Using computed tomography and magnetic resonance imaging, we prospectively studied 100 patients hospitalized with a lacunar infarct. Our aim was to evaluate the capabilities of magnetic resonance imaging in the detection and delineation of lacunes in a project of clinicotopographic correlations. Seventy-nine patients had a classic lacunar syndrome; 35 had pure motor stroke, 26 had ataxic hemiparesis, seven had sensorimotor stroke, and 11 had pure sensory stroke. A miscellaneous group of 21 patients had less typical lacunar syndromes, primarily with brainstem signs and symptoms. Among a total of 153 lacunes, magnetic resonance imaging detected at least one lacune appropriate to the symptoms in 89 patients. In 16 patients at least two lesions correlated with the clinical features, and precise clinicotopographic correlations were possible in 68 patients. Magnetic resonance imaging was more effective when it was performed a few days after the stroke. Lesions causing different types of lacunar syndromes had significantly different volumes, suggesting that the size of the lesion may influence clinical features. Magnetic resonance imaging may be the imaging technique of choice in the study of lacunar syndromes. (Stroke 1990;21:546–554)

Cerebral lacunes were described in the middle of the 19th century when the proposed mechanism was the resorption of small softenings. However, there was some confusion over the terminology between lacune and dilatation of perivascular spaces (état crible) until the works of Marie and others. Sixty years later, Fisher used detailed anatomic studies to describe the pathologic features of lacunes and particularly the small-vessel arteriopathies lipohyalinosis and microatherosclerosis underly-
ing lacunes. A later paper detailed the pathologic findings of infarcts that presented clinically as strokes. Fisher and his colleagues also correlated the clinical features of lacunes with the topography as pure motor stroke (PMS), ataxic hemiparesis (AH) and the closely related dysarthria-clumsy hand syndrome, pure sensory stroke (PSS), and sensorimotor stroke (SMS). The relation between lacunes and hypertension was also stressed. In a more recent review, Fisher discussed the possibility that other syndromes, most of them with brainstem tegmental signs and symptoms, may also be due to lacunar infarcts, though the underlying vascular pathology has not been well established.

Lacunes account for approximately 20% of all strokes, but clinicopathologic correlations have been few because of the low mortality associated with this type of stroke. Even with improvements in technology, the ability of x-ray computed tomography (CT) to identify lacunes accurately is limited. Magnetic resonance imaging (MRI) better delineates pathologic tissue, and has no bone artifacts even in the posterior fossa, and is of value in the assessment of lacunes. Our purpose was to study prospectively whether MRI correlated favorably with clinical syndromes thought to result from lacunar infarcts.

Subjects and Methods

Between June 1985 and October 1988, we studied the 120 consecutive patients admitted to our unit with a clinical diagnosis of lacunar stroke. We included in the classic lacunar syndrome group those with either proportional or partial PMS, PSS, AH (including the dysarthria-clumsy hand syndrome and the homolateral ataxia and crural paresis syndrome), and SMS. Patients with one of the brainstem syndromes described by Fisher were included in the miscellaneous group.

All patients had a CT scan without contrast, with 9-mm-thick slices in the orbitomeatal plane, to rule out a hematoma. A primary intracerebral hematoma was shown in six patients (5%, 95% confidence interval
Two patients had an AH, one an SMS, and the other three a PSS. Ten patients could not have an MRI study for the following reasons: obesity (in one with PMS), cardiac pacemaker (in two with PMS), intracranial metallic fragments (in one with PMS and one with AH), claustrophobia (in one with PMS, one with PSS, and two with lower basilar branch syndrome), and refusal (in one with PMS).

