Vertebrobasilar insufficiency

Part 2: Microsurgical treatment of intracranial vertebrobasilar disease

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Posterior circulation transient ischemic attacks have an associated risk of subsequent infarction of approximately 5% per year. Intracranial vertebrobasilar thrombo-occlusive lesions appear particularly likely to result in repetitive ischemic symptoms and in infarction due to hemodynamic insufficiency. The authors present their experience with 45 patients with symptomatic intracranial vertebrobasilar vascular disease despite maximal medical therapy. The specific operative approaches for intracranial vertebral artery endarterectomy and extracranial to intracranial posterior circulation revascularization procedures are outlined.

**KEY WORDS**
- vertebrobasilar insufficiency
- revascularization
- cerebrovascular disease
- cerebral ischemia
- stroke

**Clinical Material**

**General Patient Characteristics**

Forty-five patients underwent intracranial microvascular surgical procedures in the treatment of thrombo-occlusive disease of the intracranial vertebral artery and basilar arteries (Table 1). There were 39 men and six women. The median age was 60 years for the men (range 37 to 78 years) and 61 years for the women (range 55 to 69 years). Seventy-five percent of the patients suffered from systemic hypertension and 20% had diabetes mellitus. These characteristics are similar to those of patients with extracranial vertebrobasilar disease.

Patients presented with repetitive TIA’s, orthostatic ischemia, posterior circulation infarction, or a combination of the three. All had definitive symptoms of posterior fossa ischemia referable to the vertebrobasilar circulation diagnosed by the rigid clinical criteria of Cartlidge, et al. Patients with intracranial pathology were in general more disabled by their vertebrobasilar disease than were the patients whom we have treated for extracranial vertebrobasilar pathology. Two-thirds of the 45 patients with intracranial disease were unstable with respect to their posterior circulation ischemia.
Microsurgical treatment of intracranial vertebrobasilar disease

Each of the 45 intracranial procedures was performed with the patient under general anesthesia. Electroencephalographic (EEG) recordings (usually obtained by compressed spectral analysis) and auditory and somatosensory evoked potential recordings were performed. Thiopental was used during every intracranial vertebrobasilar procedure and was titrated to the point of EEG burst suppression. Heparin was administered in selected cases.

Proximal Intracranial Vertebral Artery Stenosis

Management. Two patients in our series (Cases 1 and 2) had significant proximal intracranial vertebral artery stenoses that were amenable to endarterectomy (Fig. 1). In one case the contralateral vertebral artery was hypoplastic; in the other it was occluded at C-1. Neither made a significant contribution to the posterior circulation.

