Autopsy Prevalence of Coronary Atherosclerosis in Patients With Fatal Stroke

Fernando Gongora-Rivera, MD; Julien Labreuche, BS; Arturo Jaramillo, MD; Philippe Gabriel Steg, MD; Jean-Jacques Hauw, MD; Pierre Amarenco, MD

Background and Purpose—Myocardial infarction (MI) is the leading cause of long-term mortality in patients with stroke, yet the prevalence of coronary atherosclerosis in these individuals is unknown. The objective of the study was to establish the prevalence of coronary atherosclerosis and MI after fatal stroke.

Methods—Using an autopsy data bank, we studied the prevalence of coronary plaques and coronary stenoses >50% and pathologic evidence of MI in 803 consecutive autopsies of neurologic patients.

Results—Coronary plaques, coronary stenoses, and MI were present in 72.4%, 37.5%, and 40.8%, respectively, of the 341 patients with stroke and in 26.8%, 10.1%, and 12.8%, respectively, of the 462 patients with other neurologic diseases (P<0.001). Two-thirds of cases of MI were clinically silent and found at autopsy. Compared with other neurologic diseases, and after adjusting for age, gender, and heart weight, the odds ratios (95% confidence intervals) of the presence of coronary plaques, coronary stenosis, and MI in stroke patients were 3.81 (2.66 to 5.46), 2.80 (1.85 to 4.25), and 2.34 (1.58 to 3.46), respectively. The frequency of coronary atherosclerosis and MI was similar between stroke subtypes. The prevalence of coronary plaques, coronary stenosis, and MI was 79.0%, 42.9%, and 46.0%, respectively, in the presence of plaques in any segment of the extracranial and intracranial brain arteries, and 50.8%, 17.9%, and 23.9%, respectively, in the absence of plaques (adjusted P<0.01). Coronary atherosclerosis was also related to the severity of atherosclerosis in any segment of the cerebral arteries (adjusted probability value for linear trend <.005).

Conclusions—Coronary atherosclerosis and MI are highly prevalent in patients who died from a stroke regardless of the etiology. They are more frequent when atherosclerosis is present in the carotid and cerebral arteries. They are also common in stroke patients with no evidence of carotid or cerebral atherosclerosis. (Stroke. 2007;38:1203-1210.)

Key Words: atherosclerosis ■ cerebral infarction ■ coronary arteries ■ myocardial infarction

In stroke registries, atherothrombotic disease accounts for 9% to 20% of stroke cases.1,2 Overall, one-quarter of patients with stroke have a history of a symptomatic coronary event.2 After a first stroke, patients are at high risk for another stroke and at low, but not negligible, risk of myocardial infarction in the next 2 years.3 Subsequently, the 5-year risk of cardiac death is 2- to 3-times higher than that of recurrent fatal stroke.4,5 This early low risk of coronary heart disease and late high cardiac death rate justifies the use of composite endpoints (ie, stroke, myocardial infarction, and vascular death) in long-term secondary prevention trials. Yet the rate of presymptomatic coronary artery disease in patients with stroke is unknown. Small series suggest that, even in stroke patients who have never had cardiac symptoms, it could reach 20%.6,7 One series of 69 patients found more frequent positive exercise test in the presence of large artery disease as compared with penetrating artery disease or stroke of undetermined origin.8 No data exist on the relative frequency of coronary atherosclerosis according to etiologic stroke subtype in a large population.

The aim of our study is to establish the prevalence of coronary atherosclerosis and myocardial infarction after a fatal stroke and to compare it with the prevalence in patients with other fatal neurologic diseases. To achieve this aim, we analyzed the results of a unique, large, single-center, consecutive autopsy series.

