The Lausanne Stroke Registry: Analysis of 1,000 Consecutive Patients With First Stroke

Julien Bogousslavsky, MD, Guy Van Melle, PhD, and Franco Regli, MD

For the Lausanne Stroke Registry Group

We present epidemiologic, etiologic, and clinical data for 1,000 consecutive patients with a first stroke (cerebral infarction or hemorrhage) admitted to the Centre Hospitalier Universitaire Vaudois since 1982. The patients were evaluated using a standard protocol of tests (computed tomography, Doppler ultrasonography, and electrocardiography in all patients, as well as angiography and specific cardiac investigations in selected patients). Each case was coded prospectively into a computerized registry. We believe that the Lausanne Stroke Registry is the first registry with complete computed tomography and Doppler ultrasonography data on all patients, which allows correlation between clinical findings, presumed etiology, and stroke location. Although the Lausanne Stroke Registry is not population-based, it gives a good estimate of the stroke-related problems in patients admitted to a primary-care center since our hospital is the sole acute-care facility for stroke in the Lausanne area. (Stroke 1988; 19:1083-1092)
cemia (fasting blood glucose concentration of >5.6 mmol/l) and hypercholesterolemia (cholesterol concentration of >6.5 mmol/l) the day after admission; venous hematocrit on admission; history of migraine, ischemic heart disease, arrhythmia, or vascular claudication; and family history (stroke and heart disease).

**Clinical findings.** Heart and neck auscultation, neurologic features (type of stroke onset, triggering factors, neurologic picture, headaches, level of consciousness).

**Arterial disease.** Evidence of extracranial and intracranial arterial disease, if available (Doppler ultrasonography, angiography findings).

**Cardiac investigations.** Results of ECG, echocardiography, Holter monitoring.

**Functional disability.** None, mild, moderate, or severe at discharge; cause of death.

**Type of stroke.** Ischemic versus hemorrhagic, territory, presumed cause.

Other parameters (coagulation and hematologic disorders, cerebrospinal fluid [CSF] abnormalities) were not coded but were recorded if abnormal and were analyzed using the $\chi^2$ test and Fisher’s test.

**Cerebral Infarction**

This diagnosis was made when CT showed a hypodense area corresponding to the clinical picture or when no lesion was seen on CT (with ECG confirmation of the localization of the lesion in patients with superficial territory infarcts). In supratentorial infarcts, the vascular territory involved was assessed using Damasio’s template mapping.10

Infarcts in the superficial territory of the middle cerebral artery (MCA) were classified as anterior or posterior according to the location of the main part of the infarct with reference to the central sulcus. Watershed infarcts were diagnosed according to reported guidelines.11 Vertebrobasilar infarcts were classified into six groups according to the locations involved: brainstem (medulla, pons, midbrain), cerebellum, superficial, deep (thalamus), or entire posterior cerebral artery (PCA) territory, and multiple locations. We defined the presumed cause of infarction according to the following criteria:

**Atherosclerosis with stenosis.** Narrowing of >50% of the lumen diameter (four groups: 50–74%, 75–89%, 90–99%, or occlusion) of the corresponding extracranial artery or large intracranial artery (MCA, PCA, or basilar artery [BA]), in the absence of another etiology.

**Atherosclerosis without stenosis.** Plaques or <50% stenosis in the MCA, PCA, or BA, in the absence of another etiology and in patients with at least two of the following five risk factors: age ≥50 years, hypertension, diabetes mellitus, cigarette smoking, or hypercholesterolemia.

**Emboligenic heart disease.** Intracardiac thrombus or tumor, rheumatic mitral stenosis, prosthetic aortic or mitral valves, endocarditis, atrial fibrillation, sick sinus syndrome, left ventricular aneurysm or akinesthesia after myocardial infarct, acute (<3 months) myocardial infarct, or global cardiac hypokinesia or dyskinesia, in the absence of another etiology.

**Hypertensive arteriopathy.** Infarction in the territory of a deep perforating artery in a patient with known hypertension, in the absence of another etiology.

**Mixed etiologies.** Combinations of the above four etiologies.

**Other etiologies.** Arterial dissection, fibromuscular dysplasia, saccular aneurysm, arteriovenous malformation, cerebral venous thrombosis on angiography, angiitis (multiple segmental arterial narrowing on angiography, pleocytosis of CSF), hematologic conditions (polycythemia, thrombocytopenia, etc.), migraine (history of migraine, occurrence of stroke during an attack of migraine), or other. **Undetermined etiology.** None of the above causes of cerebral infarction could be determined.

**Cerebral Hemorrhage**

This diagnosis was based on CT findings, and five subdivisions were used to indicate the vascular territory involved: lenticular-capsular, lobar (frontal, temporal, parieto-occipital), cerebellar, thalamic, and brainstem. Intraventricular bleeding was also recorded.

