateral hyperdense MCAs, the ICA and main MCA trunk on the affected side were normal: single MCA branch occlusions were seen. Thus, there was corroborative angiographic evidence of thromboembolic disease of the proximal MCA only in the two patients with hyperdense MCAs on the first scan.

Lentiform nucleus obscuration occurred in nine patients on the first CT scan, but was the sole abnormality in only two (4%). In one, it was the sole abnormality on the second CT scan. In the remaining seven patients, it was seen in association with hypodensity and mass effect in the MCA distribution.

Discussion

Despite the high incidence of stroke, the number of studies documenting its clinical and radiographic features within the first few hours are relatively scant. Some recent reports have noted slight hypodensities and mass effect on plain CT scans within 6 hours,² sesame report, but the general consensus is that ischemic changes are usually not evident on a scan until 24–48 hours after infarction.¹⁶ Only one study has reported angiographic–CT correlations, and only one² has compared the radiographic findings with the clinical picture within the first 6 hours.

Our results indicate that visible evidence of infarction on plain CT scans performed within the first 5 hours in patients with ischemic hemispheric strokes is frequent (56%). Mass effect and hypodensity, alone and together, are the major features seen in that time frame. As expected, these changes are not as well defined as those seen on the later scan, but they were clearly distinguishable from the normal and localizable (Figure 1).

Multiple experimental studies have documented the pathophysiology of focal cerebral ischemia. Within 1 hour after ischemia, a 3% increase in total water occurs in the ischemic area due to vascular dysautoregulation and development of intracellular (cytotoxic) edema.¹⁸,¹⁹ Extracellular (vasogenic) fluid accumulates, in part dependent on regional perfusion, presumably from collateral flow. Analysis of the biomechanical properties of ischemic tissue indicates the measurable presence of slight, but significant, edema 1 hour after occlusion.²⁰

The early CT scan evidence of cerebral infarction is dependent on this accumulation of edematous fluid. Increases in water content result in decreases in brain tissue specific gravity, and CT attenuation is linearly proportional to specific gravity.²¹ Changes of 2.5–2.6 Hounsfield units occur for every 1% change in water content.²² However, visualization of an infarct on CT is more than hypodensity associated with specific gravity changes. In four of our first CT scans (14% of positive first scans), mass effect is seen alone, and in other scans the hypodensities are almost always mild and contiguous with areas of mass effect in which hypodensity is absent. In these situations, sulcal effacement and ventricular compression reveal the presence of an infarct without, or in association with, mild hypodensity. Sulcal effacement and ventricular compression are caused by edematous changes that do not alter specific gravity enough to be visible as a hypodensity, but which are perceptible within several hours of ictus.

Tomura et al³ reported obscuration of the lentiform nucleus in 23 of 25 patients with embolic infarctions on CT scans performed within 6 hours of ictus. Lentiform nucleus obscuration occurred alone in eight (32%), in association with slight hypodensity in 11, and with slight hypodensity and sulcal effacement in four. Bozzao et al⁸ found abnormalities in 25 of 36 plain CT scans within 4 hours. Lentiform changes occurred alone in 14 (39%) and in association with cortical hypodensity in five. Cortical hypodensity or sulcal effacement without lentiform changes was seen in the other six.

Lentiform nucleus obscuration alone was seen in two patients (4%) on the first scan, in one of whom it also occurred alone on the second scan (suggesting that it was an artifact or not an acute change). This finding has been attributed to the early development of cytotoxic edema in the basal ganglia region, but the presence of gross mass effect in conjunction with
lentiform nucleus obscuration in seven of nine patients indicates that vasogenic edema is also present within the first few hours and both forms of edema proceed pari passu. Our results suggest that within the first few hours after infarction, lentiform changes do not occur without hypodensity or mass effect as often as previously reported.7,8

Pressman et al9 found increased density in the MCA on CT scans performed within 24 hours of ictus in 15 patients. Schuierer and Huk10 reported unilateral hyperdense MCAs in four patients within 6 hours. Tomsic et al11 reported its presence in seven of 25 patients (28%) undergoing plain CT scan within 90 minutes of ictus. No other findings were noted.

In our series, hyperdense MCA was not a valuable sign. While probably indicative of a thrombus or embolus within the artery,9-11 in the two patients (4%) with ipsilateral hyperdensities only on the first scan and ICA or type II MCA occlusions on angiography, in the other six patients it most likely represents atherosclerosis with vascular calcification.

Hemorrhagic infarction, not present on any first scan, was seen on the second scan in 26% of patients. In agreement with previous reports,23-27 infarcts with visible hemorrhagic components were significantly larger than those without hemorrhagic components. We, and others,25,27 found this size factor to be present on early scans even before hemorrhagic features were evident. However, the development of hemorrhagic infarction is not solely confined to larger lesions. Patients without infarcts (5%) and 38% with small infarcts on the initial scan did develop hemorrhagic infarction subsequently.

Arterial occlusions were seen in all but one (89%) of the patients with hemorrhagic infarction who underwent angiography. This high incidence of occlusive disease in hemorrhagic infarction is compatible with either the Fisher-Adams28 model of increased vascular permeability following arterial reperfusion after clot lysis or migration, or the concept of reperfusion to the ischemic area via leptomeningeal collateral circulation if occlusion of the feeding artery persists.29 As we did not perform serial angiography or transcranial Doppler sonography, we could not assess either of these possibilities.