Methods

The Multiple Atherosclerosis Site in Stroke (MASS) study is an autopsy database of unselected patients with neurologic diseases and stroke collected at La Salpêtrière hospital in Paris between November 1982 and February 1989, at which time the autopsy rate was 73%.9

The methods have been reported previously.10 Briefly, each pathologic report detailed the anatomy of the extracranial and intracranial arteries, with systematic drawing of the site of occlusion.
as well as the site and extent of atherosclerotic plaque or stenosis, as reported previously.\textsuperscript{11,12} Stenoses of brain arteries were graded, blinded to the clinical etiologic subtypes, according to percent reduction in luminal diameter and then categorized as 0, 1 (stenosis 30\% to 74\%), 2 (stenosis 75\% to 99\%), or 3 (occlusion) at any segment (common carotid artery proximal and distal; carotid artery bulb; origin, subpetrosal segment, petrosal segment, siphon and termination of the internal carotid artery; M1 proximal to distal; M2, A1 and A2; P1 and P2). Microscopic sections of the main vascular lesions were available for review to distinguish embolic occlusion from occlusive thrombus superimposed on a ruptured plaque. The original sections of the brain and extracranial and intracranial arteries, including the aortic arch and the heart, were available (stored in formaldehyde), allowing the verification of data, if necessary. The bony segment of the brain arteries was also available, as described elsewhere.\textsuperscript{11,12} The aorta and all supra-aortic arteries were dissected and the cervical spine and base of the skull excised before gross examination and microscopic study.\textsuperscript{13}

Among 886 autopsies, 505 were from patients with neurologic diseases other than stroke and 381 were from patients with stroke, including 83 with brain hemorrhage, 288 with brain infarction, and 10 with both brain hemorrhage and infarction. The neurological disease was categorized according to clinical data and pathologic description. Stroke subtypes were classified according to the Étude du Profil Généétique de l’Infarctus Cérébral (GENIC) classification:\textsuperscript{14} (1) atherosclerotic: ipsilateral internal carotid artery stenosis of >30\% or ipsilateral stenosis of >50\% of other intracranial or extracranial artery, or a thrombotic occlusion superimposed on stenosis; (2) cardioembolic: cardiac source of embolism (recent myocardial infarction, atrial fibrillation, intracardiac thrombus or tumor, valvulopathy, or endocarditis); lacunar infarcts were defined by a deep infarct size <1.5 cm, appropriate clinical syndrome, and no cardiac, aortic, or extracranial/intracranial artery source of embolism/occlusion; (3) other cause: disseminated intravascular coagulation and other hematologic causes, inflammatory/infectious arterial disease (vasculitis), or intracranial or carotid dissection; (4) coexisting causes: ≥2 possible etiologies as defined here; and (5) unknown cause: when no identifiable cause was found.

The group with fatal neurologic diseases and no history or pathologic evidence of stroke (n = 505) included patients with degenerative and demyelinating diseases (n = 152: Alzheimer disease, Parkinson disease, progressive supranuclear palsy, amyotrophic lateral sclerosis, other degenerative diseases, and multiple sclerosis), brain tumors (n = 202), infections (n = 92: acquired immune deficiency syndrome, brain abscess, meningitis, Creutzfeldt-Jakob disease), trauma (n = 4), toxic diseases, myelopathy, polynuropathy, Guillain-Barré syndrome, myasthenia gravis, and other neurologic disorders with normal brain tissue (n = 55).

Patients who died from other neurological disease with a clinically silent brain infarct found at autopsy were classified in the other neurological disease group (n = 4). Patients with clinically symptomatic stroke were all classified in the stroke group, even if they eventually died from other neurological disease. More than 90\% of the patients in the stroke group died from their stroke (herniation or silent). Any macroscopically abnormal area seen at autopsy was sampled for microscopic study. A systematic microscopic study of a cross-section of the left ventricle at the level of mitral papillary muscle was performed in all patients.

### Statistical Analysis

Data are presented as the mean (standard deviation) for continuous variables and the percentage (count) for dichotomous variables. Patients were divided into 2 groups according to pathologic evidence of stroke. Comparisons of means and proportions was performed using Student t test and the $\chi^2$ test, respectively. Among stroke patients, the $\chi^2$ test was used to compare the prevalence of coronary atherosclerosis and myocardial infarction between brain infarction and hemorrhage, and among the main brain–infarction subtypes.