For this procedure, the patient is placed in the lateral decubitus or park-bench position with the affected vertebral artery on the upper side. The head is turned slightly toward the floor. The incision begins just medial to the mastoid and curves to the inion at the level of the superior nuchal line. The incision extends in the posterior midline to below the second cervical spinous process. The musculature is reflected from the occiput and the arch of C-1, leaving a cuff of muscle along the nuchal line to facilitate closure. The arch of C-1 is removed. The vertebral artery is exposed extradurally by coagulating and removing the surrounding venous plexus. A unilateral craniotomy, extending from the midline to the mastoid air cells (including the rim of the foramen magnum) is performed. This far lateral exposure improves access to the vertebral artery. The dura mater is opened and the cerebellar tonsil is elevated, exposing the vertebral artery. The ninth, 10th, 11th, and 12th cranial nerves are easily identified and protected during surgery. The exposure of the vertebral artery may be improved by sectioning the first two dentate ligaments and reflecting the spinal accessory nerve laterally. The vertebral artery should be seen distal to the origin of the posterior inferior cerebellar artery (PICA). The atheromatous plaque will be seen distal to the origin of the posterior inferior cerebellar artery (PICA). The atheromatous plaque will be seen distal to the origin of the posterior inferior cerebellar artery (PICA). Temporary aneurysm clips are used to occlude the vertebral artery. The proximal clip may be placed extra- or intradurally as necessary. The distal clip is usually placed just proximal to the PICA region. Small branches to the medulla are unusual in this segment, but if found they can be occluded with miniature temporary clips. The arteriotomy is extended close to the distal clip, and the plaque is removed completely from the vessel wall. The distal part of the vessel is carefully inspected, and all intimal tags are removed. The vessel is closed with a running suture of 9-0 monofilament nylon.
<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs), Sex</th>
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<th>Complications</th>
<th>Clinical Outcome, Follow-Up Period</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>65, M</td>
<td>TIA's with syncope, dysarthria, diplopia, ataxia, lt HP</td>
<td>rt VA small, lt VA 90% distal sten prox to PICA</td>
<td>lt intracranial VA endart</td>
<td>patent</td>
<td>aseptic meningitis, communicating hydrocephalus</td>
<td>required LP shunt, asymptomatic (16 mos)</td>
</tr>
<tr>
<td>2</td>
<td>58, M</td>
<td>postural TIA's with drop attacks, weakness, loss of vision, diplopia, dysarthria</td>
<td>rt VA occl at C-1, lt VA 95% distal sten prox to PICA</td>
<td>lt intracranial VA endart</td>
<td>patent</td>
<td>communicating hydrocephalus</td>
<td>LP shunt, asymptomatic (10 mos)</td>
</tr>
<tr>
<td>3</td>
<td>55, F</td>
<td>TIA's with vertigo, rt facial paresis, lt HP, CVA with brain-stem dysfunction</td>
<td>rt VA occl distal to PICA, lt VA occl at C-2, without intracranial reconstitution</td>
<td>rt occip to lt PICA bypass</td>
<td>patent</td>
<td>none</td>
<td>gradual improvement, asymptomatic (12 mos)</td>
</tr>
<tr>
<td>4</td>
<td>65, M</td>
<td>TIA's with vertigo, CVA with dysarthria, ptosis, lt HP</td>
<td>bilat VA occl at foramen magnum</td>
<td>rt occip to PICA bypass</td>
<td>patent</td>
<td>none</td>
<td>improved, asymptomatic (6 yrs)</td>
</tr>
<tr>
<td>5</td>
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<td>CVA, rt HP, dysarthria, diplopia, postural vertigo</td>
<td>rt VA occl at foramen magnum, lt VA occl at PICA</td>
<td>rt occip to PICA bypass</td>
<td>patent</td>
<td>pneumonia</td>
<td>improved, asymptomatic (6 yrs)</td>
</tr>
<tr>
<td>6</td>
<td>51, M</td>
<td>CVA, rt lateral medullary syndrome, multiple postural TIA's</td>
<td>rt VA occl prox to PICA, lt VA hypoplastic</td>
<td>rt occip to PICA bypass</td>
<td>patent</td>
<td>none</td>
<td>asymptomatic (6 yrs)</td>
</tr>
<tr>
<td>7</td>
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<td>TIA's &amp; CVA with rt HP, numbness, diplopia, drop attacks</td>
<td>rt VA sten prox to PICA, lt VA occl at PICA</td>
<td>rt occip to PICA bypass</td>
<td>patent</td>
<td>none</td>
<td>asymptomatic (5 yrs)</td>
</tr>
<tr>
<td>8</td>
<td>60, M</td>
<td>TIA's with drop attacks, vertigo, diplopia</td>
<td>rt VA occl prox to PICA, lt VA occl at foramen magnum</td>
<td>rt occip to PICA bypass</td>
<td>patent</td>
<td>none</td>
<td>asymptomatic (4 yrs)</td>
</tr>
<tr>
<td>9</td>
<td>63, M</td>
<td>TIA's with vertigo, diplopia CVA with rt HP, dysarthria, rt hearing loss, rt dysmetria</td>
<td>rt VA hypoplastic, lt VA occl at PICA</td>
<td>rt occip to PICA bypass</td>
<td>patent</td>
<td>pneumonia</td>
<td>asymptomatic (4 yrs)</td>
</tr>
<tr>
<td>10</td>
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<td>TIA's with visual loss, diplopia, vertigo, ataxia, dysarthria</td>
<td>rt VA occl distal to PICA, lt VA absent</td>
<td>lt occip to PICA bypass</td>
<td>patent</td>
<td>none</td>
<td>asymptomatic (11 yrs)</td>
</tr>
<tr>
<td>11</td>
<td>61, F</td>
<td>TIA's with diplopia, ataxia, dizziness</td>
<td>rt VA sten at C-1, lt VA absent</td>
<td>rt occip to PICA bypass</td>
<td>patent</td>
<td>aseptic meningitis post-op Day 3, resolved with steroids</td>
<td>no further TIA's (6 mos)</td>
</tr>
<tr>
<td>12</td>
<td>63, M</td>
<td>postural TIA's with drop attacks, diplopia, dysphonia, dysarthria, vertigo, ataxia</td>
<td>bilat prox VA occl, rt VA sten distal to PICA, lt VA did not fill intracranially</td>
<td>lt VA to VCA vein bypass</td>
<td>patent</td>
<td>none</td>
<td>asymptomatic (8 mos)</td>
</tr>
<tr>
<td>13</td>
