Original Contributions

Prognosis in Middle Cerebral Artery Stenosis

R. N. Corston, M.D., B. E. Kendall, F.R.C.R., and John Marshall, M.D.

SUMMARY Among 21 patients with angiographically demonstrated stenosis of the middle cerebral artery (MCA) 14 presented with a stroke and 7 with transient ischaemic attacks (TIAs). Of the former 4 had further strokes (3 fatal); of the latter one had a fatal stroke. Only 2 of the 21 patients had TIAs during the follow-up period. The present study indicates that the prognosis for patients with MCA stenosis is less good than the literature suggests. There is appreciable risk of fatal stroke especially in those who present with a stroke. This perspective is important when deciding on the need for EC/IC bypass.

MIDDLE CEREBRAL ARTERY STENOSIS (MCA) is an uncommon lesion occurring in only 7.6% of 4748 patients in the Joint Study of Extracranial Arterial Occlusion.1 In studies of MCA occlusion, stenosis is mentioned in only 2-28% of patients in the various series.2-4 One recent exception, however, reported no less than 58% of stenoses in a series of 26 cases of MCA lesions.7 In studies of all ischaemic accidents in the MCA territory, MCA stenosis was responsible in only 2% of cases.8,9

The terminology in the literature is confused, the term occlusion being used for both complete and partial obstruction of the middle cerebral artery with or without a qualifying epithet such as “partial” or “complete.” In this paper the term stenosis will be applied to a partial obstruction of the lumen and the unqualified term occlusion to complete obstruction.

In recent years the operation of extracranial-intracranial (EC-IC) artery anastomosis has been used for patients with MCA stenosis yet there is little data on the natural history of this condition against which to assess the benefit of the operation. Lascelles and Burrows1 had 9 patients with MCA stenosis in their study of MCA occlusion and of these 5 (56%) made a complete or almost complete recovery whereas only 4 of their 22 patients (18%) with occlusion of the trunk of the artery had a similar prognosis. In Allcock’s series2 of 5 of 11 patients (45%) with MCA stenosis had a good prognosis as compared with only 8 of the 29 patients (28%) with occlusions of the artery. There appears to be only one study in the literature of MCA stenosis alone, freedom from further cerebrovascular incidents being reported in 13 of 16 patients during a follow-up to a maximum of 6 years.10 The present study was undertaken with a view to providing further data on the long-term prognosis of MCA stenosis against which EC/IC bypass can be evaluated.

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Patients and Methods

Twenty-one patients were identified from the carotid angiogram reports at the National Hospitals for Nervous Diseases, Queen Square and Maida Vale as having MCA stenosis; 19 had stenosis of the trunk and 2 of branches of the artery. The angiograms were performed between January 1957 and December 1982; 15 were unilateral and 6 bilateral studies. Patients with severe ipsilateral carotid artery disease had been excluded. None had undergone an EC/IC bypass procedure.

Fourteen angiograms were still available for review. They were examined for the site and degree of stenosis, for evidence of abnormal flow distal to the lesion and for the position of the border zone between middle and anterior or posterior cerebral arteries. Site of stenosis was determined according to Allcock’s scheme2 in which lesions at the origin of the artery were designated type ‘A,’ those involving the trunk of the artery proximal to the first angiographically visible cortical branch, type ‘B’ and those distal to this branch type ‘C’ (fig. 1). The criteria of Hinton et al10 for the haemodynamic significance of the stenosis were used, namely the presence of delayed filling of the middle cerebral vessels, a border zone shift, or both.

All 21 cases were successfully traced; 18 had been re-examined at various times and for 3 the patient or relative responded to a postal questionnaire. Ten had died; data about the cause of death was obtained from hospital notes in 5 cases, of whom 2 had post mortem examinations, and from the death certificates of a further 4 patients. The cause and date of death was not determined for one patient.

Results

Clinical Findings

The age at onset is shown in table 1 and clinical details in table 2. There were 18 males and 3 females with ages ranging from 19-68 years (mean 51 years). Follow-up was for a mean of 6 years 10 months (range 3 months to 25 years).