We classified the presumed cause of cerebral hemorrhage into six groups: hypertensive arteriopathy in patients with known hypertension and without anticoagulant therapy, anticoagulant therapy in patients receiving heparin sodium or warfarin, sacciform aneurysm documented by angiography, arteriovenous malformation documented by angiography or CT, other etiologies (brain tumor documented by CT or biopsy, amyloidosis documented by biopsy, or other), or undetermined etiology.

**Results**

There were 615 men (mean ± SD age 61.1±12.2, range 20–89 years) and 385 women (mean ± SD age 60.3±15.6, range 16–91 years). Figures 1 and 2 show that the age distribution differed by sex for patients with infarction ($\chi^2=33.3, p<0.001$) and hemorrhage ($\chi^2=9.8, p<0.05$). Males predominated in all age groups for stroke in general except for infarction in patients <30 and >80 years old. An infarct was present in 891 patients and a hemorrhage in 109 (on the left in 54.9%, on the right in 37.4%, and bilateral in 7.7% of those with hemorrhage).

**Cerebral Infarction**

The infarct was seen on CT in 82.2% (732) of the 891 patients with infarction, with a hemorrhagic component in 3% (27). This high percentage was due to the fact that CT was repeated systematically in patients without visualization of the infarct on first CT. The carotid territory was involved in 68% of the 891 patients with infarcts, the vertebrobasilar territory in 26%, a watershed area in 3%, and
multiple territories in 3%. Ninety-six percent of the carotid territory infarcts involved the MCA territory (entire territory in 13%, deep in 32%, anterior superficial in 31%, posterior superficial in 20%); 3% involved the anterior cerebral artery (ACA) territory, and 1% involved the entire internal carotid artery (ICA) territory. Forty-eight percent of the vertebrobasilar infarcts were in the brainstem (mainly pons in 27%, mainly medulla in 14%, mainly midbrain in 7%), 7% in the cerebellum, 36% in the PCA territory (entire territory in 7%, deep in 11%, superficial in 18%), and 9% in multiple locations.

Table 1 correlates etiology with the location of infarction in the brain. Atherosclerosis with stenosis was the main cause of entire MCA territory infarcts (38%), superficial MCA territory infarcts (35%), and watershed infarcts (75%). Atherosclerosis, with or without stenosis, was also the presumed cause of most ACA territory and vertebrobasilar territory infarcts. The highest prevalence of embolic heart disease was found in global or superficial supratentorial infarcts and in cerebellar infarcts, but at no time was embolic heart disease found to be the most frequent cause of any type of infarction, including multiple infarcts. The combined etiology of atherosclerosis with stenosis and embolic heart disease was uncommon (3% overall), but the finding of this combined etiology was probably biased by the fact that asymptomatic heart disease was not systematically searched for. Hypertensive arteriolopathy was the most common presumed cause of deep MCA infarcts, but it should be emphasized that 32% of deep MCA infarcts were associated with an appropriate large-artery stenosis and/or embolicogenic heart disease (without hypertension in half the cases), suggesting that artery-to-artery and cardiac embolism was not a negligible cause of infarction in the territory of deep perforating arteries. Arterial dissection was found in many patients with entire MCA (15%) or ICA (25%) infarcts. Atherosclerosis without stenosis was found in 18% of the cases overall, and an undetermined etiology was found in 8% overall. Other etiologies were found in 6% of the cases overall (Table 2).

Cerebral Hemorrhage

The location of the 109 hemorrhages was most often lenticular-capsular (42%), followed in order of frequency by lobar (40%) (frontal in 19%, occipitoparietal in 8%, temporal in 13%), cerebellar (8%), brainstem (6%), and thalamic (4%). Partial intraventricular bleeding was present in 13% of the patients with hemorrhage, including 24% of the lenticular-capsular hemorrhages.

Table 3 correlates etiology with site of hemorrhage in the brain. Hypertensive arteriolopathy was the major cause of lenticular-capsular, cerebellar, brainstem, and thalamic hemorrhages, but lobar hemorrhages had diverse etiologies. Three of the six patients on anticoagulant therapy (warfarin) at the time of hemorrhage had a prothrombin time of <15% of the mean in a control group; two of these six patients had hypertension. Rupture of an aneurysm or an angioma was found only in lobar hemorrhage. Other etiologies of hemorrhage were histologically proven amyloidosis, intratumoral bleeding (with metastatic carcinoma), severe alcoholic liver disease with coagulation disturbances, autopsy-
TABLE 1. Etiology Correlated With Topography of Cerebral Infarcts in 891 Patients With First Stroke—Lausanne Stroke Registry

