Motor Strokes Sparing the Leg

Different Lesions and Causes

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Background: A considerable number of patients develop stroke without involvement of the lower limb. However, there are few reports about the motor syndrome when the leg is spared.

Objective: To study clinical findings, causative factors, and lesion topography in stroke patients with a motor deficit sparing the leg.

Patients and Methods: We studied 895 patients with paresis sparing the leg from the 3901 patients enrolled in the Lausanne Stroke Registry. They were compared with 1644 stroke patients with paresis involving the leg, by means of univariate and multivariate analysis.

Results: Eight hundred forty-four infarcts (94.3%) and 51 hemorrhages (5.7%) led to weakness sparing the leg. Different sites of lesion were found, but the majority were caused by superficial infarcts. Almost half of the lesions were confined to superficial branches of the middle cerebral artery territory, with 276 (30.8%) in the anterior (superior) and 138 (15.4%) in the posterior (inferior) middle cerebral artery. More than half of the infarcts had a presumed embolic source from large-artery disease or from the heart. In comparison with patients with paresis involving the leg, patients without leg involvement had a lower prevalence of small-artery disease ($P<.001$), but a higher prevalence of migraine ($P<.001$), transient ischemic attack ($P=.001$), atherosclerosis without stenosis ($P=.005$), large-artery disease ($P<.001$), and left hemispheric strokes ($P<.001$). They also had a lower frequency of hemorrhagic stroke.

Conclusions: Patients without leg involvement had different stroke lesions and causes and were characterized by more superficial infarcts mainly caused by emboli from large-artery disease and atherosclerosis without stenosis.

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A considerable number of patients develop stroke without involvement of the lower limb. However, there are few reports about the motor syndrome when the leg is spared. Moreover, this pattern of paresis, especially faciobrachial paresis, is attributed to ischemic lesions at different sites in the motor pathway. Although some studies had shown that faciobrachial paresis is highly suggestive of damage to the motor cortex, caused by involvement of the superficial branches of the middle cerebral artery (MCA), it is also often seen in lesions involving the complete territory of the MCA, the complete territory of the lenticulostriate arteries (striatocapsular infarct), or the territory of the lateral lenticulostriate arteries (small deep infarct of the lateral type). It also has been reported after anterior cerebral artery (ACA) infarcts.

There has been much discussion of the lesion topography when it is restricted to the corticospinal fibers, ie, pure motor stroke. The term pure motor hemiplegia was coined by Fisher and Curry to describe complete or incomplete paralysis involving the face, arm, and leg, in the absence of a sensory deficit, homonymous hemianopia, aphasia, apraxia, and agnosia. They suggested that it was caused by small-vessel occlusive disease (ie, lacunes). Subsequently, the partial syndromes of faciobrachial and brachiocrural paresis have been considered to be pure motor hemiplegia and attributed to small-artery disease. In our experience, “pure” faciobrachial paresis is frequently seen after a cortical infarct.

We performed a study of patients with stroke sparing the leg to determine (1) where the lesions causing this pattern of distribution are found, (2) the causes of these lesions, and (3) whether they differ from lesions caused by stroke affecting the leg.
PATIENTS AND METHODS

Patients were accepted for entry to the study if they met the criteria of (1) the presence of a unilateral motor deficit (paresis or plegia), (2) the deficit sparing the lower limb, ie, it involved the upper extremity and/or the face; and (3) a satisfactory level of consciousness (alertness, somnolence, or confusion; comatose patients were excluded) to allow accurate neurologic examination. We selected 895 patients from the 3901 patients enrolled from January 1, 1979, through December 31, 1997, in the Lausanne Stroke Registry, a prospective study of patients with a first stroke consecutively admitted to our center, described elsewhere.14

Diagnostic investigation included brain computed tomography (up to 3 examinations), with and without contrast (except in patients with known allergy); Doppler ultrasonography with frequency spectral analysis; 12-lead electrocardiography; and routine blood and urine tests as standard care. Magnetic resonance imaging, conventional cerebral angiography, magnetic resonance angiography, 2-dimensional transthoracic and transesophageal echocardiography, 24-hour electrocardiography (Holter), and transcranial Doppler were performed when necessary.

For all patients, the data recorded were clinical features, functional disability, and risk factors (defined according to previous guidelines14 and including hypertension, diabetes mellitus, current smoking, hypercholesterolemia, heart disease, and previous transient ischemic attack [TIA]).