Comparisons of the prevalence of coronary atherosclerosis and myocardial infarction between the 2 groups were adjusted on 3 prespecified confounding factors (age, gender, and heart weight, with the latter reflecting the presence of arterial hypertension) using logistic regression analysis. An additional adjustment was performed by including vascular risk factors (hypertension, diabetes, smoking, obesity, dyslipidemia, and atrial fibrillation) into the model. Crude and adjusted odds ratios (ORs) of the presence of coronary plaques, coronary stenoses, and myocardial infarction were calculated with 95\% confidence intervals (CIs). Sensitivity analyses were restricted to patients without a history of coronary heart disease, and to 220 pairs of patients with or without stroke matched by age (±1 year) and gender (Figure 1). The later was performed because distribution of age between the stroke and the other neurologic disease groups were different (the other neurologic disease group being younger). Matching cases and controls on age ±1 year allowed to eliminate the problem of age as the most important risk factor for coronary heart disease. Sensitivity analysis on matched groups was performed using conditional logistic regression analysis. We investigated the associations of cerebral atherosclerosis and ulcerated plaques in the aortic arch with coronary atherosclerosis and myocardial infarction among 323 stroke patients in whom a detailed report of the anatomy of the cerebral arteries was available. These associations were tested in a multiple logistic regression model including the prespecified confounding factors. Statistical testing was performed at the 2-tailed α level of 0.05. Data were analyzed using the SAS package, release 9.1 (SAS Institute).

### Results

Among 886 consecutive autopsies, 90.6\% (n = 803) underwent macroscopic autopsy examination of the coronary arteries and had complete clinical data and heart weights: 341 patients had pathologic evidence of stroke (median time between event and death, 12 days [interquartile range, 5.0 to 32 days]) and 462 patients had other neurologic diseases. The proportion of patients with missing data who were excluded from the statistical analysis was similar in both groups (10.5\% versus 8.5\%; $P = 0.32$). Patients with stroke were older, had more cardiovascular risk factors, and more frequently had a cardiovascular history (ie, any symptomatic vascular event) than patients with other neurologic diseases (Table 1).

### Prevalence of Coronary Atherosclerosis and Myocardial Infarction in Patients With Neurologic Disease

Coronary plaques were found in 72.4\% (95\% CI, 67.7 to 77.2\%) of patients with stroke versus 26.8\% (95\% CI, 22.8 to 30.9\%) of patients with other neurologic diseases (crude OR, 7.16; 95\% CI, 5.23 to 9.81; $P < 0.001$; Table 2). The presence of at least one coronary stenosis ≥50\% was found more frequently among patients with stroke than in patients with...
other neurologic diseases (crude OR, 5.31; 95% CI, 3.66 to 7.70). Pathologic (macroscopic and/or microscopic) evidence of a previous myocardial infarction was found in 40.8% (95% CI, 35.6 to 46.0%) of patients with stroke versus 12.8% (95% CI, 9.7 to 15.8%) of patients with other neurologic diseases (crude OR, 4.70; 95% CI, 3.32 to 6.66). After controlling for age, gender, and heart weight, the ORs for the presence of coronary plaques, coronary stenosis, and myocardial infarction in patients who died from stroke relative to patients who died from other neurologic diseases were 3.81 (95% CI, 2.66 to 5.46), 2.80 (1.85 to 4.25), and 2.34 (1.58 to 3.46), respectively. Results were similar after additional adjustment for cardiovascular risk factors; the corresponding ORs (95% CIs) were 3.83 (2.53 to 5.81), 2.92 (1.81 to 4.72), and 2.30 (1.47 to 3.61), respectively. The same analysis was performed in the subset of patients (709 of 803) with no history of coronary artery disease, and provided very similar results (Table 3). A sensitivity analysis restricted to age- and gender-matched groups yielded similar results. The corresponding adjusted ORs (95% CIs) were 5.28 (2.45 to 11.37), 2.32 (1.13 to 4.76), and 2.24 (1.03 to 4.86), respectively. The prevalence of coronary plaques according to age and stroke subtype is shown in Table 4.

Two-thirds of the myocardial infarctions had been clinically silent and were found only at autopsy. Of the 72 clinically symptomatic myocardial infarctions, 15% (n=11) occurred within 3 weeks before the stroke. Silent infarctions were more frequent in patients with stroke than in patients with other neurologic diseases (17.9% versus 2.4%; P<.001).