<table>
<thead>
<tr>
<th>Etiology</th>
<th>Topography of infarcts</th>
<th>Carotid territory</th>
<th>Vertebobasilar territory</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Entire MCA (n=77)</td>
<td>Deep MCA (n=193)</td>
<td>Superficial MCA (n=311)</td>
</tr>
<tr>
<td>Atherosclerosis with stenosis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>38</td>
<td>16</td>
<td>35</td>
</tr>
<tr>
<td>Embolicigenic heart disease</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>25</td>
<td>8</td>
<td>24</td>
</tr>
<tr>
<td>Combined etiology—atherosclerosis with</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>stenosis and embolicigenic heart</td>
<td>5</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>disease</td>
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<tr>
<td>Hypertensive arteriolopathy</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>0</td>
<td>47</td>
<td>0</td>
</tr>
<tr>
<td>Combined etiology—hypertensive</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>arteriolopathy and embolicigenic heart</td>
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<td>6</td>
<td>0</td>
</tr>
<tr>
<td>disease</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Atherosclerosis</td>
<td>12</td>
<td>9</td>
<td>20</td>
</tr>
<tr>
<td>without stenosis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other etiology—arterial dissection</td>
<td>15</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Other etiologies</td>
<td>0</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Undetermined etiology</td>
<td>5</td>
<td>7</td>
<td>9</td>
</tr>
</tbody>
</table>

Data are percent of column. MCA, middle cerebral artery; ACA, anterior cerebral artery; ICA, internal carotid artery; PCA, posterior cerebral artery.

*Hemodynamic cardiac cause (no embolism): hypotension due to acute bradycardia.
†Isolated thalamic infarct in all cases.

proven herpes zoster angiitis, and phenylpropanolamine angiopathy, in one patient each. It is likely that some of the 29 cases with undetermined etiology had amyloid angiopathy, especially in four patients with dementia, but this remained unproven. A toxic angiopathy (due to amphetamine compounds) may have been underestimated in patients not undergoing angiography.

Transient Ischemic Attacks

TIAs occurred prior to stroke in 234 patients (25% of the 891 patients with infarction, 8% of the 109 patients with hemorrhage); 96% of the patients with TIAs were admitted after an infarct and 4% after a hemorrhage. In patients with infarction, previous TIAs were more common when the etiology was embolic (atherosclerotic source in 29%, cardiac source in 30%) than when the etiology was hypertensive arteriolopathy (18%) (p<0.05). TIAs ipsilateral to later infarction were not more often associated with atherosclerotic than cardiac source of embolism (Table 4); however, TIAs were more common with ipsilateral stenosis (30%) than without (13%) (p<0.01).

Neurologic Deficit

Neurologic deficit was complete immediately or within a few minutes after stroke onset in 62.7% of the patients overall, progressed smoothly in 30.7% (<1 hour in 10.3%, 1–24 hours in 10.5%, >24 hours in 9.9%), and fluctuated in 6.6% (<24 hours in 3.1%, >24 hours in 3.5%). A progressive onset was most common in patients with hemorrhages, but an immediately complete deficit was usual in patients with infarcts, especially in those with embolicigenic heart disease (Table 4, p<0.05); on the other hand, a progressive onset was not uncommon in patients with hypertensive arteriolopathy (40%).

Convulsions at onset were observed in patients with cerebral venous thrombosis (two of three cases) or hemorrhages (7%) but never in patients with embolicigenic heart disease and rarely in those with atherosclerotic source of embolism (1%). Syncope (transient sudden loss of consciousness for no more than a few minutes) at onset was uncommon (1.9% overall) and had no specific etiologic meaning (Table 4). Headaches at onset occurred in 23% of the patients overall (diffuse in 24% of all headaches, frontal in 38%, temporal in 13%, occipitoparietal in 18%, occipitofrontal in 7%), more often in patients with hemorrhage (40%) than in those with infarction (15%) (Table 4, p<0.05). Neck pain occurred in six patients with an ipsilateral carotid lesion (occlusion in three, dissection in two, tight stenosis in one).

Decreased consciousness (somnolence or coma) on admission was noted in 16.8% of the patients...
TABLE 2. Unusual Causes of Cerebral Infarcts Among 891 Patients With First Stroke—Lausanne Stroke Registry

<table>
<thead>
<tr>
<th>Cause</th>
<th>Territory</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Carotid (n = 658)</td>
</tr>
<tr>
<td>Fibromuscular dysplasia</td>
<td>4</td>
</tr>
<tr>
<td>Sacciform aneurysm</td>
<td>1</td>
</tr>
<tr>
<td>Cerebral venous thrombosis</td>
<td>3*</td>
</tr>
<tr>
<td>Isolated intracranial diffuse angiopathy</td>
<td>1</td>
</tr>
<tr>
<td>Hematologic conditions</td>
<td></td>
</tr>
<tr>
<td>Polycythemia</td>
<td>0</td>
</tr>
<tr>
<td>Thrombocythemia</td>
<td>1</td>
</tr>
<tr>
<td>Myeloma with hyperviscosity</td>
<td>1</td>
</tr>
<tr>
<td>Migraine</td>
<td>9†</td>
</tr>
<tr>
<td>Arteritis</td>
<td></td>
</tr>
<tr>
<td>Histologically proven</td>
<td></td>
</tr>
<tr>
<td>Giant-cell arteritis</td>
<td>0</td>
</tr>
<tr>
<td>Churg-Strauss disease</td>
<td>0</td>
</tr>
<tr>
<td>Without histologic proof</td>
<td></td>
</tr>
<tr>
<td>Sarcoidosis</td>
<td>1</td>
</tr>
<tr>
<td>Syphilis</td>
<td>2</td>
</tr>
<tr>
<td>Herpes zoster</td>
<td>1</td>
</tr>
<tr>
<td>Systemic lupus erythematosus</td>
<td>3</td>
</tr>
<tr>
<td>Isolated angiitis of central nervous system</td>
<td>4</td>
</tr>
<tr>
<td>Mitochondrial cytopathy</td>
<td>0</td>
</tr>
</tbody>
</table>

Data are number of cases. Carotid territory includes watershed and multiple-territory infarcts from Table 1.