The presumed causes of the ischemic strokes were classified into 6 categories: (1) potential embolic source from the heart (patients with a potential cardiac source of embolism, ie, atrial fibrillation, intracardiac thrombus or tumor, rheumatic mitral stenosis, prosthetic aortic or mitral valves, endocarditis, sick sinus syndrome, left ventricular aneurysm or akinesia after myocardial infarction, acute [<3 months previously] myocardial infarction, or global cardiac hypokinesia or dyskinesia, in the absence of another cause); (2) potential embolic source from the large arteries (patients with >50% stenosis in the appropriate internal carotid artery on a duplex scan, conventional cerebral angiography, or magnetic resonance angiography); (3) small-artery disease (patients with chronic hypertension, with a <1.5-cm infarct on computed tomography or magnetic resonance imaging, limited to the territories of the deep perforators); (4) atherosclerosis without stenosis (patients with evidence of plaques with stenosis <50% and at least 2 of the risk factors of age ≥50 years, hypertension, diabetes mellitus, cigarette smoking, or hypercholesterolemia); (5) uncommon cause (arterial dissection, fibromuscular dysplasia, angiitis, and other uncommon causes of infarcts); and (6) undetermined cause (none of the above 5 causes could be determined). For cerebral hemorrhage, 3 categories were defined: (1) hypertensive arteriopathy, (2) other causes (arteriovenous malformation, aneurysm, coagulation disorder, and other uncommon causes of hemorrhage), and (3) undetermined cause.

Infarct topography was classified according to Tatu and colleagues.15,16

Details regarding 1644 noncomatose patients with unilateral paresis or plegia involving the leg, ie, faciobrachio-crural, brachio-cranial, or isolated lower limb motor deficit, were also extracted from the Lausanne Stroke Registry for the period from January 1, 1979, through December 31, 1997, and compared with those for the above patients in terms of risk factors, lesion topography, cause, and outcome by means of Fisher exact test and the χ² test. A 2-sided P value of less than .05 was considered statistically significant. Multivariate analysis using logistic regression was carried out in patients with ischemic stroke to test the relationship of leg involvement to the various factors shown to be significant by univariate analysis. A stepwise procedure was used to successively remove the nonsignificant explanatory variables.

CHARACTERISTICS OF PATIENTS WITHOUT LEG INVOLVEMENT

Of the 895 patients selected (22.9% of the total Lausanne Stroke Registry), 570 (63.7%) were men and 325 (36.3%) were women, with a mean (±SD) age of 63.2 ± 14.4 years. Three hundred eighty-six (43.1%) had a history of hypertension, diabetes mellitus, cigarette smoking, or hypercholesterolemia; 71 (7.9%) had a history of myocardial infarction, while 76 (8.5%) had a history of atrial fibrillation, intracardiac thrombus or tumor, rheumatic mitral stenosis, prosthetic aortic or mitral valves, endocarditis, sick sinus syndrome, left ventricular aneurysm or akinesia after myocardial infarction, acute [<3 months previously] myocardial infarction, or global cardiac hypokinesia or dyskinesia, in the absence of another cause.

Clinical Features

The neurologic deficit was complete immediately or within a few minutes after the stroke in 692 patients (77.3%), or progressed in less than 1 hour in 27 (3.0%), in more than 1 hour and less than 24 hours in 62 (6.9%), and in more than 24 hours in 40 (4.5%). The deficit fluctuated in 74 patients (8.3%). The motor profile showed the following distribution: 460 patients (51.4%) had an isolated motor syndrome (with or without cortical signs), 250 (27.9%) had a sensorimotor syndrome, 62 (6.9%) had a sensorimotor syndrome accompanied by hemianopia, 31 (3.5%) had a motor syndrome with hemianopia, and 92 (10.3%) had other patterns of distribution. Faciobrachial weakness was the most common pattern of motor involvement, appearing in 652 patients (72.8%), while brachial paresis was seen in 190 (21.2%) and facial paresis in 53 (5.9%). Dysarthria was present in 14.0% of the group. Broca-type aphasia was present in 19.3% of the group, Wernicke-type aphasia in 9.9%, and global aphasia in 9.4%. Agnosia was observed in 15.8% of pa-
patients and apraxia in 29.8%. Thirty-four (3.8%) were confused and 43 (4.8%) were somnolent. Overall in-hospital mortality was 0.8%. Two hundred five patients (22.9%) had no sequelae on discharge, while 421 (47.0%), 235 (26.2%), and 27 (3.0%) had minor, moderate, and severe sequelae, respectively.

