Use of a wedged microcatheter for curative transarterial embolization of complex intracranial dural arteriovenous fistulas: indications, endovascular technique, and outcome in 21 patients

PETER KIM NELSON, M.D., STEPHEN M. RUSSELL, M.D., HENRY H. WOO, M.D., ANTHONY J. G. ALASTRA, M.D., AND DANKO V. VIDOVICH, M.D.

Neurointerventional Service, Departments of Radiology and Neurosurgery, New York University School of Medicine, New York, New York

Object. The aim of this study was to describe the application of a novel transarterial approach to curative embolization of complex intracranial dural arteriovenous fistulas (DAVF). This technique is particularly useful in patients harboring high-grade DAVFs with direct cortical venous drainage or for whom transvenous coil embolization is not possible because of limited sinus venous access to the fistula site due to thrombosis or stenotic changes.

Methods. Twenty-three DAVFs in 21 patients were treated using a transarterial N-butyl cyanoacrylate (NBCA) embolization technique with the aid of a wedged catheter. In all patients, definitive treatment involved two critical steps: 1) a microcatheter was wedged within a feeding artery, establishing flow-arrest conditions within the catheterized vessel distal to the microcatheter tip; and 2) NBCA was injected under these resultant flow-arrest conditions across the pathological arteriovenous connection and into the immediate draining venous apparatus, definitively occluding the fistula. Patient data were collected in a retrospective manner by reviewing office and inpatient charts and embolization reports, and by directly analyzing all procedural and diagnostic angiograms.

Eight patients presented with the principal complaint of tinnitus/bruit, five with intracranial hemorrhage, four with cavernous sinus syndrome, and one each with seizures, ataxia, visual field loss, and hiccups. The parent (recipient) venous structure of the DAVFs in this study included 11 leptomeningeal veins, eight transverse/sigmoid sinuses, three cavernous sinuses, and one sphenoparietal sinus. The NBCA permeated the arteriovenous shunt, perifistulous network, and proximal draining vein in all DAVFs. Occlusion was confirmed on postembolization angiography studies. No complication occurred in any patient in this series. There has been no recurrence during a mean follow up of 18.7 months (range 2–46 months).

Conclusions. Transarterial NBCA embolization with the aid of a wedged catheter in flow-arrest conditions is a safe and an effective treatment for intracranial DAVFs.

KEY WORDS • dural arteriovenous fistula • embolization • N-butyl cyanoacrylate • catheter

To cure a DAVF, its pathological arteriovenous connection must be eliminated. Proximal feeding artery ligation alone by using endovascular or surgical methods is insufficient because the arterial supply of the fistula is invariably reestablished through a plethora of collateral sources. Transvenous embolization has proven to be useful in treating many DAVFs, obliterating the fistula by sacrificing the recipient venous structure. In patients harboring high-grade DAVFs with direct cortical venous drainage or for whom transvenous access is limited due to venous sinus thrombosis or occlusion, however, transvenous embolization may be impossible and other treatment methods must be considered. We report on our experience with a transarterial technique in which NBCA is injected under flow-arrest conditions for the treatment of intracranial DAVFs.

Clinical Material and Methods

Patient Population

Data regarding consecutive intracranial DAVFs in patients treated between October 1996 and August 2002 were retrospectively collected by reviewing office and in-patient charts and embolization reports, and by directly analyzing all diagnostic and procedural angiograms. During this time period 32 patients with intracranial DAVFs were treated using endovascular techniques: 21 with transarterial injection of NBCA and 11 with transvenous sinus occlusion. The patients who had undergone transvenous sinus occlusion were excluded from further analysis. The mean patient age was 52.4 years (range 3–78 years) and 33% of the patients...
Transarterial NBCA embolization of DAVFs

were female (Table 1). Eight patients presented with tinnitus/bruit, five with intracranial hemorrhage, four with cavernous sinus syndrome, and one each with seizures, ataxia, visual field loss, and hiccups. Two patients had previously undergone (incomplete) treatment of their DAVFs at other institutions: one, transvenous sinus occlusion; the other, transarterial PVA particles. All patients underwent a six-vessel diagnostic angiography study before and after embolization to confirm occlusion of their DAVFs. One patient harbored three separate DAVFs. For descriptive statistical analysis, the DAVFs were classified according to the Cognard grading scale and clinical outcomes were stratified based on the Glasgow Outcome Scale. 13

Transarterial Embolization Technique

All procedures were performed in patients after induction of general endotracheal anesthesia. To access arteries for diagnostic angiography studies and embolizations we used a No. 5 or 6 French catheter appropriate for each specific case. A variety of guidewire-directed microcatheters were used, including the Prowler-10, Prowler-14, and Rapid-Transit (Cordis Neurovascular, Miami Lakes, FL) as well as the Excel and Renegade (Boston Scientific, Fremont, CA). Polymeric embolic agents (NBCA) used in this series included Histoacryl (Yocan, Toronto, ON, Canada) and TruFill (Cordis Neurovascular). In the final six cases we switched from Histoacryl to TruFill because of its commercial availability. The volume of NBCA in Ethiodol was approximately 30 to 35% for TruFill and 20 to 25% for Histoacryl. This difference in percent volume was due to subtle differences in polymerization characteristics between the two preparations of NBCA.