*Postpartum in one case.
†With ergot intoxication in three cases.

Overall, more often in those with hemorrhage (50%) than in those with infarction (12% in embolic infarctogenic heart disease, 3% in hypertensive arteriolopathy) (Table 4,
P<0.01).

Motor plus sensory plus visual field involvement was found in 13.6% of the patients overall, motor plus sensory involvement with preserved visual fields in 23.8%, and motor involvement with sensory and visual field sparing in 39.2%. Overall, 83.2% of the patients had a motor deficit and 46.4% had a sensory deficit. Pure motor hemiparesis (face and/or upper limb and/or lower limb) was associated with hypertensive arteriolopathy (Table 4, P<0.05) but was not specific for it since 20% of embolic infarcts and 17% of hemorraghes produced pure motor hemiparesis. This was true even when only patients with proportional faciobrachiocruural pure motor hemiparesis were considered; only 41% had hypertensive arteriolopathy.

Pure sensory stroke was found in only 2% of the patients overall and was not significantly associated with hypertensive arteriolopathy (Table 4). Face-arm-leg distribution was the most common type of motor (44.4%) and sensory (55.5%) involvement, followed by face-arm distribution (32% and 29%, respectively), arm-leg distribution (13.4% and 7%), pure arm (8% and 6%), pure face (0.2% and 2%), and pure leg (2% and 0.5%) distributions.

Speech disturbances were present in 46% of the patients overall (dysarthria in 12.4%, motor aphasia in 12.7%, sensory aphasia in 9.7%, global aphasia in 11.2%). Fifty-six percent of the 549 patients with left-sided involvement had aphasia. Only one of the 17 patients with Wernicke's aphasia without other neurologic disturbance had a hemorrhage; 15 of the remaining 16 patients with ischemic stroke (temporoparietal) had embolic infarcts. Of the 16, eight had an atherosclerotic source, six had a cardiac source, one had the possibility of both sources, and one had an undetermined source of embolism. Also, only one of the 16 patients with isolated Wernicke's aphasia and hemianopia had a hemorrhage; eight of the 15 patients with ischemic stroke had an atherosclerotic source, four had a cardiac source, and three had another or an undetermined etiology of infarction. Three patients had global aphasia without other lateralizing signs in the setting of an atherosclerotic source of embolism (frontal plus temporoparietal infarct in two, temporal infarct in one).

Mean duration of stay in the hospital was 20 days. Overall mortality was 5.9%, but mortality was more predominant in patients with hemor-

TABLE 3. Etiology Correlated With Topography of Cerebral Hemorrhages in 109 Patients With First Stroke—Lausanne Stroke Registry

<table>
<thead>
<tr>
<th>Etiology</th>
<th>Lenticular-capsular (n = 46)</th>
<th>Lobar (n = 43)</th>
<th>Cerebellar (n = 9)</th>
<th>Brainstem (n = 7)</th>
<th>Thalamic (n = 4)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertensive arteriolopathy</td>
<td>72</td>
<td>19</td>
<td>67</td>
<td>57</td>
<td>100</td>
</tr>
<tr>
<td>Anticoagulant therapy</td>
<td>0</td>
<td>12†</td>
<td>11</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Sacciform aneurysm</td>
<td>0</td>
<td>19</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Arteriovenous malformation</td>
<td>0</td>
<td>14</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Other etiologies*</td>
<td>4</td>
<td>4</td>
<td>0</td>
<td>14</td>
<td>0</td>
</tr>
<tr>
<td>Undetermined etiology</td>
<td>24</td>
<td>32</td>
<td>22‡</td>
<td>29</td>
<td>0</td>
</tr>
</tbody>
</table>

Data are percent of column.