Presentation

Fourteen patients (67%) presented with a stroke, 8 of whom had had prior transient ischaemic attacks...
FIGURE I. Site of lesion: A = origin; B = from origin to first
cortical branch; C = from first cortical branch to bifurcation
or trifurcation.

(TIAs). In 3 the stroke had caused a severe hemiplegia
and in one a severe dysphasia with mild involvement
of the limbs; in the remaining 10 patients the disability
was minimal. Seven patients (33%) presented with
TIAs alone. The left hemisphere was affected in 14
patients and the right in 7. Nine patients had a diastolic
blood pressure of 100 mm Hg or more.

Follow-up

One of the 7 patients who presented with TIAs had a
stroke immediately following angiography. Of the re-
maining 6 patients one continued to have TIAs for four
months but thereafter remained well until his death
from myocardial infarction seven years later; a second
patient died of myocardial infarction after a trouble-
free interval of 15 months. A third patient kept well for
nine years before dying of a stroke. The remaining 3
patients experienced no further cerebrovascular epi-
sodes over periods of 17 months to 14 years; one of
these died after eight years of carcinoma bronchus.

Among the 14 patients who presented with a stroke
there were 4 in whom the initial disability was severe,
3 from hemiplegia and one from dysphasia. Three never-
evertheless made a good recovery being left with mini-
mal disability and having no further episodes by four,
six and nine years. The fourth patient fared badly; he
made little recovery from the first stroke, experienced
a second at two years and died from a third at five
years.

Of the 10 patients whose initial stroke was mild, 3
had further strokes at six months, four and a half years
and 24 years respectively; the first two of these proved
fatal. The remaining 7 patients included one who con-
tinued to have TIAs (he had experienced TIAs before
his mild stroke) despite being anticoagulated with war-
farin. After a year the attacks ceased and after four
years the drug was replaced by aspirin. He remained
well at nine years. The other 6 patients were trouble-
free over periods ranging from three months to 15
years. Two of the 6 died of carcinoma at four and eight
years.

Deaths

To summarize the deaths, 10 patients died during
the follow-up period. In 4 death was caused by a
stroke, 3 of these being the result of an infarct on the
side of the MCA stenosis; one patient developed a
pseudo bulbar palsy with pneumonia. The interval
from presentation to death ranged from six months to
nine years. Two patients died of myocardial infarction
after one and seven years respectively. There were 3
deaths from cancer and in one case the cause of death
was not known.

Transient Ischaemic Attacks

The TIAs before and after presentation took many
forms. Purely sensory symptoms were present in 2
patients; they only occurred in TIAs of the non-domi-
nant hemisphere. In one patient the hand, tongue and
mouth were affected and in one the hand alone. Motor
symptoms were present in 8 and consisted of: hemipa-
resis in 4 with associated dysarthria in one, weakness
of one hand or arm in 3 plus dysphasia or dysarthria in
one case each. Dysphasia occurred in a total of 4 pa-
tients (50% of patients with dominant hemisphere
TIAs) and was the sole manifestation in 3. One patient
had one episode of amaurosis fugax.

Angiography

The angiograms were available for review in 14 of
the 21 patients. Details are given in table 3. The main
stem of the artery was involved in 12 patients. Of the
other 2 one had a stenosis of the angular branch and
one of the ascending frontoparietal branch. The degree
of stenosis varied from 33–66% and the length of the
stenosis for the trunk lesion was 7.1 mm (range 3.5–25
mm). The lesion was proximal to the first angiographi-
cally visible branch in 4, was distal to this branch in 7
and involved both segments in one. Stenoses of other
arteries occurred in 6 patients. In 4 there was mild
stenosis of the ipsilateral internal carotid which affect-
ed the proximal region in 3 and termination in one
artery. In one there was also mild stenosis of the ipsi-
lateral external carotid artery. Anterior cerebral artery
stenosis ipsilateral to the MCA lesion was present in 2
and in one it was present in the contralateral artery.
Stenosis of the pericallosal artery was found in one
patient.

