We describe an analysis of 227 patients with lacunar infarcts; 177 were inpatients and the remaining 50 were outpatients. The group comprised 11% of all inpatients with cerebrovascular pathology and 16% of all consecutive inpatients with brain infarcts studied at the Department of Neurology of the Hospital de la Santa Creu i Sant Pau. The main risk factors identified in these patients were arterial hypertension in 164 (72%), diabetes mellitus in 64 (28%), and heart disease in 58 (26%). The most common clinical syndromes were pure motor hemiparesis in 125 (55%), pure hemisensory stroke in 42 (18%), the sensorimotor deficit syndrome in 34 (15%), ataxic hemiparesis in seven (3%), and the dysarthria-clumsy hand syndrome in four (2%); atypical syndromes were observed in 15 patients (7%). Lacunes were demonstrated by computed tomography in 100 patients (44%) and by magnetic resonance imaging in 35 (78%) of the 45 patients in which it was applied. Magnetic resonance imaging was significantly better (p < 0.001) than computed tomography for imaging lacunes, especially those located in either the pons (p < 0.005) or the internal capsule (p < 0.001). After the acute phase, mild or no neurologic disability was detected in 178 patients (78.4%), moderate disability persisted in 48 patients (21.1%), and severe disability was recorded in one case (0.4%). Lacunar infarcts are a clearly defined entity with characteristic clinical features and an excellent short-term prognosis. Magnetic resonance imaging is the current method of choice for demonstrating these small brain lesions. (Stroke 1990:21:842-847)
because CT may overestimate the diameter of a lacune by as much as 100%. This would double in some instances the maximum diameter of 15 mm suggested by Fisher4 as the upper limit of a lacune on the basis of his anatomic observations.

The definitions of risk factors were similar to those applied in previous studies.30,31 History of arterial hypertension was determined from the patient's records and included previously recorded blood pressures of >160 mm Hg systolic or >95 mm Hg diastolic. History of diabetes mellitus (DM) was determined from appropriate records or a distinctly abnormal fasting blood sugar level and/or glucose tolerance test. Clinical evidence of angina pectoris, coronary insufficiency, myocardial infarction, congestive heart failure, or cardiac dysrhythmia were collectively included as heart disease. Transient ischemic attack (TIA) was defined as an episodic neurologic deficit of abrupt onset, consisting of brief subjective or objective focal neurologic dysfunction, with complete recovery in ≤24 hours.

We applied MRI to the last 45 patients entered into this series using a resistive magnet operating at 0.15 T. The tomographic plane was 10 mm thick. In each patient, only one CT and one MRI were obtained between 72 hours and 3 weeks after the onset symptoms.

The degree of clinical disability was evaluated according to the scale recommended by the Ad Hoc Committee,32 and the onset of the neurologic deficit in relation to the circadian rhythm was noted.

The data were evaluated according to arithmetic means, standard deviations, medians, and percentages; the nonparametric χ2 test (with Yates' correction as required); and parametric one-way analysis of variance.33,34

### Results

The mean±SD age of the patients was 60.5±9.4 (range 29–79) years. Most lacunes (94, 41%) became symptomatic when the patients were 56–65 years old, 60 (26%) when the patients were 66–75, 43 (19%) when the patients were 46–55, 18 (8%) when the patients were <46, and 12 (6%) when the patients were >75. One hundred fifty-four patients (68%) were <46, and 12 (6%) when the patients were 56–65 years old, 43 (19%) were 60–65, 74 (31%) were 76–85, 17 (7%) were 86–90, and 31 (13%) were 91–95 years. Most lacunes (94, 41%) became symptomatic when the patients were 56–65 years old, 43 (19%) were 60–65, 74 (31%) were 76–85, 17 (7%) were 86–90, and 31 (13%) were 91–95 years.

The clinical syndromes in this series were pure motor hemiparesis in 125 patients (55%), pure hemisensory stroke in 42 (18%), the sensorimotor deficit syndrome in 15 (7%), ataxic hemiparesis in seven (3%), and the dysarthria-clumsy hand syndrome in four (2%). The onset of symptoms was instantaneous (over minutes) in 76 patients (33.5%), acute (over days) in 115 (50.5%), and subacute (over days) in 36 (16%).

Among the 125 patients with pure motor hemiparesis, the deficit involved the face, arm, and leg in 94 (75%) and only one of these sites in 14 (11%) (Table 1). Four (67%) of the six patients with pure motor hemiparesis involving the face alone had CT abnormalities that were appropriate for the symptoms; three (75%) of these four patients had a positive MRI. The genu of the internal capsule (n=3) or in the pons (n=1) were the specific locations of these lacunes. None of the four patients with pure motor hemiparesis and deficit only in the arm had a positive CT scan, and none of the four patients with pure motor hemiparesis and deficit only in the leg had a positive CT scan. All 8 patients with pure brachial or pure crural motor hemiparesis had at least two CT scans. A lacune in the internal capsule was observed in one patient studied by MRI.