Prior to microcatheterization, several appropriate arterial conduits for flow-arrest delivery were identified based on their size, length, accessibility, tortuosity, extent and type of collateralization, and flow rate. Once a feeding artery was selected, the microcatheter was advanced distally until the catheter was wedged into position. Confirmation of the flow-arrest status as well as the location and character of the targeted fistula was achieved with the aid of a microinjection of contrast agent. The microcatheter and arterial territory distal to the microcatheter tip was then flushed with D5W. Because D5W impedes polymerization of NBCA, the wedged feeding artery situated distal to the microcatheter tip was essentially converted into a functional extension of the catheter itself. In a controlled manner, NBCA was subsequently injected across the pathological arteriovenous fistula and into the parent venous apparatus, thereby occluding the lesion (Fig. 1).

In several high-flow DAVFs with extensive collateralization, inflow was diminished by preparatory embolization of accessory pedicles with the use of PVA particles (Cordis Neurovascular). These preparatory embolizations were performed specifically to optimize the subsequent injection of glue through the wedged catheter. Improvement in the subsequent process of glue injection was achieved by decreasing the competing inflow into the shunt domain, which, if unchecked, frequently causes fragmentation of the glue column and thus leads to premature polymerization and incomplete closure of the shunt. Furthermore, by preemptively reducing overall arteriovenous shunting, unintended systemic embolization of NBCA was prevented and conditions favoring slow, controlled delivery of glue to the targeted fistula were created. Although PVA particles are inadequate for the definitive occlusion of DAVFs, they are useful adjuvants for safely and acutely reducing collateral participation in the shunt supply. Polyvinyl alcohol is an embolic agent infrequently associated with ischemic cranial neuropathies, providing predictable collateral vessel penetration determined by the particle size selected (usually 45–250 μm).

Results

Angiography Evaluation

The parent (recipient) venous structure of the DAVFs in our cohort included 11 leptomeningeal veins, eight transverse/sigmoid sinuses, three cavernous sinuses, and one sphenoparietal sinus. All 23 DAVFs were supplied by multiple arteries. Based on angiography analysis, a mean 4.1 potential arterial conduits (range two–nine conduits) were identified as suitable for NBCA embolization. Nineteen (83%) of the DAVFs exhibited cortical venous drainage. Seven patients (33%) had a thrombosed major dural sinus. Cognard grades were as follows: one Grade I, three Grade II, seven Grade IIB, four Grade III, and eight Grade IV.

Treatment and Outcome

The NBCA permeation of the shunt with occlusion of the immediate venous apparatus was observed in all DAVFs treated in this series. No residual arteriovenous shunting was demonstrated on postembolization six-vessel control angiography studies of any DAVF following adequate deposition of NBCA into the recipient venous outlet. Figures 1 to 3 illustrate representative cases. Twenty of 21 patients underwent one embolization session each, whereas one patient underwent two sessions. This latter patient had three separate DAVFs, and because of the complexity of her case, which required therapeutic catheterization of both ophthalmic arteries, the embolizations were staged in two sessions to treat all three fistulas safely.

The arterial conduits for the definitive injection of NBCA were selected from branches of 10 middle meningeal arteries, five ascending pharyngeal arteries, two occipital arteries, one posterior meningeal artery, one artery of the foramen rotundum, one accessory meningeal artery, and two ethmoid divisions of the ophthalmic artery. In 16 DAVFs (70%), NBCA was successfully deposited across the pathological arteriovenous connection into the recipient venous structure on the initial injection attempt. The remaining seven DAVFs (30%) required more than one injection attempt to deposit NBCA through the fistulous connection: four cases required attempts through two pedicles, two required three, and one required four. Of the 11 NBCA injection attempts that did not initially result in adequate permeation of the fistula, seven were attributable to premature polymerization of NBCA proximal to the shunt and four resulted from inadequate occlusion of the recipient venous apparatus due to fragmentation of the glue stream arising from excessive collateral inflow. Three high-flow DAVFs were treated with preparatory devascularization of collateral inflow by using PVA particles before the definitive transarterial NBCA deposition.