<td>68, M</td>
<td>TIA's with rt HP, incoordination, vomiting, drop attacks, vertigo, diplopia</td>
<td>rt VA occl at C-1, lt VA sten at PICA</td>
<td>rt occip to PICA bypass</td>
<td>patent</td>
<td>bacterial meningitis, communicating hydrocephalus</td>
<td>resolved with antibiotics, VP shunt, asymptomatic (5 mos)</td>
</tr>
<tr>
<td>14</td>
<td>67, M</td>
<td>TIA's with vertigo, lt-sided ataxia, CVA with ataxia, internuclear ophthalmoplegia</td>
<td>rt VA hypoplastic, lt VA occl prox to PICA, ectatic BA with branch occl</td>
<td>lt occip to PICA bypass</td>
<td>patent</td>
<td>communicating hydrocephalus</td>
<td>required LP shunt, asymptomatic (2 yrs)</td>
</tr>
<tr>
<td>15</td>
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<td>postural TIA's, CVA with lt brain-stem dysfunction, lt ataxia, diplopia</td>
<td>rt VA sten prox to PICA, lt VA occl at PICA</td>
<td>rt occip to PICA bypass</td>
<td>patent</td>
<td>none</td>
<td>asymptomatic (6 mos)</td>
</tr>
<tr>
<td>16</td>
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<td>postural TIA's, ataxia, rt facial paresis</td>
<td>bilat VA occl at foramen magnum</td>
<td>lt occip to PICA bypass</td>
<td>angiography refused (bypass open on Doppler)</td>
<td>none</td>
<td>asymptomatic (3 mos)</td>
</tr>
<tr>
<td>No.</td>
<td>Age</td>
<td>Sex</td>
<td>Diagnosis</td>
<td>Symptoms</td>
<td>Treatment</td>
<td>Outcome</td>
<td></td>
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<tr>
<td>17</td>
<td>61</td>
<td>F</td>
<td>postural TIA's, drop attacks, nystagmus, vertigo, ataxia</td>
<td>rt VA sten at C-1, Lt VA occl at PICA</td>
<td>Lt occip to PICA bypass</td>
<td>occluded initially, patent in later study</td>
<td>none asymptomatic (10 mos)</td>
</tr>
<tr>
<td>18</td>
<td>64</td>
<td>M</td>
<td>TIA's with vertigo, dysarthria, Lt arm weakness</td>
<td>rt VA sten distal to PICA, Lt VA occl prox to PICA</td>
<td>Lt occip to PICA bypass</td>
<td>patent</td>
<td>none asymptomatic (6 mos)</td>
</tr>
<tr>
<td>19</td>
<td>43</td>
<td>M</td>
<td>CVA with fluctuating neurologic deficit, vertigo, ataxia, dysarthria, rt HP, nystagmus</td>
<td>rt VA PICA termination, Lt VA occl at foramen magnum, mid-BA occl, PCoA with supply to superior BA</td>
<td>Lt occip to PICA bypass</td>
<td>patent</td>
<td>none dramatic improvement, asymptomatic (3 yrs)</td>
</tr>
<tr>
<td>20</td>
<td>59</td>
<td>M</td>
<td>severe postural TIA's, CVA with rt HP, dysmetria, severe ataxia</td>
<td>bilat VA sten at foramen magnum, Lt VA sten at PICA</td>
<td>Lt occip to PICA bypass</td>
<td>patent</td>
<td>asymptomatic, HP, ataxia improved (6 mos)</td>
</tr>
<tr>
<td>21</td>
<td>55</td>
<td>M</td>
<td>TIA's with ataxia, diplopia, dysarthria</td>
<td>rt VA occl at PICA, Lt VA sten at PICA</td>
<td>rt occip to PICA bypass</td>
<td>patent</td>
<td>none asymptomatic (6 mos)</td>
</tr>
<tr>
<td>22</td>
<td>65</td>
<td>M</td>
<td>severe postural TIA's, CVA with ptosis, diplopia, dysarthria, rt HP, dysmetria</td>
<td>bilat VA occl at origin, Lt VA with minimal reconstitution, none from rt VA</td>
<td>rt occip to PICA bypass</td>
<td>patent</td>
<td>none asymptomatic (3 yrs)</td>
</tr>
<tr>
<td>23</td>
<td>72</td>
<td>M</td>
<td>TIA's with ataxia, vertigo, nystagmus, rt hyperflexia</td>
<td>rt VA occl prox to PICA, Lt VA occl distal to PICA</td>
<td>rt occip to PICA bypass</td>
<td>patent</td>
<td>asymptomatic, prolonged respiratory insufficiency normal exam, asymptomatic (8 mos)</td>
</tr>
<tr>
<td>24</td>
<td>51</td>
<td>M</td>
<td>severe postural TIA's, CVA with Lt HP, body &amp; rt facial hypalgesia, dysarthria, rt ataxia</td>
<td>rt VA occl prox to PICA, Lt VA hypoplasia with occl distal to PICA</td>
<td>rt occip to PICA bypass</td>
<td>patent</td>
<td>asymptomatic, improved HP, ataxia improved (12 mos)</td>
</tr>
<tr>
<td>25</td>
<td>46</td>
<td>M</td>
<td>postural TIA's with ataxia, Lt extremity dysynergia, rt HP</td>
<td>rt VA sten prox to PICA, Lt VA occl at C-1</td>
<td>rt occip to PICA bypass</td>
<td>patent</td>
<td>asymptomatic (6 mos)</td>
</tr>
<tr>
<td>26</td>
<td>60</td>
<td>M</td>
<td>severe orthostatic cerebral ischemia, vertigo, ataxia, nausea/vomiting</td>
<td>rt VA occl at foramen magnum, Lt VA distal to PICA</td>
<td>rt occip to PICA bypass</td>
<td>angiography refused (bypass open by Doppler)</td>
<td>none asymptomatic (2 yrs)</td>
</tr>
<tr>
<td>27</td>
<td>63</td>
<td>M</td>
<td>TIA's, CVA with rt facial paresis, diplopia, dysarthria, rt hearing loss, ataxia</td>
<td>rt VA hypoplasia (no intracranial supply), Lt VA occl distal to PICA</td>
<td>rt occip to PICA bypass</td>
<td>patent</td>
<td>asymptomatic, exam unchanged (18 mos)</td>
</tr>
<tr>
<td>28</td>
<td>37</td>
<td>M</td>
<td>TIA's with vertigo, dysarthria, ataxia, rt facial numbness, CVA with rt lateral medullary deficit</td>
<td>rt VA occl at PICA, severe Lt VA sten involving PICA</td>
<td>rt STA-SCA bypass</td>
<td>patent</td>
<td>asymptomatic (10 mos)</td>
</tr>
<tr>
<td>29</td>
<td>61</td>
<td>M</td>
<td>CVA with diplopia, Lt hemisensory loss, severe postural TIA's with rt HP, diplopia</td>
<td>rt VA PICA termination, Lt VA sten distal to PICA</td>
<td>rt STA-SCA bypass</td>
<td>patent</td>
<td>midbrain contusion with transient Lt HP full recovery, asymptomatic (2 yrs)</td>
</tr>
<tr>
<td>30</td>
<td>58</td>
<td>M</td>
<td>postural TIA's with dizziness, drop attacks, dysarthria, CVA with Lt hemianopsia</td>
<td>rt VA occl at foramen magnum, Lt VA sten at origin &amp; distal to PICA</td>
<td>rt STA-SCA bypass</td>
<td>patent</td>
<td>asymptomatic (8 mos)</td>
</tr>
<tr>
<td>31</td>
<td>69</td>
<td>F</td>
<td>headache, dizziness, diplopia, dysarthria, ataxia (progressive)</td>
<td>giant BA aneurysm</td>
<td>rt STA-SCA bypass, BA ligation</td>
<td>patent</td>
<td>transient coma complete recovery, asymptomatic (3 yrs)</td>
</tr>
</tbody>
</table>