<table>
<thead>
<tr>
<th>Table 1: Age at Onset</th>
</tr>
</thead>
<tbody>
<tr>
<td>Years</td>
</tr>
<tr>
<td>11–20</td>
</tr>
<tr>
<td>21–30</td>
</tr>
<tr>
<td>31–40</td>
</tr>
<tr>
<td>41–50</td>
</tr>
<tr>
<td>51–60</td>
</tr>
<tr>
<td>61–70</td>
</tr>
</tbody>
</table>
Haemodynamically significant lesions, as shown by border zone shift or delayed flow, were present in 6 patients. The stenosis in both the haemodynamically significant and non-significant lesions varied between 33 and 66%. Four patients had evidence of distal embolization.

Prognosis was examined in relation to two angiographic features: the site of the stenosis and whether it was haemodynamically significant. Two of the 4 patients in whom the stenosis was proximal to the first angiographically visible branch died of a stroke against 2 of the 7 in whom it was distal to this point. Likewise 2 of the 6 haemodynamically significant lesions were associated with a stroke during follow-up as were 2 of the 8 non-significant lesions.

Discussion
With one exception\(^9\) previous studies of the prognosis in MCA lesions have included both stenosis and occlusion. There is, however, evidence that intracranial arterial disease is rarely the cause of MCA occlusion; it results rather from extracranial pathological processes with embolism being a major cause.\(^8, 9, 11\) Since MCA stenosis and occlusion may result from different pathological processes it is essential to study MCA stenosis alone.

The results of the present study indicate the prognosis is less good than was suggested by Hinton et al.\(^10\) In