Among the 341 patients with stroke, the prevalence of coronary atherosclerosis and myocardial infarction was sim-

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**TABLE 1. Characteristics of Patients for Whom Complete Clinical and Autopsy Data Were Available**

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Patients With Stroke</th>
<th>Patients With Other Neurologic Diseases</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, mean (SD), y</td>
<td>72.8±11.9</td>
<td>60.3±16.2</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Male, n (%)</td>
<td>275 (59.5)</td>
<td>188 (55.1)</td>
<td>0.213</td>
</tr>
<tr>
<td>Hypertension, n (%)</td>
<td>196 (57.5)</td>
<td>49 (10.6)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Diabetes, n (%)</td>
<td>47 (13.8)</td>
<td>22 (4.8)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Smoking, n (%)</td>
<td>52 (15.3)</td>
<td>29 (6.3)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Obesity, n (%)</td>
<td>35 (10.3)</td>
<td>8 (1.7)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Dyslipidemia, n (%)</td>
<td>25 (7.3)</td>
<td>5 (1.1)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Atrial fibrillation, n (%)</td>
<td>93 (27.3)</td>
<td>12 (2.6)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Heart weight, mean (SD), g</td>
<td>421.9±103.2</td>
<td>347.4±84.6</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Cardiovascular history, n (%)</td>
<td>74 (21.7)</td>
<td>20 (4.3)</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

SD indicates standard deviation.
ilar irrespective of whether they had had a brain hemorrhage or a brain infarction (Table 2). Moreover, there was no difference between the main brain–infarction subtypes, except for a lower prevalence of myocardial infarction in brain infarction of unknown cause ($P < 0.035$ for comparison with each other main subtype).

### Association of Cerebral Atherosclerosis and Myocardial Infarction Among Patients With Stroke

As shown in Figure 2a, coronary atherosclerosis was related to the severity of atherosclerosis in any segment of the cerebral arteries (adjusted probability value for linear trend $<0.005$). The relation of myocardial infarction to severity of cerebral artery atherosclerosis was less marked (adjusted probability value for linear trend $=0.028$). Similar results were observed in relation to the degree of internal carotid artery atherosclerosis (Figure 2b).

The prevalence of coronary plaques was significantly higher in the presence of ulcerated plaques of the aortic arch than in their absence (93.4% versus 66.8%, adjusted $P < 0.001$). However, the presence of ulcerated plaques did not appear to impact on the presence of coronary stenosis or myocardial infarction at autopsy examination (Table 5).

### Discussion

The results of this study show that plaques and stenoses $>50\%$ in the coronary arteries were present in as many as 72% and 38%, respectively, of patients with fatal stroke. These figures were 2- to 4-times higher than in patients who died from other neurologic diseases, even in the sensitivity analysis that was restricted to age- and gender-matched groups. In the presence of stenosis of the internal carotid artery, we found coronary plaques and stenoses in 84% and 47% of cases, respectively. Data from randomized trials show a 2- to 3-year risk of fatal and nonfatal myocardial infarction of only 4% in the placebo group after the acute phase of a stroke.$^{15,16}$ However, randomized trials tend to recruit healthier patients without a history of coronary heart disease or previous myocardial infarction and exclude those with car-

### TABLE 2. Prevalence of Coronary Atherosclerosis and Myocardial Infarction in 803 Patients With Neurologic Diseases

<table>
<thead>
<tr>
<th>Patients, n</th>
<th>Patients With Coronary Plaques, n (%)</th>
<th>Patients With Coronary Stenosis, n (%)</th>
<th>Patients With Myocardial Infarction, n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cerebrovascular disease 341</td>
<td>247 (72.4)</td>
<td>128 (37.5)</td>
<td>139 (40.8)</td>
</tr>
<tr>
<td>Infarction 251</td>
<td>186 (74.1)</td>
<td>98 (39.0)</td>
<td>110 (43.8)</td>
</tr>
<tr>
<td>Atherothrombotic 60</td>
<td>47 (78.3)</td>
<td>25 (41.7)</td>
<td>22 (36.7)</td>
</tr>
<tr>
<td>Cardioembolic 90</td>
<td>65 (72.2)</td>
<td>34 (37.8)</td>
<td>53 (58.9)</td>
</tr>
<tr>
<td>Lacunar 24</td>
<td>20 (83.3)</td>
<td>11 (45.8)</td>
<td>10 (41.7)</td>
</tr>
<tr>
<td>Unknown cause 32</td>
<td>22 (68.8)</td>
<td>10 (31.3)</td>
<td>5 (15.6)</td>
</tr>
<tr>
<td>Coexisting causes 36</td>
<td>31 (86.1)</td>
<td>18 (50.0)</td>
<td>20 (55.6)</td>
</tr>
<tr>
<td>Hemorrhage* 90</td>
<td>61 (67.8)</td>
<td>30 (33.3)</td>
<td>29 (32.2)</td>
</tr>
<tr>
<td>Other neurologic diseases 462</td>
<td>124 (26.8)†</td>
<td>47 (10.1)†</td>
<td>59 (12.8)†</td>
</tr>
</tbody>
</table>

