Pure Monoparesis
A Particular Stroke Subgroup?
Malin Maeder-Ingvar, MD; Gus van Melle, PhD; Julien Bogousslavsky, MD

Background: Acute stroke presenting as monoparesis is rare, with a pure motor deficit in the arm or leg extending to an isolated facial paresis.

Objective: To raise the question if acute stroke presenting as monoparesis is a different entity from stroke with a more extensive motor deficit.

Patients: In the Lausanne Stroke Registry (1979-2000), 195 (4.1%) of 4802 patients met the clinical criteria for pure monoparesis involving the face (22%), arm (63%), or leg (15%).

Results: In the vast majority of cases (>95%), monoparesis corresponded to ischemic stroke with a favorable outcome, with initial computed tomography scans or magnetic resonance images showing no signs of hemorrhage. The lesion for a facial deficit was most frequently located subcortically (internal capsule); for an arm deficit, in the superficial middle cerebral artery; and for a leg deficit, in the anterior cerebral artery territory. In pure monoparesis, only 17% of the patients had more than 1 risk factor as compared with 26% of those with bimodal and trimodal hemiparesis and with 46% of all patients with stroke other than those with pure motor stroke. The only frequent risk factor was hypertension (53%); however, this frequency was no different from that in other patients with stroke. No major stroke etiology could be identified in any of the 3 subgroups of monoparesis.

Conclusion: Our finding of a wide range of stroke localization and etiology in monoparesis without any particular subgroup suggests that no specific plan of investigation can be recommended for these patients.

Arch Neurol. 2005;62:1221-1224

METHODS
We studied all patients admitted to our neurology department between 1979 and 2000 who met the following criteria by having (1) a first acute stroke (ischemic or hemorrhagic), when hospitalized and examined by a senior resident and staff neurologist; (2) a motor deficit in a single part of the body (face, arm, or leg); (3) no sensory deficit (taking into account the history, subjective complaints, clinical examination of superficial deficits and pain, proprioception, vibration deficits, central sensory deficits, and such cortical sensory deficits as neglect); and (4) no visual field deficit (visual confrontation), all of whom have been included in the prospective Lausanne Stroke Registry. The work-up included computed tomography (CT) and magnetic resonance imaging (MRI) in selective cases, ultrasonography, electrocardiogram (ECG), and a standard blood test. Angiography (MRIs with angiographic sequences or conventional intraarterial arteriography) and echocardiography were performed on selected patients. The vas-
A significant difference was seen, with facial deficit being most frequently located subcortically, arm deficit located in the superficial middle cerebral artery, and leg deficit located in the anterior cerebral artery ($P < .001$).

Table 2. Comparison of Individual Risk Factors in Patients With Pure Motor Stroke and Other Patients With Stroke, Showing a Significant Difference Only in the Presence of Arterial Claudication ($P = .003$)

<table>
<thead>
<tr>
<th>Risk Factors</th>
<th>Monoparesis, %</th>
<th>Other Patients With Stroke, %</th>
<th>$P$ Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertension</td>
<td>53</td>
<td>51</td>
<td>&gt; .80</td>
</tr>
<tr>
<td>Diabetes</td>
<td>15</td>
<td>14</td>
<td>&gt; .80</td>
</tr>
<tr>
<td>Hyperglycemia</td>
<td>3</td>
<td>4</td>
<td>&gt; 5.0</td>
</tr>
<tr>
<td>Tobacco consumption</td>
<td>25</td>
<td>29</td>
<td>&gt; 10</td>
</tr>
<tr>
<td>Hypercholesterolemia</td>
<td>22</td>
<td>27</td>
<td>&gt; 10</td>
</tr>
<tr>
<td>Family history of stroke or heart infarction</td>
<td>7</td>
<td>8</td>
<td>&gt; .70</td>
</tr>
<tr>
<td>Ischemic heart disease</td>
<td>23</td>
<td>20</td>
<td>&gt; .38</td>
</tr>
<tr>
<td>Arterial claudication</td>
<td>10</td>
<td>5</td>
<td>.003</td>
</tr>
<tr>
<td>History of arrhythmia</td>
<td>9</td>
<td>13</td>
<td>.15</td>
</tr>
</tbody>
</table>

In the pure monoparesis group, only 17% of the patients had more than 1 risk factor compared with 26% in the bimodal and trimodal groups and compared with 46% in all patients with stroke except for those with pure motor stroke ($P < .001$). Comparing individual risk factors in patients with pure motor stroke showed a significant difference only in the presence of arterial claudication ($P = .003$, Table 2).

Preceding transitory symptoms were present in 16% of patients compared with 14% of other patients with stroke ($P = .02$) and headache in 8% compared with 20% of other patients with stroke ($P < .001$).

### RISK FACTORS

In the pure monoparesis group, only 17% of the patients had more than 1 risk factor compared with 26% in the bimodal and trimodal groups and compared with 46% in all patients with stroke except for those with pure motor stroke ($P < .001$). Comparing individual risk factors in patients with pure motor stroke showed a significant difference only in the presence of arterial claudication ($P = .003$, Table 2).