MRI was performed with a 0.5-T CGR Magniscan 5000 (78530 Buc, France) with a superconducting magnet. All patients had multislice spin-echo pulse sequences with contiguous T2-weighted images (resonance time 2,000 msec, echo time 60–120 msec) in the axial plane, from the medulla to the vertex. The sections were 6 or 9 mm thick. The MRI images were analyzed independently by a radiologist and a neurologist who were unaware of the patient's clinical signs and symptoms. The observers recorded the presence and topography of any MRI abnormalities in 10 areas of the brain according to a brain atlas. A lesion was considered to be present only if it was reported in the same area by both observers based on criteria established by both observers. A lacune defined as a hyperintense signal shown in the first and second echo images with sharp margins, <2.5 cm in diameter, and located in the deeper parts of the brain supplied by the perforating arteries. Infarction due to a large-artery occlusion (i.e., nonlacunar) was defined as a hyperintense signal of any size located in an area of the brain not supplied by the perforating arteries. Etat crible, the dilatation of perivascular spaces, was diagnosed in cases of multiple small, round, hyperintense signals, disseminated at the level immediately above the bifurcation of the internal carotid artery, isointense with the nonflowing cerebrospinal fluid. A hyperintense signal of unknown nature was reported if one of the observers could not allocate an MRI abnormality to one of the above categories or if the observers disagreed. The interobserver variation in the location or nature of the lesion was determined. The anatomic sites of lacunes were correlated with the clinical signs and symptoms. The volume of symptomatic lacunes was estimated according to the method proposed by Nelson et al. In four patients with normal CT scans, MRI demonstrated an infarct due to a large-artery occlusion (one with SMS due to a thalamopeduncular infarct, one with AH due to a medullocerebellar infarct attributed to angiographically verified vertebral artery thrombosis, one with AH due to a left-sided small corticosubcortical infarct, and one with PSS due to a right-sided corticosubcortical infarct). Therefore, 100 cases were included in the present report. There were 65 men and 35 women with a mean±SD age of 65±11.4 years. Ten patients had a history of stroke or transient ischemic attack. The clinical examination took place a mean of 1.8 days (Figure 1) and CT was performed a mean of 3.4 days after stroke onset. The clinical syndromes diagnosed were PMS in 35, AH in 26, SMS in seven, PSS in 11, and miscellaneous in 21 patients (Table 1). MRI was performed a mean of 17.8 days after the stroke. In 90% of the cases the images were of good quality; the 10% poor-quality images were attributed either to equipment malfunction (3%) or patient movement (7%).

We used the $\chi^2$ test, with Yates' correction, CI, and nonparametric Kruskall-Wallis analysis of variance. The level of statistical significance was defined as 5%.

### Results

There was good interobserver agreement in the anatomic location of the lesions ($\kappa=0.7$) and the nature of the abnormal signals ($\kappa=0.65$). One hundred fifty-three lacunes were observed (Table 2), with 89 patients (89%, 95% CI: 0.83–0.95) demonstrating a lacunar site compatible with the signs and symptoms (Table 3). In 16 patients, at least two lacunes or a lacune and a hyperintense signal of unknown nature could have explained the signs and symptoms (Figure 2). A lacune was more often 547
TABLE 2. Topography of 153 Lacunes in 100 Patients Examined With Magnetic Resonance Imaging

<table>
<thead>
<tr>
<th>Location</th>
<th>Right</th>
<th>Left</th>
<th>Bilateral</th>
<th>Total No.</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Corona radiata</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>8</td>
<td>5</td>
</tr>
<tr>
<td>Putamen</td>
<td>4</td>
<td>5</td>
<td>4</td>
<td>17</td>
<td>11</td>
</tr>
<tr>
<td>Pallidum</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Internal capsule</td>
<td>17</td>
<td>23</td>
<td>4</td>
<td>48</td>
<td>31</td>
</tr>
<tr>
<td>Caudate nucleus</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>4</td>
<td>3</td>
</tr>
<tr>
<td>Thalamus</td>
<td>12</td>
<td>6</td>
<td>2</td>
<td>22</td>
<td>14</td>
</tr>
<tr>
<td>Mesencephalon</td>
<td>5</td>
<td>1</td>
<td>0</td>
<td>6</td>
<td>4</td>
</tr>
<tr>
<td>Pons</td>
<td>14</td>
<td>16</td>
<td>4</td>
<td>38</td>
<td>25</td>
</tr>
<tr>
<td>Medulla</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Cerebellum</td>
<td>3</td>
<td>2</td>
<td>1</td>
<td>7</td>
<td>5</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>153</strong></td>
<td></td>
<td></td>
<td><strong>100</strong></td>
<td></td>
</tr>
</tbody>
</table>

shown in patients with classic lacunar syndromes (75 of 79, 95%) than in those with miscellaneous syndromes (14 of 21, 67%) ($\chi^2=10.8, p<0.001$; Table 3). Sometimes the responsible lacune was shown, but delineation of the abnormal hyperintense signal did not permit a precise clinicotopographic correlation (Figure 3).