* BA = basilar artery; contrib = contributions; CVA = cerebrovascular accident; endart = endarterectomy; HP = hemiparesis; LP = lumboperitoneal; occl = occllal artery; occl = occlusion; PCA = posterior cerebral artery; PCoA = posterior communicating artery; PICA = posterior inferior cerebellar artery; prox = proximal; SCA = superior cerebellar artery; STA = superficial temporal artery; sten = stenosis; SX = symptoms; TIA's = transient ischemic attacks; unilat = unilateral; VA = vertebral artery; VP = ventriculoperitoneal.

(TABLE 1 continued ↓)
<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs), Sex</th>
<th>Clinical Presentation</th>
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<th>Clinical Outcome, Follow-Up Period</th>
</tr>
</thead>
<tbody>
<tr>
<td>32</td>
<td>73, M</td>
<td>headache, dementia, ataxia (progressive)</td>
<td>giant BA aneurysm</td>
<td>rt STA-SCA bypass, BA ligation</td>
<td>patent</td>
<td>delayed sepsis</td>
<td>initial improvement, 6 wks postop sepsis, died</td>
</tr>
<tr>
<td>33</td>
<td>60, M</td>
<td>TIA's with diplopia, rt facial numbness, lt HP, ataxia</td>
<td>lt VA sten at PICA, mid-BA sten, hypoplastic PCoA bilat</td>
<td>rt STA-SCA bypass</td>
<td>patent</td>
<td>seizure, cardiac dysrhythmia</td>
<td>medical therapy for seizure &amp; dysrhythmia, asymptomatic (18 mos)</td>
</tr>
<tr>
<td>34</td>
<td>56, M</td>
<td>TIA's with dizziness, dysarthria, diplopia, ataxia</td>
<td>mid-BA sten, no PCoA contrib</td>
<td>rt STA-SCA bypass</td>
<td>patent</td>
<td>aseptic meningitis, resolved with steroids</td>
<td>asymptomatic (18 mos)</td>
</tr>
<tr>
<td>35</td>
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<td>TIA's with drop attacks, nausea, diplopia, weakness, ataxia</td>
<td>prox BA sten, small unilat PCoA</td>
<td>rt STA-SCA bypass</td>
<td>patent</td>
<td>none</td>
<td>asymptomatic (3 mos)</td>
</tr>
<tr>
<td>36</td>
<td>53, M</td>
<td>TIA's with diplopia, dysarthria, unsteadiness</td>
<td>mid-BA sten (&gt; 90%), PCoA not seen bilat</td>
<td>rt STA-SCA bypass</td>
<td>patent</td>
<td>confusion for 48 hrs, resolved</td>
<td>no further TIA's (6 mos)</td>
</tr>
<tr>
<td>37</td>
<td>62, M</td>
<td>CVA with dysarthria, mild ataxia, TIA's with diplopia, dysarthria, rt HP, ataxia</td>
<td>mid-BA sten (90%), small PCoA bilat</td>
<td>rt STA-SCA bypass</td>
<td>occluded</td>
<td>none</td>
<td>no change in SX (12 mos)</td>
</tr>
<tr>
<td>38</td>
<td>52, M</td>
<td>CVA with diplopia, dysarthria, TIA's with drop attacks, loss of vision, ataxia</td>
<td>mid-BA occl, no PCoA contrib</td>
<td>rt STA-SCA bypass</td>
<td>patent</td>
<td>none</td>
<td>gradual recovery, no further TIA's (8 mos)</td>
</tr>
<tr>
<td>39</td>
<td>60, M</td>
<td>CVA with mild ataxia, postural TIA's with drop attacks, diplopia, weakness</td>
<td>mid-BA sten (90%), unilat PCoA contrib</td>
<td>rt STA-SCA bypass</td>
<td>patent</td>
<td>aseptic meningitis post-op Day 3, communicating hydrocephalus (resolved with steroids/LP shunt)</td>
<td>occasional postural SX, less frequent &amp; less severe (12 mos)</td>
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<tr>
<td>40</td>
<td>48, M</td>
<td>CVA with rt 3rd nerve palsy, lt HP, TIA's with postural diplopia, black out spells, dysarthria, rt HP</td>
<td>bilat VA occl at foramen magnum, severe BA disease, no PCoA contrib</td>
<td>rt STA-PCA bypass</td>
<td>patent</td>
<td>seizure, postop hematoma</td>
<td>improved lt HP, asymptomatic (1 yr)</td>
</tr>
<tr>
<td>41</td>
<td>66, F</td>
<td>postural TIA's with vertigo, dysarthria, diplopia, lt HP; CVA with dysarthria, lt HP, rt 6th nerve palsy; 2nd CVA with rt HP &amp; mutism</td>
<td>rt VA occl distal to PICA, mid-BA sten, hypoplastic PCoA</td>
<td>rt STA-PCA bypass</td>
<td>occluded</td>
<td>none</td>
<td>no improvement, refused further treatment (4 yrs)</td>
</tr>
<tr>
<td>42</td>
<td>48, M</td>
<td>CVA with lt HP, dysmetria ataxia, postural TIA's, dysarthria</td>
<td>rt VA sten at PICA, lt VA occl at foramen magnum, no PCoA contrib</td>
<td>rt STA-PCA bypass</td>
<td>patent</td>
<td>rt temporal lobe hematoma with increased lt HP</td>
<td>improved, asymptomatic (5 yrs)</td>
</tr>
<tr>
<td>43</td>
<td>59, M</td>
<td>CVA with dysarthria, lt HP, TIA's with dizziness, dysarthria, increased HP</td>
<td>bilat VA sten prox to PICA, rt VA sten distal to PICA, poor visualization of PCoA bilat</td>
<td>rt STA-PCA bypass</td>
<td>patent</td>
<td>none</td>
<td>improved, asymptomatic (3 yrs)</td>
</tr>
<tr>
<td>44</td>
<td>66, M</td>
<td>TIA's with postural dizziness, alternating hemianopia, rt HP, dysmetria</td>
<td>mid-BA sten, slight lt PCoA contrib</td>
<td>rt STA-PCA bypass</td>
<td>patent</td>
<td>none</td>
<td>improved, asymptomatic (5 yrs)</td>
</tr>
<tr>
<td>45</td>
<td>56, F</td>
<td>CVA with rt HP, dysarthria, 2nd CVA with lt HP, ataxia</td>
<td>bilat VA sten at VA-BA junction, small PCoA bilat</td>
<td>rt subclavian-PCA vein bypass</td>
<td>patent</td>
<td>pneumonia</td>
<td>recovered, asymptomatic (3 yrs)</td>
</tr>
</tbody>
</table>

* BA = basilar artery; contrib = contributions; CVA = cerebrovascular accident; endart = endarterectomy; HP = hemiparesis; LP = lumboperitoneal; occip = occipital artery; occl = occlusion; PCA = posterior cerebral artery; PCoA = posterior communicating artery; PICA = posterior inferior cerebellar artery; prox = proximal; SCA = superior cerebellar artery; STA = superficial temporal artery; sten = stenosis; SX = symptoms; TIA's = transient ischemic attacks; unilat = unilateral; VA = vertebral artery; VP = ventriculoperitoneal.
Microsurgical treatment of intracranial vertebrobasilar disease

Outcome. Postoperative angiography revealed patency of the endarterectomized vessels, and both patients were relieved of ischemic symptoms. One patient required lumboperitoneal shunting following the procedure.

Bilateral Compromise of Proximal Vertebral Arteries

Management. Twenty-four patients (Cases 3 to 11 and 13 to 27) with bilateral compromise (stenosis, occlusion, or hypoplasia) of the proximal intracranial vertebral arteries were treated with an occipital artery-PICA bypass procedure. An additional patient (Case 12) had a saphenous vein bypass graft from the vertebral artery (at C-1) to the PICA. This patient had bilateral occlusion of the vertebral arteries in the neck, with reconstitution of the left vertebral artery by muscular collateral vessels. In an attempt to treat his recurrent postural TIA's, an occipital artery to distal extracranial vertebral artery bypass was performed. This bypass became occluded due to a stenotic intracranial vertebral artery lesion that impeded runoff from the graft. Because collateral flow into the extracranial vertebral artery was good, a saphenous vein graft was used to bypass the intracranial stenosis by anastomosing it to the PICA.