Table 2 Clinical Features of Patients with Middle Cerebral Artery Stenosis

<table>
<thead>
<tr>
<th>No</th>
<th>Age</th>
<th>Sex</th>
<th>Blood pressure (mm Hg)</th>
<th>TIA</th>
<th>Stroke</th>
<th>Duration</th>
<th>Therapy</th>
<th>Events</th>
<th>Cause of death</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>61</td>
<td>F</td>
<td>190/120</td>
<td></td>
<td>mild hemiparesis &amp; dysphasia</td>
<td>8 yr 5 mo</td>
<td>—</td>
<td>—</td>
<td>carcinoma</td>
</tr>
<tr>
<td>2</td>
<td>58</td>
<td>M</td>
<td>145/85</td>
<td>3 mo</td>
<td>—</td>
<td>17 mo</td>
<td>warfarin 17 mo</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>3</td>
<td>53</td>
<td>M</td>
<td>130/90</td>
<td>mild dysphasia</td>
<td>—</td>
<td>6 mo</td>
<td>aspirin 6 mo</td>
<td>—</td>
<td>unknown</td>
</tr>
<tr>
<td>4</td>
<td>21</td>
<td>F</td>
<td>120/80</td>
<td>2 in 24 hrs</td>
<td>severe hemiplegia &amp; dysphasia</td>
<td>4 yr</td>
<td>warfarin 16 mo</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>5</td>
<td>68</td>
<td>M</td>
<td>160/100</td>
<td>1 mo</td>
<td>mild hemiparesis &amp; dysphasia</td>
<td>6 mo</td>
<td>—</td>
<td>—</td>
<td>cerebral infarct</td>
</tr>
<tr>
<td>6</td>
<td>60</td>
<td>M</td>
<td>140/90</td>
<td>Many over 1 mo</td>
<td>—</td>
<td>7 yr 9 mo</td>
<td>warfarin 18 mo</td>
<td>subdural hematoma 18 mo</td>
<td>—</td>
</tr>
<tr>
<td>7</td>
<td>63</td>
<td>M</td>
<td>165/85</td>
<td>2 mo</td>
<td>—</td>
<td>6 mo</td>
<td>—</td>
<td>stroke at angiography</td>
<td>—</td>
</tr>
<tr>
<td>8</td>
<td>46</td>
<td>M</td>
<td>160/100</td>
<td>3 mo</td>
<td>mild hemiparesis</td>
<td>9 yr 9 mo</td>
<td>warfarin 4 yr</td>
<td>aspirin 5 y 9 mo</td>
<td>TIA's for 1 yr</td>
</tr>
<tr>
<td>9</td>
<td>30</td>
<td>M</td>
<td>130/80</td>
<td>2 in 24 hrs</td>
<td>—</td>
<td>14 yr 3 mo</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>10</td>
<td>54</td>
<td>M</td>
<td>150/90</td>
<td>1 in 3 days</td>
<td>severe hemiplegia</td>
<td>6 yr 4 mo</td>
<td>heparin for 10 days</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>11</td>
<td>42</td>
<td>F</td>
<td>200/120</td>
<td>Many over 15 yr</td>
<td>mild hemiparesis &amp; dysphasia</td>
<td>25 yr</td>
<td>warfarin</td>
<td>3 yr 9 mo</td>
<td>series of strokes after 24 yr</td>
</tr>
<tr>
<td>12</td>
<td>36</td>
<td>M</td>
<td>160/105</td>
<td>—</td>
<td>mild hemiparesis &amp; dysphasia</td>
<td>15 yr 9 mo</td>
<td>warfarin 1 yr</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>13</td>
<td>64</td>
<td>M</td>
<td>180/100</td>
<td>6 mo</td>
<td>—</td>
<td>15 mo</td>
<td>warfarin 14 mo</td>
<td>—</td>
<td>myocardial infarct</td>
</tr>
<tr>
<td>14</td>
<td>46</td>
<td>M</td>
<td>170/130</td>
<td>—</td>
<td>aphasia &amp; hemiparesis</td>
<td>5 yr 7 mo</td>
<td>warfarin</td>
<td>5 yr 7 mo</td>
<td>stroke at 1 yr</td>
</tr>
<tr>
<td>15</td>
<td>56</td>
<td>M</td>
<td>160/100</td>
<td>2 mo</td>
<td>mild hemiparesis &amp; hemiparesis</td>
<td>4 mo</td>
<td>aspirin &amp; dipyridamole</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>16</td>
<td>64</td>
<td>M</td>
<td>140/85</td>
<td>1 mo</td>
<td>mild hemiparesis &amp; hemiparesis</td>
<td>4 yr</td>
<td>—</td>
<td>—</td>
<td>carcinoma</td>
</tr>
<tr>
<td>17</td>
<td>61</td>
<td>M</td>
<td>140/90</td>
<td>2 in 3 wks</td>
<td>—</td>
<td>8 yr</td>
<td>warfarin</td>
<td>1 yr 6 mo</td>
<td>—</td>
</tr>
<tr>
<td>18</td>
<td>42</td>
<td>M</td>
<td>120/80</td>
<td>—</td>
<td>mild hemiparesis</td>
<td>3 mo</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>19</td>
<td>61</td>
<td>M</td>
<td>130/80</td>
<td>1 in 2 wks</td>
<td>mild hemiparesis</td>
<td>5 yr 3 mo</td>
<td>warfarin</td>
<td>3 yr 6 mo</td>
<td>stroke at 4 yr</td>
</tr>
<tr>
<td>20</td>
<td>19</td>
<td>M</td>
<td>110/60</td>
<td>—</td>
<td>severe hemiplegia</td>
<td>9 yr 7 mo</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>21</td>
<td>65</td>
<td>M</td>
<td>180/110</td>
<td>1 mo</td>
<td>—</td>
<td>9 yr</td>
<td>aspirin</td>
<td>—</td>
<td>stroke</td>
</tr>
</tbody>
</table>

Table 3 Details of Deaths

<table>
<thead>
<tr>
<th>Number</th>
<th>Time after diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cerebrovascular disease</td>
<td>4</td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>2</td>
</tr>
<tr>
<td>Carcinoma</td>
<td>3</td>
</tr>
<tr>
<td>Cause unknown</td>
<td>1</td>
</tr>
</tbody>
</table>
our series 24% of the patients suffered strokes during
the follow-up period whilst they reported no new
strokes after discharge from hospital. Again in our
study 19% of patients died of cerebrovascular disease
and 10% of myocardial infarction, whilst Hinton et al10
reported no deaths from cerebrovascular or cardiovas-
cular disease during follow-up.