To evaluate the time at which MRI is most likely to detect lacunes, we compared the 50 studies performed within the first 13 days after onset with the 50 performed later. MRI detected at least one lacune in a site compatible with the symptoms and signs in 49 (98%) of those studied early, while only 40 studies (80%) were positive in the 50 patients who had MRI later ($\chi^2=6.5, p<0.01$).

The mean±SD volume of the lacunes thought to be responsible for the clinical signs and symptoms was 0.81±0.92 ml, with a maximum of 4.4 ml. Volumes of the different types of lacunes differed significantly (SMS> PMS> AH> PSS> miscellaneous, Kruskal-Wallis analysis of variance, $p<0.0001$; Table 3).

Discussion

MRI will not be successful with claustrophobic patients or in patients with metallic fragments or cardiac pacemakers. Our failure to obtain MRI in 10 (8%) of our 120 patients is very similar to the rate reported generally. We were able to obtain a CT scan in all 10 patients.

An important practical question is how confident can the clinician be that patients with lacunar syndromes have small infarcts caused by occlusion of a single penetrating artery? That intracerebral hemorrhages may produce lacunar syndromes is well known, but the frequency of such hemorrhages is still a matter of debate. In six of our 120 patients (5%), the lacunar syndrome was due to an intracerebral hemorrhage, a figure similar to that reported in a community-based study. Sometimes a lacunar syndrome may be due to occlusion of a large artery, and in this situation anticoagulation might be more appropriate than in small-vessel disease. Nelson et al reported that 16% of patients with lacunar syndromes had large cortical infarcts, mostly in the right hemisphere. In four of our cases (3%), MRI demonstrated that the lacunar syndrome was due to occlusion of a large artery. MRI may therefore be of therapeutic consequence in discriminating occlusion of small perforating vessels from that of large arteries, especially in the brainstem, where the resolution of CT is lower.

The higher than usually reported proportion of AH in our study is probably due to our definition of this syndrome. In clinical practice, it is often difficult to demonstrate ataxia when paresis is present because there is insufficient motor power. The different rates of evolution of paresis and ataxia (with ataxia usually lasting longer than paresis) have been stressed. In our study, a patient with pure motor hemiparesis in whom

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TABLE 3. Magnetic Resonance Imaging Features of 100 Patients With Lacunar Stroke

<table>
<thead>
<tr>
<th>Clinical syndrome</th>
<th>Lacune shown consistent with signs and symptoms</th>
<th>Clinicotopographic correlations</th>
<th>Volume (ml)</th>
<th>Mean</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>One</td>
<td>At least two</td>
<td>None</td>
<td>Precise</td>
<td>Imprecise</td>
</tr>
<tr>
<td>Classic</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pure motor stroke</td>
<td>27</td>
<td>6</td>
<td>2</td>
<td>27</td>
<td>2</td>
</tr>
<tr>
<td>Ataxic hemiparesis</td>
<td>21</td>
<td>5</td>
<td>0</td>
<td>19</td>
<td>6</td>
</tr>
<tr>
<td>Sensorimotor stroke</td>
<td>7</td>
<td>0</td>
<td>0</td>
<td>7</td>
<td>0</td>
</tr>
<tr>
<td>Pure sensory stroke</td>
<td>6</td>
<td>3</td>
<td>2</td>
<td>6</td>
<td>4</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>12</td>
<td>2</td>
<td>7</td>
<td>9</td>
<td>4</td>
</tr>
<tr>
<td>Total</td>
<td>73</td>
<td>16</td>
<td>11</td>
<td>68</td>
<td>16</td>
</tr>
</tbody>
</table>
ipsilateral ataxia was observed later, when the paresis was resolving, was considered to have AH.