The patient is placed in the lateral decubitus position with the side to receive the anastomosis upward. The course of the occipital artery is identified with the Doppler probe and marked on the scalp. A hockey-stick incision is made from the mastoid to the inion, extending down the midline to below the C-2 spinous process. The scalp and musculature are separated from the occiput and the arches of C-1 and C-2, taking care to leave the occipital artery in continuity. The occipital artery is dissected from the muscles until it has been isolated as far as the mastoid process. Small branches are coagulated and divided. This vessel adheres to the surrounding muscles and is much more difficult to dissect free than is the superficial temporal artery (STA). A unilateral suboccipital craniotomy extending through the rim of the foramen magnum is made. The arch of the C-1 vertebra is removed. After the dura mater is opened the caudal loop of the PICA can be seen under the tonsil, posterior to the medulla (Fig. 2). Arachnoidal adhesions are removed and a small plastic dam is placed under the artery. The PICA can be elevated further, making access easier by placing a small piece of Gel-foam under the dam. A Microvac suction device* is placed underneath or near the dam to constantly clear cerebrospinal fluid (CSF). The occipital artery is clipped proximally, divided distally, and flushed with heparinized saline solution. The orifice is prepared by removing its adventitia and spatulating the end. Temporary clips are placed on the PICA, and a linear arteriotomy is made without excising any of the vessel wall. The anastomosis is completed by stitching the occipital artery to the PICA with 10-0 sutures at either end of the arteriotomy. After the bypass is complete, the clips are removed first from the PICA, then from the occipital artery. The anastomosis should be dry and clearly patent. The dam is removed, and the dura mater is reaproximated. A watertight closure is not possible, so the defect is covered with Gelfoam. A careful, watertight closure of the muscles and scalp is critical and is facilitated by moving the patient's head out of flexion during closure.

As noted, one patient developed occlusion of an occipital artery-vertebral artery bypass and required a vertebral artery-PICA saphenous vein interposition graft procedure. The scalp incision and muscle dissection were carried out as above. Due to the good pulsation in the vertebral artery at the C-1 level, this was used as the donor vessel for the bypass. The extracranial vertebral artery, which had previously been isolated, was clipped and the occluded occipital graft removed. A lateral suboccipital craniotomy was performed, the dura mater opened, and the PICA isolated as described above. A segment of saphenous vein taken from the lower leg was sutured to the vertebral arteriotomy using a 9-0 monofilament suture. The PICA was clipped, and the vein graft was anastomosed to a long linear arteriotomy in the recipient vessel. Immediately preceding

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* Suction device manufactured by PMT Corp., Hopkins, Minnesota.
completion of the anastomosis the vertebral artery was temporarily unclipped to allow expulsion of air from the graft. Under gentle heparinized saline irrigation, the vein graft-PICA anastomosis was completed. The distal PICA clip was removed first, followed by the proximal PICA clip, and then the vertebral artery clips. There was an excellent pulse in the vein graft at completion.

Outcome. Twenty-three of the occipital artery-PICA anastomosis patients underwent postoperative angiography and had patent bypasses. Two patients who refused angiography underwent Doppler studies that suggested patency. Four patients had transient postoperative complications: meningitis in three (aseptic in two, bacterial in one) and communicating hydrocephalus which required shunting in one. All 25 patients were relieved of ischemic symptoms.

Compromise of Vertebral and/or Basilar Artery

Management. Twelve patients (Cases 28 to 39) underwent STA to superior cerebellar artery (SCA) anastomosis for either severe bilateral vertebral artery compromise distal to the PICA, for vertebral artery-basilar artery junction disease, or for low to mid-basilar artery stenosis or occlusion. In these cases there was either poor flow or no angiographic evidence of flow to the distal basilar artery from the anterior circulation via the posterior communicating arteries. Two of these patients (Cases 31 and 32) underwent basilar artery ligation after their STA-SCA bypass procedure for the treatment of giant basilar artery aneurysms.

Five patients (Cases 40 to 44) with angiographic evidence of compromise of the distal basilar artery circulation due to bilateral vertebral artery disease, mid-basilar artery disease, or both were treated with STA to posterior cerebral artery (PCA) anastomoses. Another patient (Case 45) underwent placement of a saphenous interposition graft from the subclavian artery to the PCA. None of these patients had evidence of significant collateral blood supply from the anterior circulation via fetal variants or posterior communicating arteries.

For these procedures, the SCA or the PCA is exposed through a very low subtemporal approach, routinely on the right side to avoid threatening the dominant temporal lobe. The vein of Labbé must be spared and the temporal lobe retraction minimized to avoid causing venous infarction or contusion. Mannitol administration and CSF drainage relaxes the brain for a safe exposure. The tentorium is incised to allow adequate exposure of the lateral portion of the SCA. The posterior cerebral artery (PCA) and trochlear nerve (IV) are depicted.

device is placed to keep the anastomotic site clear of CSF. In all but one case the STA was used as the donor vessel. The STA is isolated in standard fashion during the opening. When the recipient vessel has been prepared, the STA is temporarily clipped proximally, divided distally, and flushed with heparinized saline. Barbiturates are administered before the recipient vessel is clipped. The orifice of the STA, which has been spatulated, is sutured to a linear arteriotomy in the selected vessel with 10-0 monofilament nylon. This technically challenging portion of the procedure requires long instruments specially designed for bypass procedures† to reach the deep recipient vessel. After the anastomosis is completed, the temporary clips are removed first from the SCA or PCA, and last from the STA. The closure is routine. The STA graft must not be compromised during dural approximation, bone flap fixation, or muscle closure.

In one case a saphenous vein graft was used because the patient had hypoplastic superficial temporal vessels. The patient was systemically heparinized before the recipient vessel (the PCA) was clipped to avoid graft thrombosis. The anastomosis between the PCA and the vein graft is placed first. Two running sutures are used to...

† Instruments manufactured by Codman & Shurtleff, Inc., Randolph, Massachusetts.
to close the back and front walls of the anastomosis to insure a tight seal. After the intracranial anastomosis is completed, the vein graft is passed subcutaneously, posterior or anterior to the ear, to the neck incision where it may be anastomosed to either the external carotid artery or the subclavian artery.

