 TABLE 1. Pressure Ratio of Cell Suspension to Buffer After 6 Minutes' Filtration

<table>
<thead>
<tr>
<th>Cells</th>
<th>Controls (n=10)</th>
<th>Cerebral infarction patients (n=10)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Granulocytes</td>
<td>4.73±0.41</td>
<td>5.60±0.72*</td>
</tr>
<tr>
<td>Lymphocytes</td>
<td>5.74±1.18</td>
<td>6.28±0.76</td>
</tr>
<tr>
<td>Monocytes</td>
<td>11.46±2.02</td>
<td>15.98±1.83*</td>
</tr>
</tbody>
</table>

Data are mean±SD. *p<0.01 different from control.

cyte and granulocyte subpopulations, requires continued study to understand further their role in acute cerebral ischemia.

REFERENCES


To the Editor:

We read with interest the report of Drs. Gumerlock and Neuwelt entitled “Carotid endarterectomy: To shunt or not to shunt,” which appeared to indicate that routine bypass shunting during carotid endarterectomy significantly reduces the risk of intraoperative neurologic deficit. We recently completed a study of 103 patients undergoing carotid endarterectomy, who were monitored with a two-channel computerized electroencephalographic/compressed spectral array analysis. The decision to place a shunt was based solely on the presence of CEEG signs of cerebral ischemia detected during carotid cross-clamping. Fourteen patients (13.6%) were shunted, and three of the 14 awoke with new transient (<24 hours) neurologic deficits. In comparison, 89 patients (86.4%) were not shunted and three of the 89 awoke with new neurologic deficits (two transient and one permanent). The most important finding in our study was that five of these six patients who awoke with new neurologic deficits had CEEG signs of cerebral ischemia late during carotid cross-clamping (arteriotomy closure), either after shunt removal or when shunting was no longer technically feasible in the nonshunted patients.

It is possible that routine shunting of all our patients would have prevented the three deficits that occurred in nonshunted patients. However, three other patients suffered a new neurologic deficit despite bypass shunt placement. In addition, routine shunting is not without complications and appears not to have been warranted in the majority of our patients who were neurologically unchanged postoperatively and in whom CEEG did not reveal any ischemic changes. In view of our findings and those of Drs. Gumerlock and Neuwelt, the real question concerning carotid endarterectomy may not be “to shunt or not to shunt,” but “when to shunt and based on what criteria.”

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REFERENCES


Role of Dopamine in Ischemic Neuronal Damage

To the Editor:

Kawano et al1 have recently described a large increase in extracellular dopamine release during ischemia in the striatum of spontaneously hypertensive stroke-resistant rats. The authors hypothesize that the excessive leakage of dopamine may be a causal factor in the development of postischemic neuronal damage. The possibility that dopaminergic neurotransmission might contribute to the vulnerability of the striatum during ischemia has been clearly established by some of our recent sequential studies. Using the microdialysis technique in rats subjected to 20 minutes of four-vessel occlusion, we documented an acute and massive increase of dopamine release into the striatal extracellular space.2 Dopamine deafferentation, by prior unilateral sub-
Role of dopamine in ischemic neuronal damage.
M Y Globus

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 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/20/6/827.2.citation