The occurrence of mass effect on all CT scans demonstrating hemorrhagic infarction supports the findings of Lodder24 and Hornig et al25 that edema and mass effect are more prominent in hemorrhagic than in nonhemorrhagic infarction. The predilection for larger infarcts to develop hemorrhagic infarction with mass effect is also compatible with the theory that extensive edema causes small-vessel compression and blood-flow stasis. Subsequent decreases in edema may allow reperfusion of vessels, which by then have endothelial disruption, and diapedesis of blood follows.25

As many as 10% of all cerebral infarcts are claimed to be watershed lesions;30 the diagnosis is based on characteristic CT infarcts situated between the border zones of two or more arterial territories.6,12,21,32

Angiographic–CT correlations have associated CT lesions in watershed areas with severe carotid artery disease;12,30-33 and have implicated perfusion pressure hemodynamics in the production of focal cerebrovascular hypoperfusion of the distal most territories of branched arteries.31

In this study, four patients (8%) exhibited evidence of acute infarctions in watershed territories on CT scan. Angiography demonstrated occlusion of arteries whose territories supply the infarcted areas in two of these patients, one with a superficial temporoparieto-occipital cortical infarction (MCA type III) and one with a subcortical infarction (MCA type I). Angeloni et al34 described seven patients with distal pial MCA branch occlusions and a CT pattern of subcortical watershed infarctions, and Torvik30 reviewed cases of watershed infarcts caused by either cholesterol35 or tumor36 emboli. Taken in conjunction with these reports, our results suggest that infarctions in the watershed areas on CT are not necessarily specific or diagnostic of hemodynamic “low-flow” states and may occur in occlusive disease of thromboembolic etiology.

Our incidence of arterial occlusions is lower than the 10% ICA and 66% intracranial occlusions of Fieschi et al2 in 80 patients and the 83% incidence of arterial occlusive disease of Bozzao et al8 in 36 patients. Both reports studied acute ischemic stroke within 6 hours. While we concur with Bozzao and colleagues regarding an association between early acute changes on CT scan and intracranial arterial disease, our results differ in degree. Whereas they found intracranial arterial occlusions in all patients with early CT findings, we documented occlusions in 65% of such patients. Another 10% of our patients had delayed flow through the superior division of the MCA, and 25% had normal intracranial angiograms (Table 3). Further, we and Bozzao et al found intracranial arterial occlusions in 22% and 45%, respectively, of patients with normal early CT scans. We, in addition, found delayed flow in 22% of patients with normal early scans.

The presence of acute CT scan changes correlated with the degree of intracranial occlusive disease. With occlusions of two or more branch arteries (MCA types I–III), evidence of infarction was visible on 86% of first scans (Table 3) and on all second scans. Severity of occlusive disease was associated with lesion size on both scans. Intracranial flow abnormalities without intracranial arterial occlusions were less productive of acute CT changes (33% on the first scan, 50% on the second scan), and normal intracranial arteries were even less frequently associated with abnormal scans (25% on the first scan, 27% on the second scan). In addition, severe ICA stenosis (grade 3) or occlusion (grade 4) were not related to visible infarcts on either scan.

Analyses of cerebral infarctions through the 1980s focused on three types with, in large part, distinct and separable etiologies: lacunar infarcts in small-vessel disease (microatheroma or lipohyalinosis) secondary
to hypertension;37-40 watershed infarcts in low-flow states resulting from carotid disease;12,30-34 and territorial infarcts of thromboembolic origin.6,12 The CT correlates have been established, thereby allowing for radiographic discernment of each type of infarct. However, the results of this and other very recent studies may temper these concepts.

New and convincing reports raise doubts as to the select or even predominant role of hypertension in lacunar states;41,42 find that carotid disease with low flow43 and cerebral microembolic disease42 can also produce lacunes, and question the very existence of a definitive ischemic condition with the "lacune" appellation.42,44 Similarly, we and Angeloni et al34 find vascular occlusive disease on angiography in the presence of watershed infarcts on CT scanning, thereby disputing the exclusivity of low flow in the etiology of this condition.

Our finding that four patients had normal angiograms despite visible infarcts in the MCA distribution on CT scan, while not excluding thromboembolic territorial infarction, is of particular interest. A clot or an embolus could produce an infarct and break up before the angiogram, or microemboli could lodge in cerebral arteries too small to be visible on routine angiography, but normal angiography within 5 hours postictus raises the possibility of other mechanisms for these infarctions.

Consequently, although it would be convenient to subdivide our patients according to these three etiologies, attributing infarcts that were not clearly lacunar (4%) or watershed (4%) to thromboembolism (92%), we are convinced that thromboembolic processes exist only in the 45% of patients with intracranial occlusions. We are uncertain as to the infarct etiologies in the remaining patients. These concerns are relevant in view of recent advances in thrombolytic therapy and suggest that angiography or transcranial Doppler sonography is important in the workup of the early stroke patient.

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KEY WORDS • angiography • cerebral infarction • tomography, x-ray computed
Computed tomographic-angiographic findings within the first five hours of cerebral infarction.

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