**Outcome.** Postoperative angiography revealed bypass patency in 16 of the 18 patients. One patient with an STA-SCA anastomosis (Case 37) and one with an STA-PCA anastomosis (Case 41) had occluded bypasses, and had no change from their preoperative ischemic symptoms. Fourteen of 16 patients with patient anastomoses had relief of symptoms. Two patients had a reduction of symptoms following their respective procedures, one of whom (Case 32) died 6 weeks after surgery from septicemia. Postoperatively, there were two hematomas and one midbrain contusion among this group of patients, each of which resolved with supportive therapy. One patient required shunting for communicating hydrocephalus.

**Results**

There were no postoperative posterior circulation infarctions among the 45 patients treated with intracranial vertebrobasilar thrombo-occlusive disease. There were no late complications (other than one death) in a follow-up period that averaged 18 months. The single death was not neurologically related and occurred 6 weeks postoperatively in a patient with a giant basilar artery aneurysm. The patient demonstrated postoperative neurological improvement following placement of an STA-SCA anastomosis and basilar artery ligation but developed septicemia 5 weeks after surgery from a urinary tract source. He died 1 week later.

In 41 (91%) of the 45 patients with intracranial vertebrobasilar disease the ischemic symptoms were improved after revascularization procedures (Table 2). Two patients had continuing symptoms, albeit at a reduced frequency (one of these was the patient who died). In two patients symptoms remained unchanged after surgery; in both, postoperative angiography showed occluded bypasses (one an STA-SCA and the other an STA-PCA anastomosis).

Of 19 early complications (up to 30 days postoperatively) among the 45 patients treated, 12 were minor and all were transient. Five patients had postoperative communicating hydrocephalus, which was treated with lumbarperitoneal shunting in four and ventriculoperitoneal shunting in one. One patient developed bacterial menigitis and three others had aseptic meningitis which was treated with appropriate antibiotic (bacterial) or steroid (aseptic) therapy. Two patients contracted postoperative pneumonia and were treated effectively with aggressive pulmonary and antibiotic therapy. One patient suffered a generalized seizure and cardiac dysrhythmia 3 days after an STA-SCA anastomosis. These conditions were treated with Dilantin (phenytoin) and digoxin, respectively. One patient was confused for 2 days after surgery but his mentation improved to normal on the 3rd day without specific treatment.

Seven patients had more significant temporary complications in the early postoperative period. Three patients with preoperative pulmonary disease developed complex pneumonia or respiratory distress which required prolonged ventilatory support and steroid and antibiotic therapy. All three recovered without sequelae. In two patients a postoperative CT scan revealed a hematoma at the operative site: one presented with a seizure, and the second patient's preoperative hemiparesis increased. Both patients improved with supportive therapy. One of two patients treated for a giant basilar artery aneurysm was comatose after an STA-SCA bypass procedure and subsequent basilar artery ligation. She gradually made a full recovery and remained asymptomatic at her last follow-up examination 3\(\frac{1}{2}\) years postoperatively. The final patient with a complication had undergone placement of an STA-PCA bypass. The patient suffered a new postoperative hemiparesis, and a CT scan revealed a midbrain contusion. This patient recovered full function and strength with supportive care.

**Discussion**

Managing patients with intracranial vertebrobasilar atherosclerosis should be guided by the pathology and natural history of the specific lesion within the vertebrobasilar arterial tree. If indicated, surgical therapy should be directed at the causative lesion to eliminate its associated pathophysiological effects.

**Pathology**

Clinically significant vertebrobasilar occlusive disease is more commonly associated with intracranial arterial stenoses or occlusions than with extracranial vertebrobasilar lesions. Intracranial vertebrobasilar lesions often have a greater hemodynamic impact on posterior circulation blood flow than do extracranial vertebral artery lesions because the intracranial lesion is beyond the rich vascular collateral network existent in the neck and at the base of the skull.

The fourth segment of the vertebral artery (that portion between the dura mater and the vertebrobasilar
junction) is frequently compromised by atherosclerotic disease; this is particularly true of the portion of the vessel from the dura mater to the PICA. Twenty-five patients in our series had either bilateral high-grade stenoses or occlusions of the intracranial vertebral artery proximal to or involving the origin of the PICA's, if they had unilateral disease with contralateral extracranial vertebral artery disease, vertebral artery hypoplasia, or PICA termination. The vertebral artery segment extending from the PICA to the basilar artery was compromised in eight patients, and only one patient had stenosis or occlusion involving the vertebral arteries at the vertebrobasilar junction.

Basilar artery stenosis was less common than was intracranial vertebral artery thrombo-occlusive disease, and occurred in the mid-basilar portion (four patients); the distal or upper basilar artery was not affected in any patient. These findings are similar to those in the series reported by Castaigne, et al.15 Two patients had focal lesions of the intracranial vertebral artery proximal to the PICA; these were low enough to be accessible for direct vertebral artery endarterectomy. In these patients friable ulcerated lesions were removed. Allen, et al.,1 and Ausman and colleagues3,4 have also found ulcers at this level, some of which were calcified.

Pathophysiology

Three mechanisms may be responsible for vertebrobasilar ischemia and/or infarction from intracranial vertebrobasilar pathology: hemodynamic insufficiency, intra-arterial embolization, and atherosclerotic occlusion of brain-stem perforating branches.

Hemodynamic insufficiency may be responsible for the majority of symptoms of vertebrobasilar ischemia. Naritomi, et al.,38 measuring regional cerebral blood flow with xenon-133, documented a loss of posterior circulation autoregulatory mechanisms in patients with vertebrobasilar ischemia, and reported reductions in posterior circulation perfusion with postural changes. All patients in our series (excluding those with giant basilar artery aneurysms) had bilateral compromise of the vertebral artery blood supply or a marked stenosis (or occlusion) of the basilar artery, highlighting the significance of flow restriction in the genesis of vertebrobasilar symptoms. Postural changes in many of these patients either caused or exacerbated symptoms. For example, one patient (Case 2) who underwent intracranial vertebral artery endarterectomy for 95% stenosis of the left vertebral artery (the right vertebral artery was occluded at C-1) was bedridden preoperatively. Elevating his head above 45° caused symptoms of dysarthria, diplopia, and weakness of the extremities, which ceased after surgery.