*Associated with arteriolopathy in 10 patients.
†Two patients had hypertension.
### Table 4. Clinical Features Related to Selected Etiologies—Lausanne Stroke Registry

<table>
<thead>
<tr>
<th>Clinical features</th>
<th>Atherosclerosis (n = 427)</th>
<th>Embolic heart disease (n = 204)</th>
<th>Hypertensive arteriolopathy (n = 147)</th>
<th>Cerebral hemorrhage (n = 109)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Transient ischemic attack(s)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Previous</td>
<td>29</td>
<td>30</td>
<td>18</td>
<td>8</td>
</tr>
<tr>
<td>Ipsilateral</td>
<td>23</td>
<td>23</td>
<td>14</td>
<td>6</td>
</tr>
<tr>
<td>Deficit</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Immediately complete</td>
<td>66</td>
<td>82</td>
<td>54</td>
<td>44</td>
</tr>
<tr>
<td>Progressive</td>
<td>27</td>
<td>13</td>
<td>40</td>
<td>52</td>
</tr>
<tr>
<td>Fluctuating</td>
<td>7</td>
<td>5</td>
<td>6</td>
<td>4</td>
</tr>
<tr>
<td>At onset</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Convulsions</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>7</td>
</tr>
<tr>
<td>Syncope</td>
<td>2</td>
<td>3</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Headaches</td>
<td>17 (17)</td>
<td>18 (19)</td>
<td>7 (7)</td>
<td>40 (51)</td>
</tr>
<tr>
<td>Decreased consciousness</td>
<td>10</td>
<td>9</td>
<td>3</td>
<td>28</td>
</tr>
<tr>
<td>Somnolence</td>
<td>3</td>
<td>3</td>
<td>0</td>
<td>22</td>
</tr>
<tr>
<td>Coma</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lesion seen on computed tomography</td>
<td>82</td>
<td>92</td>
<td>71</td>
<td>100</td>
</tr>
<tr>
<td>Pure motor hemiparesis</td>
<td>20</td>
<td>21</td>
<td>54</td>
<td>17</td>
</tr>
<tr>
<td>Pure sensory stroke</td>
<td>2</td>
<td>1</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Death</td>
<td>5</td>
<td>5</td>
<td>1</td>
<td>20</td>
</tr>
</tbody>
</table>

Data are percent of column. Cases with mixed etiologies have been included. Percent in noncomatose patients in parentheses.

rhage (Table 4, p<0.01). Death occurred a mean of 4.5 weeks after admission. Fifty-one of the 59 deaths were directly or indirectly due to stroke, including six bronchopneumonias. Among the survivors, 6.7% had no sequelae at discharge, 40.7% had minor sequelae and could return to all previous activities, 35.5% had moderate sequelae that limited previous activities, and 11.6% had severe sequelae and could not return to any previous activities. Disability at discharge was not influenced significantly by the etiologic type of stroke.

### Risk Factors

The distribution of risk factors among selected etiologic types of stroke is summarized in Table 5. Hypertension was present in 45.5% of the patients overall, diabetes mellitus in 12.6%, cigarette smoking in 45.6%, hypercholesterolemia in 14.5%, and venous hematocrit of >0.45 in 25.8% (≥0.50 in 4.6%). Apart from hypertensive arteriolopathy, for which hypertension was a diagnostic criterion, the prevalence of hypertension was highest in hemorrhage (55%). The prevalence of cigarette smoking was highest in patients with infarction due to atherosclerotic source of embolism (Table 5, p<0.01).

A history of oral contraceptive use was elicited in 64% of the women <45 years old. A history of migraine was present in 26% of the patients overall. Vascular claudication of the lower limbs was present in 5.1%, a history of rheumatic fever in 0.7%, and a history of ischemic heart disease in 21.7% (isolated angina in 13.5%, old [<3 months] myocardial infarction in 7.3%, recent [<3 months] myocardial infarction in 0.9%).

There were ischemic changes on ECG in 9.9% of the patients without known ischemic heart disease. Atrial fibrillation was known in 6% of the cases overall, and in 2.3% of the patients with no history of atrial fibrillation ECG at admission or Holter monitoring demonstrated atrial fibrillation; 27% of atrial fibrillations were discovered in the hospital. Left ventricular hypertrophy on ECG was found in 74 patients. Sick sinus syndrome was present in 12 patients, nine had the syndrome previously diagnosed and in three patients it was found on Holter monitoring. Holter monitoring in 99 patients showed an undiagnosed embolic dysrhythmia in 16 patients, including tachycardia-bradycardia syndrome in three, paroxysmal atrial fibrillation in four, and other major dysrhythmia in nine.

Two-dimensional echocardiography in 281 patients showed no abnormalities in 41%. Among the remaining 59% undergoing echocardiography, mitral valve prolapse was suspected on auscultation in 70% and demonstrated in 57 patients. Segmental left ventricular akinesia was demonstrated in 48 patients including five with a visible thrombus, mitral stenosis in six, global dilatation of the heart in six patients including one with an intraventricular thrombus, endocarditis in two, aortic and mitral stenosis and insufficiency in two, aberrant left ventricular chorda tendinae in one, left intra-atrial thrombus in one,
TABLE 5. Risk Factors Related to Selected Etiologies—Lausanne Stroke Registry