Stroke type and localization are presented in Table 1 and Table 2.

### Causes

Among the 51 patients with a hemorrhage, the presumed cause was hypertension in 26 (51.0%) and other causes in 10 (19.6%), while in 15 (29.4%) no cause was found. Of the 844 patients with infarcts, 475 (56.3%) had a presumed embolic source: 251 (52.8%) from the large arteries and 199 (41.9%) from the heart, while 23 (26.2%), and 27 (3.0%) had minor, moderate, and severe sequelae, respectively.

Our findings confirm that different brain sites of vascular lesions can lead to a motor deficit sparing the lower limb. However, most were caused by superficial infarcts (Table 1). Almost half were restricted to superficial branches of the MCA and about one third to the anterior MCA territory. This was the most common motor pattern when lesions involving superficial branches of the MCA were studied. In our study of MCA infarcts, faciobrachial paresis accounted for three quarters of all types of motor deficit after anterior MCA infarcts, and for two thirds after posterior MCA infarcts. Motor deficits are almost always present after anterior MCA lesions, whereas in posterior MCA lesions, if present, they are generally milder. In anterior MCA infarcts, precentral gyrus ischemia is usually caused by loss of blood supply by the Rolando (central sulcal) artery. In posterior MCA infarcts, the pattern is thought to be secondary involvement of motor pathways coming from the precentral gyrus in the subcortical white matter. Foix and Levy proposed that faciobrachial plegia is frequently found after complete (anterior and posterior MCA territory) MCA infarct, since motor fibers from the lower limb cortical area are affected only when the deep territory of the MCA is involved.

### Pure Motor Stroke

Even when patients with only motor deficits sparing the leg were selected (without aphasia, apraxia, agnosia, or visual field defects), the most common site of lesion was still the territory of the anterior MCA (34.5%). A potential embolic source from the heart or large arteries was the presumed cause in 48.4% of patients with cerebral infarction, while small-artery disease was the presumed cause in only 16.0% (Tables 2 and 3).

### COMPARISON BETWEEN PATIENTS WITH AND WITHOUT LEG INVOLVEMENT

Of the 1644 patients with this profile of motor deficit, the majority (1377 [83.8%]) showed faciobrachialcru-ral hemiparesis, 232 (14.1%) had brachiochromic paresis, and 35 (2.1%) had crural paresis. These patients showed a higher prevalence of hypertension than patients with deficits sparing the leg. A higher prevalence of diabetes mellitus was also observed but did not reach statistical significance (P = .07). Cerebral hemorrhages were more common in these patients. However, cigarette smoking, migraine, and the presence of TIA were less common in patients with leg involvement when cerebral infarctions were compared. The outcome, the side of stroke, its cause, and localization were also different and are summarized in Table 2, Table 3, and Table 4.

After multivariate analysis of patients with ischemic stroke, including risk factors and presumed causes, only the following variables were positively associated with a motor deficit sparing the leg: migraine (P < .001), TIA (P = .001), large-artery disease (P < .001), atherosclerosis without stenosis (P = .005), and lesions in the left hemisphere (P < .001). Small-artery disease was inversely associated with a motor deficit sparing the leg (P < .001).