Echocardiography was performed in the acute phase (during hospitalization) for 37% of the pure monoparesis group, the same frequency as for the other patients with stroke ($P = .60$). In this group of patients, the echocardiogram was normal in 15% of patients or showed an akinetic segment in 6% of patients, nonembolic abnormalities in 6%, embolic abnormalities (cardiac thrombus, fibroelastoma) in 5%, a permeable foramen ovale in 4%, or other abnormalities in 2%. Compared with the other patients with stroke, there was no difference in the distribution of cardiac abnormalities.

### INVESTIGATION

Cerebral imaging was initially performed by CT scan for 158 patients; it showed lesions in 104 patients and no lesions in 54 patients. In the other 37 patients, first imaging by CT scan was negative or an initial examination by MRI showed a vascular lesion (ischemic or hemorrhagic) in 31 patients and no structural abnormality in 6 patients. The overall distribution of neurosonologic findings in patients with monoparesis differed from that in the other patients with stroke ($P < .001$). Neurosonologic neck ipsilateral examination (obtained in all patients) showed internal carotid plaque with calcifications without stenosis (less than 50%) in 36% of the patients and no carotid abnormalities in 34%. These 2 groups with no or minor carotid plaque formation ac-

### GENERAL DESCRIPTION OF THE GROUP WITH MONOPARESIS

One hundred ninety-five (4.1%) of 4802 patients fulfilled the criteria of pure motor deficit in the face, arm, or leg without sensory deficit. The sex distribution was 61% male and 39% female, the same as in the other patients in the registry ($P > .90$). The mean ± SD age was 66 ± 14 years, with a range of 22 to 89 years, the group being slightly older than the other patients with stroke ($P = .07$).

The clinical presentation was facial weakness in 42 patients (22%), arm paresis in 123 (63%), and leg paresis in 30 (15%). A significant difference was seen, with facial deficit most frequently located subcortically, arm deficit located in the superficial MCA, and leg deficit located in the anterior cerebral artery ($ACA; P < .001$; Table 1).

Preceding transitory symptoms were present in 16% of patients compared with 14% of other patients with stroke ($P = .02$) and headache in 8% compared with 20% of other patients with stroke ($P < .001$).
counted for 70% of the group compared with 60% in the rest of the stroke population (P = .04). Abnormalities in the vertebrobasilar system were rare in monoparesis (4% vs 12% in the other patients with stroke; P = .001). The investigation was completed by an angiography (MRI with angiographic sequences or conventional intra-arterial arteriography) in 28% of the patients, the same frequency as in the remaining stroke group (P = .30).

**VASCULAR TERRITORY**

The vascular territory of the stroke in patients with pure monoparesis was identified as superficial MCA in 48%, subcortical (anterior lenticulostriate) in 31%, brainstem in 8%, and ACA in 8%. In the remaining 10 patients, the vascular lesions were spread between other territories (Table 1).

In terms of topographic localization, there was a significantly different distribution between the monoparesis subgroups (P < .001), with facial deficits being most frequently located subcortically, arm deficits located in the superficial MCA, and leg deficits located in the ACA (Table 1).

**PRESUMED ETIOLOGY OF STROKE**

There was no difference in etiologies between the different monoparesis groups (P = .11; data not shown). In the group with no identified lesion, no particular etiology was found. However, when patients with monoparesis were compared with those with more extensive motor deficit (bimodal distribution in the face and arm or leg and arm or complete hemiparesis), the frequency of hypertension was significantly different (P < .001), mainly because of the higher incidence in the complete hemiparesis group (data not shown).

In terms of the vascular territories involved, monomodal and bimodal deficits were very similar whereas complete hemiparesis was predominantly located in the subcortical regions (centrum ovale or internal capsule; **Table 3**). The vascular territories involved in the different groups of motor presentation were not significantly affected by the presence or absence of hypertension.

**FUNCTIONAL OUTCOME**

Our analysis of patients leaving the hospital, using a modified Rankin scale, indicated that 41% of the patients with monoparesis returned to their former activities, 41% required some assistance, 16% were partially dependent, and 2% were completely dependent. One percent of patients died during hospitalization.

Comparison of the different groups of motor deficit showed a significant difference in outcome (P < .001), mainly due to the favorable outcome for the monoparesis group (and a trend to a worse outcome with increased extent of the lesion; data not shown).

