The results indicate that MDL 72.974 A potentiates the effects of I individual doses of levodopa. Due to its prolonged symptomatic action the drug may improve motor fluctuations and improve night-time and early morning akinesia in patients with Parkinson's disease. Whether this potent and selective inhibitor of MAO-B reduces the progression of the underlying Parkinsonism by protective mechanisms is still to be investigated.

MDL 72.974 A was kindly provided by Marion Merrell Dow product development, Strasbourg, France.

R MARCONI, M BONNET, Y AGID
Laboratoire de M é dicine Expérimentale, INSERM U289, Hôpital de la Salpêtrière, 75651 PARIS Cédex 13, France

5 Esteguy M, Bonnet AM, Lhermitte F, Agid Y. "Le test a la L-Dopa" dans la maladie de Parkinson. Rev Neurol (Paris) 1985; 141:413-5.

Sensorimotor syndrome relates to lacunar rather than to non-lacunar cerebral infarction

In a recent study in this journal, Landi et al found similar clinical and CT scan features in patients with sensorimotor and those with lacunar stroke, as opposed to non-lacunar stroke patients. Their data favour the view that sensorimotor stroke is commonly due to lacunar infarction.

Prompted by this study we compared stroke patients with sensorimotor syndrome (N = 47) with those with other lacunar syndromes (pure motor, pure sensory, dysarthria-clumsy hand, and ataxic hemiparesis syndrome) (N = 99), and also with a group of patients with a cortical syndrome (N = 214), who were registered in a prospective database of all patients with a first, supratentorial brain infarct, as described elsewhere. At the time this analysis 347 patients were registered. Thirteen patients with a cause other than atherothrombotic cardiac or thromboembolism, and one with unknown initial stroke syndrome were excluded. For definitions and diagnostic criteria of the different stroke subgroups, we refer to a previous report.

Isolated monoparesis was taken as a cortical syndrome because it is generally caused by superficial infarction.

Striatocapsular infarcts were classified as "cortical" because they resemble such infarcts with their clinical presentation and pathogenesis. We compared the frequency of both potential cardiac and cortical (> 50% diameter reduction on non-invasive testing) sources of embolism between sensorimotor syndrome and both lacunar and cortical syndromes. We studied the number of patients with severe initial handicap (a score of 5 on the Rankin scale) and the one month case fatality rate, and the frequency of lacunar and non-lacunar infarcts on CT scan.

All except 11 cortical syndrome patients had at least one CT. Further three of the sensorimotor syndrome, 81 of the other lacunar syndrome group, and 139 of the cortical syndrome patients had non-invasive carotid testing.

The results are shown in the table. Differences were analysed by means of odds ratios (OR) with (Yates' corrected) 95% confidence interval (CI); differences in age were compared by two tailed Mann-Whitney U test.

Patients with sensorimotor syndrome had similar age distribution as those with lacunar syndrome, whereas they differed from those with cortical syndrome, who were older. The frequencies of both potential cardiac and cortical embolic source were equally low in sensorimotor and lacunar syndromes, but higher in cortical syndrome, suggesting that sensorimotor like lacunar syndrome most likely result from small vessel disease rather than from cardiac or carotid embolism. Sensorimotor syndrome resembled lacunar syndrome in a low number of severe strokes, a low early one month case fatality rate, and a low number of non-lacunar (mostly involving the cortex) infarcts on CT, whereas they differed significantly from cortical syndrome. There were no differences between the sensorimotor syndrome patients with, and those without a CT lesion. The group with a lesion contained more cases with a potential cardiobolic stroke cause, and also with a Rankin score of 5, than those without a lesion. These differences were not statistically significant but do not suggest that sensorimotor syndrome without a CT lesion would be more likely to resemble cortical syndrome.

Our results agree with those of Landi et al and suggest that sensorimotor syndrome should be regarded as a lacunar syndrome, which means that it usually results from lacunar infarction due to cerebral small vessel disease, rather than non-lacunar infarction due to cardiac or carotid embolism. This conclusion is particularly important in patients with normal CT scans, for whom extensive cardiac investigation and angiography aimed at detecting operable carotid lesions are of dubious value, as with a CT proven lacunar infarct. Taking accurate patient histories and performing a neurological examination, to differentiate between lacunar and cortical stroke syndrome therefore remain important in determining management options.

Correspondence to: Dr Lodder, Department of Neurology, University Hospital Maastricht, PO Box 5800, 6202 AZ Maastricht, The Netherlands.


Table: Features of patients with sensorimotor syndrome compared with lacunar syndrome patients and cortical syndrome patients

<table>
<thead>
<tr>
<th>Sensorimotor syndrome N (%)</th>
<th>Lacunar syndrome N (%)</th>
<th>OR 95%CI</th>
<th>p-value</th>
<th>Cortical syndrome N (%)</th>
<th>OR 95%CI</th>
<th>p-value (two-tailed)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td>47</td>
<td>99</td>
<td></td>
<td>214</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male/female</td>
<td>22/25</td>
<td>58/41</td>
<td></td>
<td>NS</td>
<td>115/99</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Mean age (SD)</td>
<td>65.9 (10.4)</td>
<td>67.3 (11.7)</td>
<td></td>
<td>4.66 (0.22-9.84)</td>
<td>80 (37)</td>
<td>0.01-5 (0.00-60)</td>
</tr>
<tr>
<td>Carotid embolism</td>
<td>6 (13)</td>
<td>9 (9)</td>
<td></td>
<td>NS</td>
<td>46 (33)</td>
<td>0.05 (0.01-23)</td>
</tr>
<tr>
<td>Carotid sten &gt;50%</td>
<td>1 (2)</td>
<td>12 (15)</td>
<td>0.01 (0.02-112)</td>
<td>NS</td>
<td>36 (43)</td>
<td>0.05 (0.01-23)</td>
</tr>
<tr>
<td>Rankin score 5</td>
<td>6 (13)</td>
<td>10 (10)</td>
<td></td>
<td>NS</td>
<td>94 (29)</td>
<td>0.09 (0.08-44)</td>
</tr>
<tr>
<td>30-day mortality</td>
<td>6 (13)</td>
<td>12 (15)</td>
<td></td>
<td>NS</td>
<td>36 (43)</td>
<td>0.05 (0.01-23)</td>
</tr>
<tr>
<td>Lacunar infarct on CT</td>
<td>50 (152)</td>
<td>0.70 (0.30-1.63)</td>
<td></td>
<td>19 (9)</td>
<td>3.27 (1.48-7.26)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Non-lacunar infarct on CT</td>
<td>5 (11)</td>
<td>18 (18)</td>
<td>0.54 (0.14-2.01)</td>
<td>NS</td>
<td>145 (71)</td>
<td>0.05 (0.02-111)</td>
</tr>
</tbody>
</table>
Sensorimotor syndrome relates to lacunar rather than to non-lacunar cerebral infarction.

J Lodder, J Boiten and L Heuts-Van Raak

*J Neurol Neurosurg Psychiatry* 1992 55: 1097
doi: 10.1136/jnnp.55.11.1097

Updated information and services can be found at:
http://jnnp.bmj.com/content/55/11/1097.citation

**Email alerting service**

*These include:*

Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

**Notes**

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/