Among the 42 patients with pure hemisensory stroke, the deficit involved the face, arm, and leg in 31 (74%) and either the face and arm or the arm and leg in the remaining 11 (26%). The sensory deficit affected mostly superficial sensation and concomitantly the deep sensory modalities in 27 (64%).
sensory deficit was dissociated in 15 patients (36%), and approximately half complained of thalamic pain at the beginning (11 patients, 26%) or at the end (10 patients, 24%) of the acute ictal phase.

Among the 34 patients with the sensorimotor deficit syndrome, the motor deficit involved the face, arm, and leg in 32 (94%) and a combined superficial and deep sensory deficit was noted in 11 (32%). The degree of motor deficit in patients with the sensorimotor deficit syndrome was more severe than in patients with pure motor hemiparesis.

Ataxic hemiparesis was seen in seven patients, and 15 (7%) had atypical manifestations (hemiataxia in three, pure crural ataxia in one, pure motor hemiparesis with atypical aphasia in three, hemidystonia in two, the syndrome of hemichorea and hemiballismus in two, the hypothalamic syndrome of Guillain and Alajouanine35 in one, isolated dysarthria in one, the bilateral paramedian thalamic infarct syndrome in one, and the cerebelloloppyramidal syndrome of Marie and Foix36 in one).

Rare manifestations were seen among patients with classical lacunar infarcts. In seven patients (3%) a focal neurologic deficit was associated with a pseudobulbar syndrome, headache was recorded in 27 (12%), and dysarthria existed in 56 (24.5%). Among the patients with the sensorimotor deficit syndrome, 81% (13 of 16) who were dysarthric also had a severe motor deficit initially. In contrast, severe motor deficit existed in only 39% of the patients without dysarthria (seven of 18) (p<0.02).

Each patient had at least one CT scan of the head; a lesion corresponding to the neurologic deficit was demonstrated in 100 (44%). The mean diameter of a lacune was 15.9 (range 7-32) mm, and the mean volume was 1,778 (range 329-7,576) mm³; 62 patients (27%) showed concomitant ventricular dilatation and cortical atrophy. In 40 patients (18%) other vascular lesions (mostly old lacunes [in 26, 11%]) were visible. The percentages of patients with positive CT scans for each syndrome are shown in Table 2. Compared with other lacunar syndromes, the lowest positive correlation between CT abnormalities and clinical deficits was observed in patients with pure hemisensory stroke (p<0.003) and the highest in patients with the sensorimotor deficit syndrome (p<0.02). Among the patients with a positive CT scan, the mean lesion volume was 1,909 mm³ for those with pure motor hemiparesis, 1,931 mm³ for those with the sensorimotor deficit syndrome, and 984 mm³ for those with pure hemisensory stroke.

In 45 patients MRI revealed more lesions of a lacunar type (35, 78%) than did CT (16, 36%) (p<0.001); 20 patients (44%) with a positive MRI had a negative CT scan (Table 3). MRI reached the highest positivity in patients with pure motor hemiparesis (22, 84.5%) and was significantly superior to CT (10, 38.5%) (p<0.01). Among the remaining patients considered jointly, positive correlation as defined above was significantly higher with MRI than with CT (13, 68.5% versus 6, 31.5%) (p<0.05). Significantly more cerebral vascular lesions, especially old lacunes, were visualized better with MRI (18, 40%) than with CT (nine, 20%) (p<0.01). Among 12 patients with pontine lacunes visualized with MRI, only two lesions (17%) were demonstrable by CT (p<0.005). Of 17 patients with capsular lacunes demonstrated by MRI, only five (29%) had the same lesion visualized by CT; this difference is also favorable to MRI (p<0.001).

Locations of 120 lacunes by clinical syndrome are given in Table 4. By atypical lacunar syndrome, the lacune was located in the pons in all three patients with hemiataxia, in the thalamus in the one with pure crural ataxia, in the internal capsule and basal ganglia in both with pure motor hemiparesis with atypical aphasia, in the internal capsule and basal ganglia in one and in the thalamus in the other with hemidystonia, in the internal capsule and basal ganglia in one with the syndrome of hemichorea and hemiballismus, in the pons in the one with isolated dysarthria, in the thalamus in the bilateral paramedian thalamic infarct syndrome, and in the pons in the one

### Table 2. Patients With Positive Computed Tomograms by Clinical Lacunar Stroke Syndrome

<table>
<thead>
<tr>
<th>Clinical syndrome</th>
<th>Total (n=227)</th>
<th>PMH (n=125)</th>
<th>PHS (n=42)</th>
<th>SMS (n=34)</th>
<th>AH (n=7)</th>
<th>D-CH (n=4)</th>
<th>ALS (n=15)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Positive scan</td>
<td>100</td>
<td>44</td>
<td>55</td>
<td>44</td>
<td>7</td>
<td>17</td>
<td>22</td>
</tr>
</tbody>
</table>

% of N or n.