*Including 10 infarctions.
†$P<0.001$ for the comparison with stroke patients after adjusting for age, gender, and heart weight.

### TABLE 3. Prevalence of Coronary Atherosclerosis and Myocardial Infarction in 709 Patients Without Coronary Heart Disease

<table>
<thead>
<tr>
<th>Patients, n</th>
<th>Patients With Coronary Plaques, n (%)</th>
<th>Patients With Coronary Stenosis, n (%)</th>
<th>Patients With Myocardial Infarction, n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cerebrovascular disease 267</td>
<td>182 (68.2)</td>
<td>79 (29.6)</td>
<td>78 (29.7)</td>
</tr>
<tr>
<td>Infarction 188</td>
<td>131 (69.7)</td>
<td>55 (29.3)</td>
<td>59 (31.4)</td>
</tr>
<tr>
<td>Atherothrombotic 48</td>
<td>37 (77.1)</td>
<td>16 (33.3)</td>
<td>13 (23.1)</td>
</tr>
<tr>
<td>Cardioembolic 58</td>
<td>38 (65.5)</td>
<td>14 (24.1)</td>
<td>25 (43.1)</td>
</tr>
<tr>
<td>Lacunar 19</td>
<td>15 (79.0)</td>
<td>7 (36.8)</td>
<td>6 (31.6)</td>
</tr>
<tr>
<td>Unknown cause 27</td>
<td>17 (63.0)</td>
<td>5 (18.5)</td>
<td>2 (7.4)</td>
</tr>
<tr>
<td>Coexisting causes 28</td>
<td>23 (82.1)</td>
<td>13 (46.4)</td>
<td>13 (46.4)</td>
</tr>
<tr>
<td>Hemorrhage* 79</td>
<td>51 (64.6)</td>
<td>34 (30.4)</td>
<td>19 (24.1)</td>
</tr>
<tr>
<td>Other neurologic diseases 442</td>
<td>109 (24.7)‡</td>
<td>36 (8.1)‡</td>
<td>48 (10.9)‡</td>
</tr>
</tbody>
</table>

*Including 9 infarctions.
‡$P<0.001$, †$P<0.02$ for the comparison with stroke patients after adjusting for age, gender, and heart weight.
dioembolic stroke or brain hemorrhage and patients with early mortality. Consequently the data from these trials underestimate the rates of coronary artery disease and cardiac death. An observational study, for example, found a much higher 5-year risk of fatal and nonfatal myocardial infarction of 21%. Acute-phase trials recruiting patients early after the onset of stroke found that 2% to 6% of patients with stroke had a fatal cardiac event within 3 months of their stroke. Clinical studies using stress testing found presymptomatic cardiac disease in 20% to 41% of patients with stroke or transient ischemic attack.

Furthermore, in our study, ≈40% of patients with fatal stroke had evidence of myocardial infarction on pathology, and in two-thirds this was an unexpected autopsy finding. The