---

**Table 3. Comparison of the Distribution of Involved Vascular Territories in Patients With Monomodal or Bimodal Deficits or Complete Hemiparesis**

<table>
<thead>
<tr>
<th>Vascular Territory</th>
<th>Monomodal, %</th>
<th>Bimodal, %</th>
<th>Complete, %</th>
<th>Others, %</th>
<th>Total, No.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Middle cerebral artery</td>
<td>48</td>
<td>56</td>
<td>14</td>
<td>37</td>
<td>1759</td>
</tr>
<tr>
<td>Subcortical</td>
<td>31</td>
<td>26</td>
<td>60</td>
<td>24</td>
<td>1374</td>
</tr>
<tr>
<td>Brainstem</td>
<td>9</td>
<td>6</td>
<td>13</td>
<td>16</td>
<td>698</td>
</tr>
<tr>
<td>Anterior cerebral artery</td>
<td>8</td>
<td>4</td>
<td>2</td>
<td>1</td>
<td>99</td>
</tr>
<tr>
<td>Other</td>
<td>5</td>
<td>8</td>
<td>11</td>
<td>21</td>
<td>872</td>
</tr>
<tr>
<td><strong>Totals, No. (%)</strong></td>
<td><strong>195 (100)</strong></td>
<td><strong>493 (100)</strong></td>
<td><strong>544 (100)</strong></td>
<td><strong>3570 (100)</strong></td>
<td><strong>4802</strong></td>
</tr>
</tbody>
</table>

Abbreviations: Bimodal, motor deficit in the face and arm or leg and arm; Complete, hemiparesis with facial involvement; Monomodal, motor deficit in the face, arm, or leg.

*Monomodal and bimodal deficits have very similar distributions of the vascular territories whereas complete hemiparesis showed involvement mainly of the subcortical regions.
period of time (1979-2000). The only study of a large collection of patients with monoparesis (35/4818) over a 5-year period was recently published⁴ with a clinical presentation restricted to distal arm motor paresis showing a close correlation of arm monoparesis to small cortical lesions in the MCA territory with MRI and diffusion-weighted imaging. Their results correlate with our study showing first a preponderance of MCA lesions in arm monoparesis (Table 1) and second the finding that no particular vascular etiology was found in this group of patients with stroke.

However, the weakness in our study could be that the prospective inclusion in the Lausanne Stroke Registry was rather long (1979-2000). Although the patients were all hospitalized and investigated in our neurology department, one must consider the evolution of the noninvasive imaging techniques (CT scans with high definition, MRIs with angiographic sequences and diffusion-weighted imaging). The fact that only 37% of the patients underwent an echocardiography during hospitalization correlates to earlier data⁹ showing that if there were no history or clinical indication for a cardioembolic source, echocardiography performed after the hospitalization in the majority of the cases did not give further information.

The results showing a significant increase in subcortical lesions with facial weakness and in MCA lesions with arm deficits and a significant preponderance of ACA localization with leg deficits have all been reported⁸-¹³ but only recently in a prospective stroke registry regarding a restricted entity that is distal monoparesis of the arm.¹

Stroke in the vast majority of patients with monoparesis was ischemic, hemorrhagic lesions being found in only 3% of patients (6/195), in agreement with a single report in the literature of 1 case with pure motor monoparesis due to intracerebral hemorrhage.¹⁹ No particular etiology (small or large vessel disease, positive cardioembolic source, or other) was found in any monoparesis subgroup.

More than 1 risk factor was seen in only 17% of patients with pure monoparesis, significantly less (P<.001) than the 26% in the bimodal or trimodal group and the 46% in the remaining patients with stroke (data not shown).

The only frequent risk factor was hypertension,⁸ which was present in 53% of patients with monoparesis, a frequency not significantly different from that in the other patients with stroke. However, the presence or absence of hypertension in the 3 monoparesis groups did not alter the etiology of the stroke.

No particular etiology was identified for the 3 different types of monoparesis (data not shown). This was probably due to the rather small number of patients in each subgroup.

In our study, the functional outcome was generally very good with a spontaneous favorable evolution, allowing the patient to return to former activities (41%) or resulting in a slight handicap requiring some assistance (41%). When comparing monoparesis with more extensive motor deficit, there was a significant difference in outcome, which was mainly due to the favorable outcome in monoparesis (data not shown).

We conclude that the presentation of monoparesis in patients with acute stroke is rare, being found in only 4.1% of 4802 patients in a 21-year registry. Facial deficits were most frequently associated with subcortical lesions, arm deficits with MCA lesions, and leg deficits with ACA lesions. No special risk factor or cause could be associated with any of the monoparesis subgroups. The outcome was generally favorable. These results show that investigations in patients with monoparesis need to consider the same range of etiologies as in patients with stroke in general.

Accepted for Publication: March 1, 2005.
Correspondence: Malin Maeder-Ingvar, MD, Department of Neurology, BHo7, University Hospital of Vaud, Rue du Bugnon 46, 1011 Lausanne, Switzerland (Malin.Maeder-Ingvar@chuv.hospvd.ch).
Author Contributions: Study concept and design: Maeder-Ingvar and Bogousslavsky. Acquisition of data: Maeder-Ingvar and van Melle. Drafting of the manuscript: Maeder-Ingvar. Critical revision of the manuscript for important intellectual content: Maeder-Ingvar, van Melle, and Bogousslavsky. Statistical analysis: van Melle. Obtained funding: Maeder-Ingvar and Bogousslavsky. Administrative, technical, and material support: Maeder-Ingvar. Study supervision: Bogousslavsky.

REFERENCES