PMH, pure motor hemiparesis; PHS, pure hemisensory stroke; SMS, sensorimotor deficit syndrome; AH, ataxic hemiparesis; D-CH, dysarthria-clumsy hand syndrome; ALS, atypical lacunar syndromes.

### Table 3. Comparison of CT and MRI in 45 Patients With Lacunar Stroke

<table>
<thead>
<tr>
<th>Imaging modality</th>
<th>CT</th>
<th>MRI No.</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>+</td>
<td>...</td>
<td>16</td>
</tr>
<tr>
<td></td>
<td>+</td>
<td>...</td>
<td>35</td>
</tr>
<tr>
<td></td>
<td>+</td>
<td>+</td>
<td>15</td>
</tr>
<tr>
<td></td>
<td>+</td>
<td>-</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>-</td>
<td>+</td>
<td>20</td>
</tr>
<tr>
<td></td>
<td>-</td>
<td>-</td>
<td>9</td>
</tr>
</tbody>
</table>

CT, computed tomography; MRI, magnetic resonance imaging.
TABLE 4. Location of Lesion in 120 Patients With Lacunar Syndrome

<table>
<thead>
<tr>
<th>Location</th>
<th>No.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pure Motor Hemiparesis</td>
<td></td>
</tr>
<tr>
<td>Internal capsule</td>
<td>52</td>
</tr>
<tr>
<td>Internal capsule and basal ganglia</td>
<td>6</td>
</tr>
<tr>
<td>Pons</td>
<td>9</td>
</tr>
<tr>
<td>Pure Hemisensory Stroke</td>
<td></td>
</tr>
<tr>
<td>Thalamus</td>
<td>8</td>
</tr>
<tr>
<td>Ataxic Hemiparesis</td>
<td></td>
</tr>
<tr>
<td>Internal capsule</td>
<td>5</td>
</tr>
<tr>
<td>Pons</td>
<td>2</td>
</tr>
<tr>
<td>Dysarthria-clumsy hand syndrome</td>
<td></td>
</tr>
<tr>
<td>Pons</td>
<td>3</td>
</tr>
<tr>
<td>Sensorimotor deficit syndrome</td>
<td></td>
</tr>
<tr>
<td>Internal capsule</td>
<td>16</td>
</tr>
<tr>
<td>Internal capsule and basal ganglia</td>
<td>4</td>
</tr>
<tr>
<td>Pons</td>
<td>2</td>
</tr>
<tr>
<td>Thalamus</td>
<td>1</td>
</tr>
<tr>
<td>Atypical lacunar syndrome</td>
<td></td>
</tr>
<tr>
<td>Internal capsule and basal ganglia</td>
<td>4</td>
</tr>
<tr>
<td>Pons</td>
<td>5</td>
</tr>
<tr>
<td>Thalamus</td>
<td>3</td>
</tr>
</tbody>
</table>

Location as revealed by computed tomography and/or magnetic resonance imaging.

with the cerebellopyramidal syndrome of Marie and Foix.

Among 181 patients in whom the onset of symptoms could be established with respect to the circadian rhythm, 59 (33%) occurred between 12:01 and 6 AM, 44 (24%) occurred between 6:01 AM and noon, 57 (31%) occurred between 12:01 and 6 PM, and 21 (12%) occurred between 6:01 PM and midnight. The preponderance of onset during waking hours (122, 67%) may be attributable to physiologic variations in blood pressure.

Medical complications during the acute phase included urinary infections in 14 patients (6%), respiratory infections in five (2%), and cardiac symptoms in eight (3.5%). Among the latter, congestive heart failure and/or supraventricular arrhythmias were the most common.

Mean follow-up of the 227 patients was 11 (range 1–60) months. Eighty-four patients have been followed until now (Table 5). No disability was observed in 19 (23%) of the 84 patients at the end of the acute phase of the disease, in 40 (48%) after 3 months, in 44 (52%) after 6 months, and in 53 (63%) after 12 months. These 84 patients may not be representative of the entire group of 227 patients.

Twenty-three (10%) of the 227 patients have been readmitted to the hospital, 15 (6.5%) because of recurrent cerebrovascular pathology, six (2.5%) for treatment of cardiac disease, and two (1%) because of associated neoplastic disease.

Discussion

In four other comparable series, lacunes comprised 11%–19% of all patients with vascular pathology of the brain. During our study 1,607 inpatients with cerebrovascular diseases were evaluated in the Department of Neurology; 1,084 had ischemic infarcts and 523 had brain hemorrhages. The 177 inpatients with lacunar infarcts represent 11% of all inpatients and 16% of those with brain infarcts.