Occlusion of the vertebral artery or basilar arteries probably represents a progression of the atherosclerotic disease process. While emboli from a more proximal arterial lesion (or from the heart) may cause sudden vessel occlusion, the majority of posterior circulation occlusions are due to local thrombosis superimposed on existing atheromatous disease (stenosis). Castaigne and colleagues15 discovered that 94% of basilar artery occlusions and 68% of vertebral artery occlusions were caused by thrombosis on preexisting atherosclerotic stenotic lesions. The morbidity from these vascular occlusions varies and depends on temporal factors (acute vs. gradual), the vessel affected, the location of the occlusion along the vessel, and the available collateral circulation. Ischemia distal to vessel occlusion is most often due to hemodynamic insufficiency.

Intra-arterial embolization from proximal atherosclerotic lesions has been reported as a cause of posterior circulation ischemia and infarction.7,15,22,33 This phenomenon appears to explain the symptoms of vertebrobasilar ischemia in patients with significant unilateral vertebral artery disease with a patent contralateral vertebral artery. Few patients in the literature fulfill these criteria; and it may be that these patients are best treated with antiplatelet aggregating agents. None were included in this series. The majority of patients in our series were referred for surgical evaluation because their symptoms persisted despite maximal medical therapy. Although intra-arterial embolization is a predominant factor in the genesis of TIA's in the anterior circulation, it may be less significant in the posterior circulation. Propagation of thrombus to occlude brain-stem perforating vessels or atherosclerotic occlusion of these small end arteries is probably the least common mechanism for generating repetitive symptoms of vertebrobasilar ischemia and the least likely to respond to subsequent medical or surgical therapy.

Natural History of Vertebrobasilar Ischemia and Intracranial Vertebral Artery Disease

Vertebrobasilar TIA's diagnosed by the rigid criteria advocated by Cartlidge, et al.,14 have an associated rate of infarction similar to that of anterior circulation TIA's: approximately 22% to 35% over 5 years (4.5% to 7% per year).14,25 Significantly, the vast majority of patients in our series who suffered posterior circulation strokes before evaluation (19 of 22) had multiple warning TIA's in the days and weeks preceding the infarction. Similarly, Fisher19 reported that 80% of patients who experienced vertebrobasilar strokes in his series had posterior circulation TIA's over a mean period of 7 to 8 weeks before the infarction.

The prognosis for patients who suffer a vertebrobasilar territory infarction is debatable. Norris and associates39 reported more favorable morbidity and mortality rates after vertebrobasilar stroke than after anterior circulation infarction. In contrast, several authors have described a significantly greater acute mortality rate (20% to 30%) from posterior circulation infarction than from strokes in the carotid system.29,37,40 Jones, et al.,29 reported that 54% of patients who suffered vertebrobasilar strokes experienced progressive neurological deterioration or a remitting-relapsing course after the initial onset of symptoms, and that this course might not stabil-
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ize for 48 to 96 hours. Their mortality rate after 1 week was 27% among vertebrobasilar stroke victims. 2.5 times greater than the 10.6% mortality rate that Jones and Millikan28 had previously described for patients with acute carotid system strokes.

Generalizations about the natural history of vertebrobasilar insufficiency are inadequate to guide appropriate therapy for specific lesions within the vertebrobasilar vascular tree. Different lesions have a different import on the risk of subsequent infarction, the risk of repetitive symptoms, and the likelihood of successful therapeutic intervention (medical or surgical). Each specific lesion type should be analyzed separately.11,12

Intracranial vertebral artery occlusion may be well tolerated if the disease process is unilateral and not acute. Lateral medullary infarction or cerebellar hemisphere infarctions rarely occur and are usually well tolerated by patients with adequate collateral blood supply from the contralateral vertebral artery.10,12,13 Severe stenosis or occlusion of one vertebral artery complicated by contralateral vertebral artery occlusive disease, hypoplasia, or PICA termination more frequently causes vertebrobasilar ischemia and infarction. The mechanism for ischemic events in these instances is most likely hemodynamic, although the possibility of intra-arterial embolization from the site of occlusion cannot be excluded.

Bilateral preocclusive and occlusive intracranial vertebral artery disease is associated with a very high risk of stroke. All nine patients with bilateral occlusions of the intracranial vertebral arteries reported by Caplan10 died of brain-stem and/or cerebellar infarction despite vigorous medical therapy including anticoagulation therapy. Thirty-four of the 45 patients with intracranial disease in this series had various combinations of bilateral vertebral artery compromise which led to repetitive vertebrobasilar TIA’s and infarctions. These patients responded to surgical therapy (endarterectomy or vascular bypass procedures) directed at improving posterior circulation blood flow. This prerequisite of bilateral vertebral artery compromise in patients who improve after revascularization of the posterior fossa has been reported by other investigators.2,3,7,26,45,49

Atherosclerotic disease of the basilar artery also carries a significant risk of brain-stem stroke.12 Basilar artery occlusion most often results from thrombosis at the site of preexistent atherosclerotic disease;15 thus, the patient with basilar artery stenosis is at risk for distal intra-arterial embolization and, most importantly, for subsequent basilar artery occlusion. Occasionally, basilar artery occlusion is tolerated.10,12 In these cases collateral blood supply from the cortical PICA vessels to the SCA branches and contributions from the carotid system (posterior communicating arteries and/or fetal trigeminal arteries) are essential to maintain adequate perfusion. Patients without significant angiographically documented collateral channels are at high risk for persistent ischemia and subsequent infarction. These patients are candidates for surgical procedures designed to improve blood flow in the distal basilar artery territory.

Exclusive of the risk of infarction, the repetitive nature of TIA’s in such patients is often disabling. In our experience, patients with intracranial disease were much more susceptible to symptoms after postural changes and had more frequent and severe symptoms than did patients with extracranial posterior circulation compromise. Half of the patients with intracranial posterior fossa disease had already experienced a posterior circulation cerebrovascular accident of mild to moderate severity.