<table>
<thead>
<tr>
<th>Risk factor</th>
<th>Atherosclerosis (n=427)</th>
<th>Embolic heart disease (n=204)</th>
<th>Hypertensive arteriolopathy (n=147)</th>
<th>Cerebral hemorrhage (n=109)</th>
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</thead>
<tbody>
<tr>
<td>Hypertension</td>
<td>46</td>
<td>32</td>
<td>100</td>
<td>55</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
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<td>8</td>
<td>20</td>
<td>9</td>
</tr>
<tr>
<td>Cigarette smoking</td>
<td>64</td>
<td>39</td>
<td>31</td>
<td>30</td>
</tr>
<tr>
<td>Hypercholesterolemia</td>
<td>19</td>
<td>12</td>
<td>22</td>
<td>6</td>
</tr>
<tr>
<td>Hematocrit &gt; 0.45</td>
<td>30</td>
<td>25</td>
<td>25</td>
<td>21</td>
</tr>
<tr>
<td>History of ischemic heart disease</td>
<td>22</td>
<td>51</td>
<td>20</td>
<td>9</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>2</td>
<td>29</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>On admission</td>
<td>1</td>
<td>31</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>&gt;50% stenosis or occlusion of corresponding artery</td>
<td>53</td>
<td>13</td>
<td>0</td>
<td>4</td>
</tr>
</tbody>
</table>

Data are percent of column. Cases with mixed etiologies have been included.

and left atrial myxoma (confirmed at operation) in one patient. Only one of 13 patients with prosthetic mitral or aortic valves had a visible (intra-atrial) thrombus. Overall, only 7% of the 137 patients with a potential cause of embolism demonstrated on echocardiography had no history of heart disease. In 29 patients, echocardiography showed anomalies (such as valvular sclerosis, aortic insufficiency, and mitral annulus calcification) without well-established embolic risk.

Doppler ultrasonography detected a ≥50% ipsilateral ICA stenosis or occlusion in 34% of the patients with infarcts in the carotid system (50–74% stenosis in 9%, 75–90% stenosis in 3%, >90% stenosis in 4%, occlusion in 18%) (Table 5); a <50% ipsilateral ICA stenosis was suspected in 34% of the patients. On the asymptomatic side, 46% of the patients had a <50% stenosis, 4% had a 50–74% stenosis, 2% had a 75–90% stenosis, 1% had a >90% stenosis, and 1% had an occlusion. In 24% of the patients with vertebrobasilar infarcts, one vertebral artery could not be detected or showed no diastolic blood flow.

Cerebral angiography in 288 patients showed intracranial disease and dysplasia not detected on Doppler ultrasonography in 20% of the patients. Angiography also added significant information in 55% of the patients with extracranial lesions detected by Doppler ultrasonography, including morphologic data, tandem lesions, and distal intracranial occlusions.

Discussion

The Lausanne Stroke Registry was limited to patients with first stroke admitted to the Centre Hospitalier Universitaire Vaudois and was not population-based. Epidemiologically, it is also probably biased negatively in some categories of stroke, such as rapidly lethal strokes or those with rapidly reversible symptoms. However, the Lausanne Stroke Registry may give useful information on patients with a first stroke who are admitted to a primary-care hospital.

Our findings confirm the general male preponderance in stroke but also suggest that there may be a female preponderance in extreme age groups (<30 and ≥80 years old). We also found an association between sex and the ischemic (female>male) or hemorrhagic (male>female) nature of stroke in patients younger than 50. These findings may be partially explained by a differential distribution of risk factors, such as oral contraceptive use by two thirds of the women <45 years old. Female preponderance in those >80 years old may be related to lower life expectancy in men in general and earlier death in men with vascular risk factors; the >80-year-old age group was the only one in which the sex distribution was equal to that of the general population.

The most common risk factors were hypertension and cigarette smoking. Hypertension was associated mainly with deep infarcts and hemorrhages, and cigarette smoking was associated significantly with infarcts due to atherosclerosis. While smoking remains a controversial risk factor for stroke, it is one of the main factors associated with development and progression of carotid atherosclerosis.

With reference to stroke etiology, one of the main differences between our Lausanne Stroke Registry using systematic Doppler ultrasonography and previous studies is that we found a lower proportion of infarcts attributed to atherosclerosis (Table 6). In a recent review of the studies completed in the West, Kurtzke found that 68.5% of strokes were due to thromboembolism, 5% to cardioembolism, and 11.5% to hemorrhage. We attributed 43.2% of strokes to atherosclerosis, including 16.2% with atherosclerosis without stenosis and 2.8% with coex-
### Table 6. Diagnosis of Stroke—Survey of Literature