### Table 4. Prevalence of Coronary Plaques According to Age Group and Diagnosis

<table>
<thead>
<tr>
<th>Age Group, y</th>
<th>Brain Infarction</th>
<th>Brain Hemorrhage*</th>
<th>Other Neurologic Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Patients, n</td>
<td>Patients With</td>
<td>Patients, n</td>
</tr>
<tr>
<td></td>
<td>Patients With</td>
<td>Coronary Plaques, n (%)</td>
<td>Patients, n</td>
</tr>
<tr>
<td>&lt;40</td>
<td>5</td>
<td>0 (0.0)</td>
<td>1</td>
</tr>
<tr>
<td>40–49</td>
<td>7</td>
<td>1 (14.3)</td>
<td>2</td>
</tr>
<tr>
<td>50–59</td>
<td>20</td>
<td>13 (65.0)</td>
<td>9</td>
</tr>
<tr>
<td>60–69</td>
<td>36</td>
<td>25 (69.4)</td>
<td>20</td>
</tr>
<tr>
<td>70–79</td>
<td>99</td>
<td>77 (77.8)</td>
<td>35</td>
</tr>
<tr>
<td>80–89</td>
<td>75</td>
<td>62 (82.7)</td>
<td>21</td>
</tr>
<tr>
<td>≥90</td>
<td>9</td>
<td>8 (88.9)</td>
<td>2</td>
</tr>
</tbody>
</table>

*Including 10 with both brain hemorrhage and brain infarction.

Figure 2. Prevalence of coronary atherosclerosis and myocardial infarction according to severity of cerebral arteries atherosclerosis: (a) any segment of the cerebral arteries; (b) internal carotid artery origin.
data from clinical trials and observational studies, together with the high prevalence of silent and previously sympotomatic myocardial infarction found at autopsy, emphasize the importance of early recognition of coronary artery disease in patients with stroke.\textsuperscript{15,16} The finding that almost half of the patients with stroke had significant coronary artery stenoses provides a strong rationale for a global approach to the prevention of cardiac and cerebrovascular diseases in patients with stroke.\textsuperscript{15,16} Few data exist on the risk of myocardial infarction after various types of ischemic stroke. Our results were similar across stroke subtypes, including brain hemorrhage. Indeed, patients with brain hemorrhage and ischemic stroke share the same risk factors for atherosclerotic disease. Unexpectedly, patients with lacunar stroke had similar rates of coronary plaque and stenoses to those of patients classified with atherothrombotic or other stroke subtypes. Patients with lacunar stroke are well-recognized to be at very low risk for fatal stroke, which is reflected in the low rate of lacunar stroke in our series. One might attribute this finding to some misclassification of lacunar stroke patients. However, all

| TABLE 5. Prevalence of Coronary Atherosclerosis and Myocardial Infarction in 319 Patients With Stroke |
|--------------------------------------------------|------------------|------------------|
| Plaques in Cerebral Arteries | Absence | Presence | $P^*$ |
| Prevalence of coronary plaques | |
| Any segment of the extracranial and intracranial arteries | 34 (50.8) | 199 (79.0) | <0.001 |
| Any segment of the carotid arteries | 55 (56.7) | 178 (80.2) | <0.001 |
| Internal carotid artery origin | 70 (56.5) | 163 (83.6) | <0.001 |
| Common carotid artery | 171 (69.8) | 62 (83.8) | 0.042 |
| Basilar artery | 144 (67.0) | 88 (85.4) | 0.005 |
| Subclavian or vertebral origin arteries | 146 (67.9) | 87 (83.7) | 0.010 |
| V2, V3, and V4 segments of vertebral artery | 178 (69.1) | 63 (85.1) | 0.019 |
| Posterior cerebral artery\textsuperscript{†} or middle cerebral artery | 149 (70.3) | 83 (78.3) | 0.461 |
| Ulcerated plaques in the aortic arch | 162 (66.8) | 71 (93.4) | <0.001 |
| Prevalence of coronary stenosis | |
| Any segment of the extracranial and intracranial arteries | 12 (17.9) | 108 (42.9) | 0.004 |
| Any segment of the carotid arteries | 21 (21.7) | 99 (44.5) | <0.001 |
| Internal carotid artery origin | 28 (22.6) | 92 (47.2) | <0.001 |
| Common carotid artery | 88 (35.9) | 32 (43.2) | 0.422 |
| Basilar artery | 75 (34.9) | 44 (42.7) | 0.507 |
| Subclavian or vertebral origin arteries | 69 (32.1) | 51 (49.0) | 0.010 |
| V2, V3, and V4 segments of vertebral artery | 90 (37.0) | 28 (37.8) | 0.791 |
| Posterior cerebral artery\textsuperscript{†} or middle cerebral artery | 75 (35.4) | 44 (41.5) | 0.653 |
| Ulcerated plaques in the aortic arch | 84 (36.4) | 36 (47.4) | 0.234 |
| Prevalence of myocardial infarction | |
| Any segment of the extracranial and intracranial arteries | 16 (23.9) | 116 (46.0) | 0.011 |
| Any segment of the carotid arteries | 27 (27.8) | 105 (47.3) | 0.004 |
| Internal carotid artery origin | 39 (31.5) | 93 (47.7) | 0.014 |
| Common carotid artery | 91 (37.1) | 41 (55.4) | 0.011 |
| Basilar artery | 81 (37.7) | 50 (48.5) | 0.262 |
| Subclavian or vertebral origin arteries | 84 (39.1) | 48 (46.2) | 0.344 |
| V2, V3, and V4 segments of vertebral artery | 85 (35.0) | 45 (60.8) | <0.001 |
| Posterior cerebral artery\textsuperscript{†} or middle cerebral artery | 82 (38.7) | 50 (47.2) | 0.294 |
| Ulcerated plaques in the aortic arch | 95 (39.1) | 37 (48.7) | 0.300 |