Evaluation

In the evaluation of vertebrobasilar ischemia, nonvascular causes of vertebrobasilar symptoms (cardiac, hematological, and migraine-related symptoms) must be considered and excluded with appropriate clinical and laboratory investigations including electrocardiography.11 High-resolution CT is useful to define regions of hemorrhage or infarction in the posterior circulation distribution. Thin-section images through the posterior fossa are required to reduce the bone artifact which frequently compromises the clarity of these studies. Small areas of brain-stem infarction are often obscured for this reason. Magnetic resonance imaging is an invaluable aid to the clinician in the assessment of patients with suspected vertebrobasilar ischemia because it has an exquisite sensitivity for early ischemic pathology, offers high-quality resolution, and generates no bone artifact.23,48,57 Cerebral blood flow studies utilizing xenon-133 have defined regions of ischemia in cases of vertebrobasilar ischemia.38 With improvement in the quality and resolution of the positron emission tomography (PET) images, PET scanning may also prove beneficial in the evaluation of this disease process.30

All portions of the cerebral vasculature, from the aortic arch and cervical segments to the intracranial anterior and posterior vascular distributions, must be examined with selective angiography. Tandem lesions, branch occlusions, and channels of vascular collateral and external carotid artery branches must be identified to assess the severity of the disease and to plan potential revascularization procedures. The safety of cerebral angiography has improved significantly over recent years with the refinement of angiographic techniques. Several recent reviews have demonstrated that vertebral artery angiography carries no more risk than that associated with carotid angiography. The risk of death or permanent neurological impairment is less than 1% for these procedures.17,34-36 Digital intravenous angiography does not provide the precise definition of extracranial carotid angiography necessary for the evaluation and treatment of patients with vertebrobasilar insufficiency.53 It is, however, useful for the postoperative assessment of bypass patency and vascular reconstruction.
Treatment

The surgical approach for an individual patient is determined by the location of the lesion(s), the pathophysiology responsible for the ischemic symptoms, and individual variations in both intracranial and extracranial vascular anatomy. Allen, et al.,1 and Ausman, et al.,3 reported their experience with intracranial vertebral artery endarterectomies involving a total of six patients (two and four cases, respectively). Of six attempted endarterectomies, three were patent postoperatively, one became occluded, and two were aborted due to lengthy plaque extension or erosion through the vertebral artery wall. Both publications emphasized the complexity of the procedure and advocated careful patient selection and an intensive trial of medical therapy after surgery. Both patients in our series who underwent this procedure (Cases 1 and 2, Table 1) had discrete and severely stenotic lesions proximal to the PICA, one of which was ulcerated. This direct approach to the offending pathology is ideal because it improves flow in the vertebral artery and also eliminates the vertebral artery plaque and the site of genesis of potential intra-arterial emboli.1,2,4

An occipital artery to PICA bypass procedure has been performed for the treatment of significant fourth-segment vertebral artery occlusive disease. This procedure is directed at lesions proximal to the origin of the PICA and is responsible for distal hemodynamic insufficiency in the vertebrobasilar vascular distribution. An interposition vein graft may be employed when extracranial carotid artery vessels are inadequate or unavailable to serve as donor vessels.40 Several groups have reported their experiences with this procedure and have demonstrated the efficacy of EC-IC bypass in the treatment of intracranial vertebrobasilar thrombo-occlusive disease.3,26,31,32,42,45,49,50,52 Sundt and Piepgras49 performed 39 of these procedures with a perioperative mortality rate of 5% and a graft occlusion incidence of 12.8% (five of 39 cases). Fifteen patients had an excellent outcome with relief of symptoms, 18 were improved, and four remained unchanged. Ausman and coworkers3 have reported eight occipital artery to PICA bypass cases of STA-SCA bypass. They had no perioperative neurological and one from cardiac causes) for a 23% mortality rate. Sundt and Piepgras49 described seven cases of STA-SCA bypass. They had no perioperative deaths but concluded that five of these procedures were unsuccessful due to poor flow through the graft.49 Ausman, et al.,3 reported that 11 of their 14 patients became asymptomatic after an STA-SCA anastomosis. One graft was discovered to be occluded postoperatively and that occlusion, in association with basilar artery thrombosis, led to the only death in their series (7% mortality).

Our results with 45 posterior fossa EC-IC bypass procedures compare favorably to the outcome in other reported series. A high rate of patency was achieved (95%), and the majority of patients were improved (95.5%). Based on this combined experience with intracranial vertebrobasilar thrombo-occlusive disease, it appears that intracranial revascularization procedures are justified for selected patients suffering from recalcitrant symptoms of vertebrobasilar ischemia.1,2,3,6,18,26,27,31,32,42,45,49,50,52 Few postoperative vertebrobasilar strokes and relatively low perioperative mortality rates have been reported. We and others have experienced a marked incidence of postoperative complications (most of which have been temporary) after intracranial vertebrobasilar revascularization procedures. This directly reflects the compromised state of the patients preoperatively and the technical difficulty associated with performing these deep microvascular anastomoses in the posterior fossa.

The long-term efficacy of EC-IC bypass procedures in the posterior fossa remains to be proven. These revascularization procedures may be more effective than anterior circulation EC-IC bypass procedures because patients with vertebrobasilar ischemia usually only become symptomatic when a portion of the brain stem or cerebellum becomes isolated with little or no collateral blood supply. The posterior circulation EC-IC anastomosis requires the reperfusion of a smaller unit of ischemic tissue, unlike an STA-middle cerebral artery bypass, which is intended to reperfuse a significant portion of the cerebral hemisphere. In addition, posterior circulation ischemia appears to be predominantly related to hemodynamic insufficiency, which theoretically can be ameliorated with revascularization. In the anterior circulation, emboli may play a larger role in the genesis of ischemic symptoms, a fact that may contribute to their higher postoperative stroke rate.

Conclusions

The 95% patency rate in this series testifies to the technical feasibility of revascularizing the brain stem. The relief of symptoms in 91% of patients suggests that these techniques can ameliorate hemodynamically induced vertebrobasilar ischemic symptoms in appropriately selected patients. The lack of surgical mortality and relatively low serious morbidity in this series of
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patients treated for intracranial vertebrobasilar atherosclerosis (excluding aneurysm patients) suggest the safety of these techniques. However, this conclusion may be misleading because the series is small, and the morbidity and mortality rates in similar series by recognized experts have been appreciably higher. The technical difficulty of these procedures and the prerequisite of a team approach between neuroanesthesia and neurosurgery cannot be overemphasized.

References

34. Mani RL, Eisenberg RL: Complications of catheter cerebral arteriography: analysis of 5,000 procedures. II. Relation of complication rates to clinical and arteriographic diagnoses. AJR 131:867–869, 1978
35. Mani RL, Eisenberg RL: Complications of catheter cerebral arteriography: analysis of 5,000 procedures. III. Assessment of arteries injected, contrast medium used,

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