<table>
<thead>
<tr>
<th>Study</th>
<th>No.</th>
<th>Atherosclerosis</th>
<th>Embolic heart disease</th>
<th>Hypertensive arteriolopathy (lacunar stroke)</th>
<th>Intraparenchymal hemorrhage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Framingham, Massachusetts*15</td>
<td>74</td>
<td>77</td>
<td>18</td>
<td></td>
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<tr>
<td>Goulburn, Australia16</td>
<td>142</td>
<td>71</td>
<td>7</td>
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<td>19</td>
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<tr>
<td>Fargo, North Dakota–Moorhead, Minnesota17</td>
<td>378</td>
<td>41</td>
<td>4</td>
<td></td>
<td>17</td>
</tr>
<tr>
<td>Rochester, Minnesota*18</td>
<td>930</td>
<td>75</td>
<td>10</td>
<td></td>
<td>11</td>
</tr>
<tr>
<td>Chicago Stroke Study19</td>
<td>198</td>
<td>80</td>
<td>1</td>
<td></td>
<td>12</td>
</tr>
<tr>
<td>Tartu, USSR20</td>
<td>667</td>
<td>(80)</td>
<td></td>
<td></td>
<td>13</td>
</tr>
<tr>
<td>Framingham, Massachusetts*21</td>
<td>294</td>
<td>59</td>
<td>14</td>
<td></td>
<td>5</td>
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<tr>
<td>Harvard Cooperative Stroke Registry2</td>
<td>649</td>
<td>36</td>
<td>33</td>
<td></td>
<td>20</td>
</tr>
<tr>
<td>National Survey of Stroke22</td>
<td>927</td>
<td>60</td>
<td>9</td>
<td></td>
<td>14</td>
</tr>
<tr>
<td>Tilburg, The Netherlands*23</td>
<td>502</td>
<td>76</td>
<td>10</td>
<td></td>
<td>11</td>
</tr>
<tr>
<td>Austin, Australia24</td>
<td>616</td>
<td>32</td>
<td>8</td>
<td></td>
<td>21</td>
</tr>
<tr>
<td>Oxfordshire Community Stroke Project*24</td>
<td>140</td>
<td>(93)</td>
<td></td>
<td></td>
<td>7</td>
</tr>
<tr>
<td>Oxfordshire Community Stroke Project*3</td>
<td>269</td>
<td>(90)</td>
<td></td>
<td></td>
<td>10</td>
</tr>
<tr>
<td>South Alabama*25</td>
<td>151</td>
<td>48†</td>
<td>28</td>
<td></td>
<td>13</td>
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<tr>
<td>Pilot Stroke Data Bank3</td>
<td>809</td>
<td>21</td>
<td>25</td>
<td></td>
<td>12</td>
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<tr>
<td>Lehigh Valley, Pennsylvania26</td>
<td>1,026</td>
<td>76</td>
<td>13</td>
<td></td>
<td>8</td>
</tr>
<tr>
<td>Stockholm, Sweden27</td>
<td>402</td>
<td>59</td>
<td>20</td>
<td></td>
<td>8</td>
</tr>
<tr>
<td>Community hospital-based stroke programs8</td>
<td>4,132</td>
<td>65</td>
<td>22</td>
<td></td>
<td>10</td>
</tr>
<tr>
<td>Lausanne Stroke Registry, Switzerland (current study)</td>
<td>1,000</td>
<td>39 (43)</td>
<td>16 (20)</td>
<td>13 (15)</td>
<td>11</td>
</tr>
</tbody>
</table>

Cases with mixed etiology included in parentheses.
*Population-based study.
†Including unspecified origin of infarction.

isting embolic heart disease. This difference may be due to the fact that in previous studies, hypertensive lacunar infarcts (arteriolopathy; 13.2% in our registry) were usually included in the "arterial thrombosis" group and that cardioembolic strokes (20.4% in our registry) were probably underestimated, as suggested recently. In the Lausanne Stroke Registry, atherosclerotic stenoses or occlusions were the most common cause of entire or superficial MCA territory infarcts and watershed infarcts, while embolic heart disease was a nonnegligible cause of superficial MCA territory and cerebellar infarcts. It must be emphasized that one third of infarcts in the territory of a deep perforating artery were associated with an appropriate atherosclerotic stenosis or a potential cardiac source of embolism, often in the absence of hypertension. This finding suggests that several so-called "lacunar" infarcts may be due not to intracranial hypertensive arteriolopathy but to embolism related to extracranial atherosclerosis or heart disease. A separate as-yet unpublished study in this group of patients showed that the size of small deep infarcts on CT was not related to any specific etiology. Also, multiple asymptomatic infarcts on CT did not indicate a particular cause of infarction.

Causes of infarction other than atherosclerosis, embolic heart disease, intracerebral arteriolopathy (lacune), and dissection were uncommon (6%). In only 8.3% of the patients was no cause of infarction found, while in another 18.2% the sole determined etiology was a minor arterial lesion (plaques, <50% stenosis), the etiologic significance of which remains controversial.

Lenticular-capsular hemorrhages were associated with hypertension, but lobar hemorrhages were due to more varied causes. In more than one quarter of the patients with hemorrhage no etiology could be determined, but undiagnosed amyloidosis, small angioma, or amphetamine use may be the most likely causes in these patients.

Our Lausanne Stroke Registry confirms that TIAs more often precede an infarct than a hemorrhage, but 8% of the patients with hemorrhage may have suffered previous TIAs, as in the Harvard Cooperative Stroke Registry. We did not find that previous TIAs ipsilateral to cerebral infarction were more suggestive of atherosclerosis than of embolic heart disease. However, infarcts due to ≥50%
stenosis or occlusion were significantly associated with previous ipsilateral TIAs.