\textsuperscript{*}Adjusted for age, gender, and heart weight (multiple logistic regression analysis).
\textsuperscript{†}In P1 and P2 segments.

ulcerated plaques in the aortic arch in our series had a particularly high prevalence of coronary atherosclerosis. This probably accounts for their previously reported high risk of cardiac events.\textsuperscript{25}

Patients with ulcerated plaques in the aortic arch in our series had a particularly high prevalence of coronary atherosclerosis. This probably accounts for their previously reported high risk of cardiac events.\textsuperscript{25}
prevalence of cardioembolic strokes and low prevalence of lacunar strokes. However, categorizing the patients by etiologic stroke subtype allowed a good approximation of the relative prevalence of coronary atherosclerosis between each stroke subtype. One could argue that, although we adjusted for age, the imbalance in mean age between cases and controls could partly account for the difference in the prevalence of coronary artery disease. However, in the sensitivity analysis, restricted to 220 patients with stroke matched for age and gender to 220 patients without, we found the same results. One cannot exclude the fact that cardiac deaths might be responsible for a fraction of the deaths in patients with stroke, leading to some bias and overestimation of the prevalence of coronary atherosclerosis in this population. Although assessment of plaque and stenosis in extracranial and intracranial arteries was systematic in the cerebrovascular disease group, it was not in the other neurologic disease group, precluding any comparison between the 2 groups. Another limitation was the fact that coronary artery stenoses were not evaluated on pressure-fixed arteries. We also did not specify the location of stenoses in the coronary artery tree. Finally, because of the multiple testing performed, we cannot exclude the possibility that the association reported may be caused by chance or may be overestimated.

In conclusion, coronary atherosclerosis appears to be highly prevalent in patients with fatal stroke. Future work should determine the prevalence of coronary plaques and stenoses in nonfatal strokes with arterial wall imaging.

Acknowledgments
The authors are indebted to Professor Nathan Bornstein for his helpful comments on this manuscript. The authors thank Dr Sophie Rushton-Smith who provided editorial assistance in the preparation of this manuscript.

Sources of Funding
SOS-ATTAQUE CEREBRALE Association supported the work for this paper.

Disclosures
Dr Gongora received a neurovascular research scholarship from FUNSALUD and Sanofi-Synthelabo Mexico (ADEMASS) with the support of the Instituto Nacional de Neurologia y Neurocirugia in Mexico. Dr Jaramillo received honoraria for clinical research from Hospital Clinico de la Universidad Catolica in Santiago, Chile. The sponsors had no involvement in the collection, analysis, and interpretation of data; in the writing of the manuscript; and in the decision to submit the paper for publication. Dr Amareno had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

References


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Fernando Gongora-Rivera, Julien Labreuche, Arturo Jaramillo, Philippe Gabriel Steg, Jean-Jacques Hauw and Pierre Amarenco

Stroke. 2007;38:1203-1210; originally published online March 1, 2007;
doi: 10.1161/01.STR.0000260091.13729.96
Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/38/4/1203

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