Our results could not have been validated from an autopsy study considering the natural history of lacunar infarcts, but only from the clinicoradiologic correlation method that we used. The number of lacunes per patient that we visualized with MRI (1.5) was lower than in autopsy studies, in which 1.3–4.4 (mean 3.4) lacunes per brain were counted. This result is not surprising because in autopsy studies many more strokes may have occurred before death. The partial volume effect caused by slice thickness and noise pat-
FIGURE 3. Top: Axial T2-weighted magnetic resonance image of upper pons in 79-year-old man with left unilateral internuclear ophthalmoplegia and right atactic hemiparesis showed hyperintense signal projecting onto left medial longitudinal fasciculus (arrow). Bottom: Axial T2-weighted image of same patient shows bilateral hyperintense signals projecting onto both pyramidal tracts and pontine nuclei (arrows).

terns may also have contributed to our lower number of lacunes per patient. The distribution of lacunes in the various anatomic areas was also different. In our study, lacunes were more frequent in the internal capsule and pons, of similar frequency in the thalamus, and less frequent in the lenticular nucleus than in autopsy studies. This result is probably explained by differences in the selection of cases. In our study, all patients
presented with a lacunar syndrome, with the most strategic brain areas affected. In autopsy studies the lacunes were sometimes difficult to correlate with the clinical history, and less symptomatic regions were affected; in patients with lacunar syndromes, routine CT scans were positive in 30–50% of the cases and
FIGURE 5. Top: Sagittal T2-weighted magnetic resonance image (first echo) of pontine lacune (arrows) of 64-year-old man with pure motor stroke and transient one-and-a-half syndrome. Study was performed 10 days after stroke. Bottom: Axial T2-weighted image (second echo) of same patient on same day. Lacune volume was 0.7 ml.
CT had to be repeated during the following weeks and months to reach a 70% positive rate.22,23,28 Our 89% positive rate with MRI corroborates previous clinicoradiologic studies on lacunes.27,44 We could not compare the sensitivity of CT and MRI because CT was performed sooner after the stroke than MRI, and the machine used in most cases was a second-generation CT. Using CT, lacunes have sometimes been described in the brainstem.28 With MRI, Rothrock et al27 reported a 29% rate of pontine lacunes. We detected a pontine lacune in 38 of 153 lesions (25%) and in 31 of 79 patients with classic lacunar syndromes (39%). In 16 of our cases, there were at least two lesions that might have explained the clinical history. With CT, brainstem lesions are usually not visible; therefore, these cases of tandem lesions raise some doubts about the accuracy of clinicoradiologic correlation studies based only on CT images.

MRI recognizes the ischemic or hemorrhagic nature of a previous stroke.45 Some months after a hemorrhagic stroke, the hemoglobin has been transformed to hemosiderin, which produces a hypointense signal on T2-weighted images.46 In two of our patients, a hemorrhagic scar was shown in the internal capsule and cerebellum (Figure 4). The association between lacunes and hemorrhages has been described in autopsy studies,5,6 but at frequencies greater than in our study (32% for Marie3 and 35% for Fisher4). The death rate due to intracerebral hematomas was 17% for Ferrand3 and 40% for Fisher.6 These high frequencies of fatal hemorrhages obviously contribute to the differences between the rate of hematomas in autopsy studies and in our MRI study. In Fisher's study,6 40% of the hematomas were small, suggesting that some small hematomas could have been present in our patients and not diagnosed by MRI. The sensitivity of MRI in this area remains to be established, but the prevalence of hemorrhagic scars seems to be lower than reported in autopsy studies. Rothrock et al27 reported a higher positive rate of MRI early after lacunar infarction, which is confirmed in our study. The shrinkage of the lacunes reported by Fisher6 and Donnan et al,23 and illustrated in our Figures 5 and 6, together with the decrease in the volume of the lacune may contribute to this result.

In 11 of our cases, the volume of the lacunes was >1.7 ml, which was the upper limit described in Fisher's autopsy study.6 DeWitt et al47 have shown an excellent correlation between MRI and autopsy. For acute bland infarction, reduction in the size of a lacune after months is also illustrated in one of our cases (Figures 5 and 6). This decrease in volume may be due to resorption of edema and necrosis; however, with MRI, wallerian degeneration and gliosis have a

FIGURE 6. Sagittal T2-weighted magnetic resonance image (second echo) of lacune shown in Figure 5 14 months later. Lacune volume was 0.36 ml.
prolonged T2 signal and may induce an overestimation of the volume. Thus, in our study most of the largest lacunes could have had volumes consistent with giant lacunes. The differences in the volumes of the lacunes and the clinical syndromes suggest that volume may influence the clinical signs and symptoms, as has been previously proposed.

In conclusion, MRI may be the imaging technique of choice in the clinical evaluation of lacunar infarcts.

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Key Words: cerebrovascular disorders, lacunar infarction, magnetic resonance imaging.
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