Few clinical characteristics differentiated patients with infarcts from those with hemorrhages because large infarcts mimicked the classical picture of hemorrhage, while lobar or small deep hemorrhages sometimes mimicked infarction. Also, we did not find specific criteria that differentiated clinically and with accuracy the infarcts due to atherosclerosis, embolic heart disease, and hypertensive arteriolopathy. A maximal neurologic deficit was the most common type of onset in patients with infarction in general and was particularly suggestive of embolic heart disease, as in the Harvard Cooperative Stroke Registry2 and the Michael Reese Stroke Registry. A progressive onset first suggested hemorrhage, but also hypertensive arteriolopathy, confirming the findings of the Harvard Cooperative Stroke Registry.2 Focal convulsions at onset were very uncommon, but when present they were very suggestive of venous thrombosis or hemorrhage. As reported previously,9 we found that coma first suggested hemorrhage (one of four to five patients with hemorrhage), then entire MCA territory infarction. We could confirm that the classical "hemorrhagic picture," with headaches, progressive neurologic deficit, and decreased consciousness, was significantly associated with hemorrhages, but only one third of the patients with hemorrhage had this picture, which limits its clinical usefulness.

A pure motor deficit was not at all specific for hypertensive arteriolopathy since pure motor deficit was associated with another type of infarction or with hemorrhage in two thirds of the patients. This was true also for pure motor hemiparesis with proportional faciobrachiocrural distribution, in which only half the patients had hypertensive arteriolopathy, while lobar or small deep hemorrhages mimicked the classical "hemorrhagic picture," with headaches, and was associated with another type of infarction or hypertensive arteriolopathy since pure motor deficit was associated with another type of infarction or hemorrhage. As reported previously,9 we found that coma first suggested hemorrhage (one of four to five patients with hemorrhage), then entire MCA territory infarction. We could confirm that the classical "hemorrhagic picture," with headaches, progressive neurologic deficit, and decreased consciousness, was significantly associated with hemorrhages, but only one third of the patients with hemorrhage had this picture, which limits its clinical usefulness.

The presence of speech disturbances had no particular etiologic association, except for Wernicke's aphasia; this type of aphasia suggested atherosclerotic or cardiac sources of embolism.

In-hospital mortality was 5.9% in general and 20% in patients with hemorrhage, which is lower than the usual figures of 15–30%.8,22,31,32 The explanation for this is not clear, but it may be partially related to our referral patterns, which limited entry to patients with a first stroke, and to improved care in the acute phase. This latter point is suggested by the fact that functional disability in survivors was similar to that reported in previous studies.8,22

We confirmed that ECG can detect and quantify many cardiac abnormalities, and we believe that ECG should be a routine investigation in stroke patients. Holter monitoring was also an extremely useful tool in selected patients in whom a paroxysmal dysrhythmia was suspected. Although Holter monitoring was used in less than one tenth of our patients, in 16% of those patients such monitoring allowed us to diagnose an embolic dysrhythmia. Echocardiography could not be considered a good screening tool except in the search for mitral valve prolapse in young adults, but echocardiography did prove useful in the investigation of patients with known cardiac abnormality, such as the detection of a thrombus in patients with atrial fibrillation or myocardial infarction. Doppler ultrasonography was of limited use in patients with vertebrobasilar infarcts but extremely valuable in those with ICA territory infarcts because it permitted detection of ipsilateral $\geq 50\%$ stenosis or occlusion in 33% of the patients and in 8% contralaterally. Angiography in selected patients showed an abnormality not detected by Doppler ultrasonography in 20% and allowed us to better delineate the arterial lesion in 55%.

The Lausanne Stroke Registry has provided us with extremely valuable information on the epidemiology, etiology, clinical characteristics, and short-term prognosis in patients with first stroke. This knowledge, as well as specific studies in particular subgroups of patients, would not have been possible without this registry. We agree with Mohr, who recently reviewed the value of stroke data banks,1 that to obtain reliable information it is necessary to see every patient and to code the individual data. With such a personal investment of time and energy by the investigators, detailed, complete, and accurate data may be obtained prospectively with limited financial investment.

Acknowledgments

We thank all the people whose work allowed us to realize this registry, with a special mention to the residents and staff of the Departments of Neurology, Radiology (A. Uske, MD; G. Candardjis, MD), and Cardiology (L. Kappenberger, MD, Centre Hospitalier Universitaire Vaudois, Lausanne, Switzerland; Mona Britton, MD, Stockholm, Sweden, and Jay P. Mohr, MD, New York, New York, provided helpful comments on the first version of the manuscript.

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KEY WORDS • cerebrovascular disorders • epidemiology • tomography, x-ray computed • ultrasonics
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J Bogousslavsky, G Van Melle and F Regli

Stroke. 1988;19:1083-1092
doi: 10.1161/01.STR.19.9.1083

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