### COMMENT

Our findings confirm that different brain sites of vascular lesions can lead to a motor deficit sparing the lower limb. However, most were caused by superficial infarcts (Table 1). Almost half were restricted to superficial branches of the MCA and about one third to the anterior MCA territory. This was the most common motor pattern when lesions involving superficial branches of the MCA were studied. In our study of MCA infarcts, faciobrachial paresis accounted for three quarters of all types of motor deficit after anterior MCA infarcts, and for two thirds after posterior MCA infarcts. Motor deficits are almost always present after anterior MCA lesions, whereas in posterior MCA lesions, if present, they are generally milder. In anterior MCA infarcts, precen-
fractures were the most common. Although in 1 report\(^\text{17}\) predominant leg weakness was often found to be caused by embolic hemispheric lesions, isolated leg weakness represented only 2.1% of our group of patients with leg involvement, while faciobrachiocrural hemiparesis accounted for 83.8%. Faciobrachial paresis is also probably common after some classic deep lesions, such as striatocapsular infarcts.\(^2\) However, as these infarcts are rare (1.4%-6%),\(^8\) the overall frequency of deep lesions was low (12.9%). The exact topography of the injuries in the internal capsule causing partial hemiparesis is not established. A recent revision of internal capsule anatomy showed that the traditional view of its somatotopic motor representation, with the head and eye in the genu, the upper extremity in the anterior part of the posterior limb, and the lower extremity in the posterior part of the posterior limb, is not well supported by clinical and stereotaxic stimulating studies. The review suggested that both the corticospinal and corticobulbar fibers lie in a compact bundle in the posterior part of the posterior limb.\(^19\) It is probable that the pyramidal tract does not maintain a fixed position in the internal capsule, shifting from anterior to posterior in its rostrocaudal course. Moreover, a large clinical study of lacunar infarcts in the internal capsule did not support its classic anatomical view.\(^20\)

Although ACA infarcts usually cause predominantly leg weakness, the presence of so few of these (0.7%) in the group studied was not surprising. The explanation for faciobrachial weakness caused by ACA infarcts is not known, but it may involve extension of the infarct to the lateral convexity or a concomitant infarct of the genu and/or anterior third of the internal capsule resulting from occlusion of small penetrating vessels arising from the proximal ACA. Recently, Chamorro and colleagues\(^21\) suggested that faciobrachial weakness reflects motor neglect caused by damage to medial premotor areas and not by commitment of primary motor pathways.

Usually, a PCA infarct does not cause weakness. Two hypotheses were proposed to explain hemiparesis on the basis of its topography, one being an associated lesion of the corticospinal pathways in the midbrain supplied by perforating branches of the PCA, and the other, involvement of the pyramidal tract in the internal capsule.\(^22\) In cerebellar infarcts, a motor deficit suggests associated brainstem lesion or compression of motor fibers by a mass effect caused by edema. Brainstem lesions cause typically faciobrachiofacial weakness and, more rarely, brachiofacial paresis. However, faciobrachial paresis has also been reported\(^13\) and may result from spreading of corticospinal fibers in the pons.\(^23\)

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**Table 2. Motor Pattern Related to Topography**

<table>
<thead>
<tr>
<th></th>
<th>No. (%)</th>
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<tbody>
<tr>
<td></td>
<td>Large-Artery Disease†</td>
</tr>
<tr>
<td>Leg involved</td>
<td>291 (19.8)</td>
</tr>
<tr>
<td>Leg not involved</td>
<td>251 (29.7)</td>
</tr>
<tr>
<td>Pure motor strokes, leg not involved</td>
<td>61 (27.1)</td>
</tr>
</tbody>
</table>

*Because of rounding, not all percentages sum to 100. Large-Artery Disease†, Atherosclerosis Without Stenosis§, Embolic Heart Disease§, Small-Artery Disease†, Undetermined Cause§, and Other Causes† are mutually exclusive categories. MCA indicates middle cerebral artery; ACA, anterior cerebral artery; and post, posterior.*

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**Table 3. Motor Pattern Related to Cause of Ischemic Strokes**

<table>
<thead>
<tr>
<th></th>
<th>No. (%)</th>
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<tbody>
<tr>
<td></td>
<td>MCA</td>
</tr>
<tr>
<td>Leg involved</td>
<td>100 (6.1)</td>
</tr>
<tr>
<td>Leg not involved</td>
<td>276 (30.8)</td>
</tr>
<tr>
<td>Pure motor strokes, leg not involved</td>
<td>81 (34.5)</td>
</tr>
</tbody>
</table>

*Because of rounding, not all percentages sum to 100. MCA indicates middle cerebral artery; ACA, anterior cerebral artery; and post, posterior.*

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\(^*\) P = .01 for comparison between patients with and without leg involvement by Fisher exact test.

\(^†\) P = .005 for comparison between patients with and without leg involvement by Fisher exact test.

\(^‡\) P = .005 for comparison between patients with and without leg involvement by Fisher exact test.

\(^§\) Striatocapsular and thalamus infarcts.

\(^\#\) P = .04 for comparison between patients with and without leg involvement by Fisher exact test.

\(^\|\) P = .03 for comparison between patients with and without leg involvement by Fisher exact test.