Prognosis for recovery from the initial stroke is,
however, better than that found in studies of MCA
occlusion. Lascelles and Burrows2 and Alcock2 re-
ported a marked recovery in only 18 and 28% respec-
tively of their patients with MCA occlusion but in 56
and 45% of their patients with MCA stenosis. This
compares with good recovery of 71% of our series and
88% of the series of Hinton et al.10 It is also of note that
the cumulative 5 year recurrence rate for brain infarc-
tion was less than that reported in the Framingham
Study.14

The prognosis for patients with MCA stenosis is also
better than that for patients with intracranial internal
carotid artery stenosis as reported in recent series12,13 in
which 30 and 43% suffered cerebrovascular events and
50 and 67% died during a follow-up period of only 30
and 45 months. The mean ages of these groups were
however higher (61.5 and 62.4 years) than in the pres-
tent study (51 years) and there was a higher incidence of
generalized vascular and coronary disease. It seems
that intracranial internal carotid artery stenosis is a
better marker of more severe generalized vascular dis-
 ease than is MCA stenosis.

There was no clear indication in the present study of
any factor which might result in or predict a good
prognosis. Age and hypertension showed no effect
though the numbers are too small for anything less than
a marked effect to be manifest. All the patients who
had further strokes were male. Again small numbers
prevent a conclusion but it is worth noticing that a
preponderance of males among patients with MCA
stenosis was found by Hinton et al10 and in intracranial
internal carotid artery stenosis by Marzewski et al12
and Craig et al.13 In studies of MCA occlusion on the
other hand the sexes have been roughly equal.2,3 A
proximal stenosis producing haemodynamic effects
appears to be more hazardous than a distal stenosis
without haemodynamic consequences but again small
numbers make a firm conclusion difficult.

As might be expected from the nature of the lesion
TIAs were common prior to presentation, occurring
with or without stroke in 15 of the 21 patients (71%)
though this was less than the 94% in the series of
Hinton et al.10 Surprisingly, continued TIAs were not a
feature of the follow-up period, occurring in only 2 of
the patients, confirming the experience of Hinton et
al10 in whom 81% suffered neither TIAs nor stroke
though their period of follow-up was a maximum of six
years. Stenosis of an artery of the size of the MCA
might be expected to produce TIAs of haemodynamic
origin but this did not appear to be so. A stroke was the
subsequent event in those who experienced further ce-
 rebrovascular episodes. It would have been of great
interest to discover if the stroke was associated with
occlusion of the MCA but unfortunately none of the
patients had a further angiogram at this point.

In conclusion it can be seen that the present series of
patients did not have as benign a prognosis as reported
by Hinton et al.10 Strokes occurred in a significant
proportion of patients during the follow-up period. It
remains to be seen whether EC/IC bypass surgery im-
TABLE 4 Angiographic Findings

<table>
<thead>
<tr>
<th>No.</th>
<th>Site</th>
<th>% stenosis</th>
<th>Length</th>
<th>Haemodynamically significant</th>
<th>Other causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>B + C</td>
<td>66</td>
<td>7 mm</td>
<td>No</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Ang br</td>
<td>33</td>
<td>4</td>
<td>No</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>C</td>
<td>50</td>
<td>8</td>
<td>Yes</td>
<td>pericallosal artery stenosis (43%)</td>
</tr>
<tr>
<td>6</td>
<td>C</td>
<td>57</td>
<td>6</td>
<td>No</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>C</td>
<td>62</td>
<td>3.5</td>
<td>No</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>F-P br</td>
<td>40</td>
<td>7</td>
<td>Yes</td>
<td>small plaque on internal carotid artery</td>
</tr>
<tr>
<td>10</td>
<td>C</td>
<td>66</td>
<td>25</td>
<td>Yes</td>
<td></td>
</tr>
</tbody>
</table>
| 13  | C         | 56         | 13     | No                           | slight internal carotid narrowing (20%)
|     |           |           |       |                              | some atheroma in siphon           |
| 14  | C         | 50         | 10     | No                           | slight atheromatous narrowing of internal carotid artery (29%) |
| 15  | C         | 50         | 8      | No                           |                                   |
| 18  | A + B     | 33         | 8      | Yes                          | angular branch occluded           |
| 19  | B         | 50         | 5      | Yes                          | anterior cerebral artery stenosis at origin (33%) |
| 20  | A + B     | 50         | 8      | Yes                          | distal carotid artery narrowed (37%)
|     |           |           |       | anterior cerebral artery, stenosis on other side (23%) |
| 21  | B         | 66         | 6      | No                           | slight common carotid artery stenosis (22%)
|     |           |           |       | mild internal carotid artery stenosis (36%)
|     |           |           |       | anterior cerebral artery stenosis (33%) |