Arterial hypertension in 72% and DM in 28% of our patients were the main risk factors; these figures agree with those from the Harvard Cooperative Stroke Registry and those collected through the Pilot Stroke Data Bank. In Fisher's autopsy study, evidence of hypertension defined by either clinical or morphologic criteria was present in 97% of the cases.

Pure motor hemiparesis was the most common syndrome among our patients with lacunes (55%). Mohr recorded pure motor hemiparesis in 60% of his lacune cases; two other studies reported 60% and 32%. Pure hemisensory stroke was observed in 18% of our patients and was the second most common syndrome. Fisher considers pure hemisensory stroke to be the most frequent lacunar syndrome, but his cohort was not homogeneous as it included patients with clinical symptoms suggestive of TIAs. Because some lacunes manifest as TIAs, we included only patients whose focal neurologic deficit lasted at least 24 hours.

The most commonly involved sites in our patients with pure motor hemiparesis were the face, arm, and leg, simultaneously; isolated motor deficits at any one of these three locations were seen in 11% of our patients. Monoplegia, in Fisher's opinion, is never associated with the occlusion of a perforating arteriole. However, Ferrand, Bennett and Campbell, Dujerine and Dujerine-Klumpke, and Donnan et al have all described lacunar infarcts associated with monoplegia.

TABLE 5. Follow-up of 84 Patients With Lacunar Stroke

<table>
<thead>
<tr>
<th>Disability</th>
<th>Acute phase</th>
<th>3 months</th>
<th>6 months</th>
<th>12 months</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
<td>No.</td>
<td>%</td>
</tr>
<tr>
<td>None</td>
<td>19</td>
<td>23</td>
<td>40</td>
<td>48</td>
</tr>
<tr>
<td>Minimal</td>
<td>42</td>
<td>50</td>
<td>36</td>
<td>43</td>
</tr>
<tr>
<td>Moderate</td>
<td>22</td>
<td>26</td>
<td>7</td>
<td>8</td>
</tr>
<tr>
<td>Severe</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
</tbody>
</table>
Thalamic pain, one of the most incapacitating clinical expressions of pure hemisensory stroke, was recorded in approximately half of our patients afflicted with this syndrome. The percentage with thalamic pain among our patients is higher than among Fisher’s patients,19 but his cohort included patients with clinical symptoms suggestive of TIAs, and in our series all patients with pure hemisensory stroke had neurologic deficits that lasted >24 hours.

Motor deficits were more severe among our patients with the sensorimotor deficit syndrome than among those with pure motor hemiparesis, in accordance with the findings of our patients, a figure lower than the 38% reported by others.22 The existence of associated vascular lesions in patients with symptomatic lacunes is not surprising as several clinicopathologic studies have shown that most lacunes are multiple and that many are asymptomatic.46,47

MRI is the imaging method of choice to diagnose lacunes; it confirmed the topographic location of the lesions in 78% of the cases analyzed. MRI was superior to CT in documenting lacunar infarcts for both the total group examined (p<0.001) and for the subgroup with pure motor hemiparesis (p<0.01). MRI has been applied to patients with lacunes in a few studies, all of which confirm its superior definition of the lesion compared with CT.20–24

Among our patients with atypical lacunar syndromes, only three had no demonstrable lesion by either CT or MRI. We excluded hemorrhage and cortical/subcortical infarction as the cause of the symptoms in these patients and assumed that the symptoms were related to small lesions. However, this hypothesis needs anatomic or imaging confirmation.

The low percentage of medical complications (12%) and the absence of deaths in our patients have been previously noted by others.1–5,48,49

In our series and in at least one other,13 the prognosis for patients with the sensorimotor deficit syndrome is worse than for those with pure motor hemiparesis. In contrast, all patients with the dysarthria-clumsy hand syndrome had favorable evolution and almost complete recovery. These two syndromes seemingly constitute the extremes in the broad spectrum of lacunes.

Twelve months after the onset of symptoms, 63% of our patients and 53.5% of Fisher’s patients29 had recovered almost entirely. After long-term follow-up, only 20% of the patients of Richter et al20 with lacunes had either moderate or severe neurologic deficit.

The existence of specific neurologic syndromes as a reflection of brain lesions known as lacunes has been questioned recently.51 However, our observations in 227 patients support the concept that well-defined neurologic syndromes associated with small, deep brain infarcts (i.e., lacunes) constitute a clearly defined entity with characteristic clinical features and an excellent short-term prognosis.

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KEY WORDS • lacunar infarction • magnetic resonance imaging • risk factors
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A Arboix, J L Martí-Vilalta and J H García

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