\(^\|\) P = .01 for comparison between patients with and without leg involvement by Fisher exact test.
The frequency of cerebral hemorrhages was very low (5.7%). Hemorrhages in distinct parts of the brain resulted in paresis without leg involvement. Cerebral hemorrhage can lead to hemiparesis caused by a mass effect and has less localizing value than infarcts.

Although the number of deep lesions increased when only pure motor strokes were studied, the most common site of the lesions was still the superficial anterior MCA. Many studies have considered brachiocebral or faciobrachial paresis (2 of 3 elements) as the classic presentation of small-artery disease. This was not true in our sample for faciobrachial paresis and reinforces our assumption that a lacunar cause for pure motor stroke should be applied only to faciobrachial paresis (2 of 3 elements) as the classic presentation of small-artery disease.

We found that large-artery disease and cardioembolism were the main causes of strokes sparing the lower limb. The importance of embolism would have been greater if we had included patients with atherosclerosis without stenosis, in whom embolism from the large arteries (arterioarterial embolism) was a possible cause. The frequency of strokes caused by small-artery disease (lacune) was low. In fact, risk factors for small-artery disease, such as hypertension and diabetes mellitus, were lower than in patients with leg involvement. In contrast, risk factors for large-artery disease, such as cigarette smoking, were higher. The higher frequency of TIA could be explained by the increased frequency of large-artery disease and atherosclerosis without stenosis in this group. The frequency of cardioembolism was the same in the 2 groups.

Migraine frequency was almost double in patients without leg involvement compared with patients with leg involvement (Table 4). Although the relationship between migraine and stroke is disputed, recent epidemiological studies suggest that it is an independent risk factor for strokes in both men and women. Patent foramen ovale, mitral valve prolapse, arterial dissection, and other causes have been incriminated, but the mechanism of stroke is not clear. In our study, the frequency of mitral valve prolapse, patent foramen ovale, and arterial dissection did not differ between the 2 groups. Our findings allow us to speculate that migraine is more probably related to involvement of superficial vessels (by embolus, vasospasm, or local abnormalities) than deep perforating vessels.

Patients without leg involvement had a higher frequency of left hemispheric strokes (Table 4). The same was true for hemorrhage, but the result was not statistically significant, possibly because of the small number of patients with this type of stroke. Left hemispheric strokes are usually accompanied by language problems, whereas cortical signs of right hemispheric lesions are milder. It can be assumed that fewer patients with right hemispheric lesions seek medical attention, especially when the motor deficits are discrete, sparing the lower limb. In fact, in a study by Norrving and Bogousslavsky, silent infarcts were more often localized to the right hemisphere. We could speculate that there is a difference in motor representation between the two hemispheres. Mohr and coworkers studied the relationship between convexity infarct and motor patterns of hemiparesis and found that the size of the infarct did not vary between the two sides, but that the location of the lesion differed; a difference in the weakness syndrome for the two sides was suggested. Another hypothesis is that, because of the asymmetry of the origin of the large vessels from the aortic arch, cardiac emboli may preferentially lodge in the left MCA. Some studies have reported a higher incidence of left MCA involvement in cardioembolic stroke. Since many of our patients were enrolled at the beginning of the Lausanne Stroke Registry and did not undergo transesophageal echocardiography, the incidence of cardioembolism may be underestimated. One study has suggested that cardioembolic stroke may be more common than has been claimed. We therefore speculate that underestimation of the frequency of cardioembolic stroke in the group of patients without leg involvement could explain the left-right asymmetry.

When selected types of infarcts were studied, faciobrachial paresis was a sign of a better prognosis. We found that short-term functional outcome was better in patients without leg involvement than in patients with leg involvement. This result might be explained by the fact that most scales available to measure outcome obviously take into account walking as one of the variables. Moreover, fewer complete MCA infarcts were present in this group and, after exclusion of these infarcts from the analysis, mortality was low and equal in the two groups.

In summary, our findings suggest that stroke patients in whom the motor deficit spares the leg have (1) a higher frequency of superficial infarcts, (2) increased embolism (because of large-artery disease and atherosclerosis without stenosis), (3) a lower frequency of hemorrhages (because of the increased frequency of small-artery disease and atherosclerosis without stenosis), (4) a lower frequency of cardioembolism, and (5) a higher frequency of left MCA involvement.
orrhage, and (4) more frequent left hemispheric damage than patients with a deficit involving the leg.

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REFERENCES