Ang br = angular branch; F-P = ascending frontoparietal branch; A-C = site as defined in figure 1.
proves the prognosis but this study provides data for comparison with surgically treated patients.

References

Prolongation of Bleeding Time and Inhibition of Platelet Aggregation by Low-Dose Acetylsalicylic Acid in Patients With Cerebrovascular Disease
GUDRUN BOYSEN, M.D., ANDERS HASAGER BOSS, M.D., NIELS ØDUM, M.D., AND JØRGEN STEEN OLSEN, M.D.

SUMMARY Platelet aggregation and bleeding time was measured in 43 cerebrovascular patients participating in a controlled, double-blind study of low-dose acetylsalicylic acid. In 19 patients with satisfactory inhibition of the platelet aggregation obtained by 50 to 70 mg acetylsalicylic acid per day the bleeding time averaged 11.2 minutes in contrast to 7.0 minutes in the placebo group, p < 0.001.

This study confirms our previous findings of platelet inhibition by low-dose acetylsalicylic acid in patients with cerebrovascular disease. The prolongation of the bleeding time demonstrates that we are dealing not merely with an in vitro phenomenon but with a significant in vivo effect. The study provides the rationale for clinical evaluations of low-dose acetylsalicylic acid in stroke prophylaxis.

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ACETYLSALICYLIC ACID (ASA) is still the most widely used pharmaceutical agent in platelet inhibition. In stroke prophylaxis two large studies, the Canadian1 and the French1 have shown a significant reduction of stroke risk by giving 1.3 g and 1 g daily, respectively. Other studies1, 2 as well as the recent Danish study3 failed to show a stroke preventing effect of 1 g of ASA daily. The latter study was thoroughly discussed in an accompanying editorial,4 in which it was pointed out that the material was too small to rule out a type II error. However, if it holds true that ASA of 1 g doses lower the stroke risk there is still room for an improvement of the therapeutic results and the possibility exists that a lower dose might be more effective.

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Address correspondence to: Gudrun Boysen, M.D., Department of Neurology, Rigshospitalet, DK-2100 Copenhagen, Denmark. Received May 13, 1983; revision 1 accepted August 29, 1983.

The dose of ASA needed to inhibit the cyclooxygenase of the platelets is much less than 1 gram.2-10 The optimal dose, however, has not been settled. The evidence of a differential effect of small doses of ASA, which inhibit the platelet cyclooxygenase without affecting that of the endothelial cell in the vessel wall speaks in favour of a low-dose regimen in order to preserve the formation of prostacyclin.5-11

We have previously shown that ASA doses of 25-75 mg a day during long-term treatment is sufficient to inhibit platelet aggregation in patients with cerebrovascular disease.12, 13 In the present study we evaluated the effect on bleeding time of small ASA doses in order to demonstrate an "in vivo" effect as a supplement to the "in vitro" aggregation test.

Patient Material
Forty-three patients who participate in a controlled, randomized double-blind study of the effect of low-
Prognosis in middle cerebral artery stenosis.
R N Corston, B E Kendall and J Marshall

doi: 10.1161/01.STR.15.2.237

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