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**TABLE 1**

*Summary of characteristics in 21 patients harboring 23 DAVFs treated with transarterial NBCA embolization*

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs), Sex</th>
<th>Presenting Symptoms</th>
<th>Cognard Grade</th>
<th>Parent Venous Structure†</th>
<th>Definitive NBCA Injection</th>
<th>Other Embolizations</th>
<th>Outcome</th>
<th>Follow Up (mos)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>32, M</td>
<td>rt tinnitus</td>
<td>IIA</td>
<td>rt ascending PhA, rt OA</td>
<td>rt SS</td>
<td>none</td>
<td>neuro intact, no tinnitus or residual DAVF</td>
<td>38</td>
</tr>
<tr>
<td>2</td>
<td>67, M</td>
<td>rt tinnitus</td>
<td>IV</td>
<td>lt MHT, lt MMA, lt OA</td>
<td>ptomesencephalic vein (sagittal sinus occl)</td>
<td>lt MMA</td>
<td>meningohypophyseal artery - PVA, lt OA - NBCA</td>
<td>36</td>
</tr>
<tr>
<td>3</td>
<td>42, M</td>
<td>seizures</td>
<td>IIB</td>
<td>lt inferolat trunk, lt accessory meningeal artery, lt artery of foramen rotundum, lt MMA</td>
<td>sphenoparietal sinus</td>
<td>lt MMA</td>
<td>accessory meningeal artery - NBCA</td>
<td>16</td>
</tr>
<tr>
<td>4</td>
<td>78, M</td>
<td>hemorrhage</td>
<td>IV</td>
<td>bilat OA, bilat MMA, bilat ascending PhA, bilat pst meningeal artery, lt artery of falx cerebelli</td>
<td>superior vermian vein</td>
<td>lt pst meningeal artery</td>
<td>none</td>
<td>14</td>
</tr>
<tr>
<td>5</td>
<td>33, M</td>
<td>rt tinnitus</td>
<td>IIA</td>
<td>lt OA, lt ascending PhA, lt deep cervical artery, bilat ascending PhA, rt inferolat trunk, rt MHT, rt MMA</td>
<td>lt TS (lt SS occl)</td>
<td>lt OA</td>
<td>none</td>
<td>17</td>
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<tr>
<td>6</td>
<td>71, M</td>
<td>pt proptosis, rt chemosis, rt visual loss</td>
<td>IIB</td>
<td>rt artery of foramen rotundum, rt inferolat trunk, rt MHT, rt MMA</td>
<td>rt CS (lt infr petrosal sinus occl)</td>
<td>rt artery of foramen rotundum PhA - NBCA</td>
<td>none</td>
<td>33</td>
</tr>
<tr>
<td>7</td>
<td>3, M</td>
<td>lt mastoid bruit</td>
<td>IIA</td>
<td>rt OA, rt ascending PhA</td>
<td>rt TS (rt SS occl)</td>
<td>rt OA</td>
<td>none</td>
<td>21</td>
</tr>
<tr>
<td>8</td>
<td>69, F</td>
<td>dysphasia, ataxia</td>
<td>IV</td>
<td>bilat ant falcial artery, bilat superficial temporal artery, bilat sphenopalatine artery, lt MMA</td>
<td>medial frontal cortical vein</td>
<td>ethmoidal division of rt OA</td>
<td>rt sphenopalatine artery - PVA, lt MMA - NBCA, lt OA - NBCA</td>
<td>22</td>
</tr>
<tr>
<td>9</td>
<td>71, F</td>
<td>lt tinnitus</td>
<td>IIB</td>
<td>lt MHT, lt OA, lt pst auricular artery, lt superficial temporal artery, lt ascending PhA, lt MMA</td>
<td>lt TS</td>
<td>lt MMA</td>
<td>none</td>
<td>19</td>
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<tr>
<td>10</td>
<td>55, M</td>
<td>hiccups</td>
<td>III</td>
<td>lt MMA, lt ant falcial artery, lt ascending PhA, lt MHT, lt OA</td>
<td>frontonatal convexity vein</td>
<td>lt MMA</td>
<td>none</td>
<td>16</td>
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<tr>
<td>11</td>
<td>32, M</td>
<td>rt tinnitus, vertigo</td>
<td>I</td>
<td>rt ascending PhA, rt SSA, rt MHT, rt MMA, rt pst meningeal artery</td>
<td>rt ascending PhA</td>
<td>none</td>
<td>none</td>
<td>46</td>
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<td>12</td>
<td>59, M</td>
<td>hemorrhage</td>
<td>IV</td>
<td>bilat MHA, bilat OA</td>
<td>parasagittal cortical vein</td>
<td>lt MMA</td>
<td>rt MMA - NBCA</td>
<td>14</td>
</tr>
<tr>
<td>Case No.</td>
<td>Age (yrs)</td>
<td>Sex</td>
<td>Presenting Symptoms</td>
<td>Cognard Grade</td>
<td>Arterial Supply</td>
<td>Parent Venous Structure</td>
<td>Definitive NBCA Injection</td>
<td>Other Embolizations</td>
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<td>---------</td>
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<td>-------------------</td>
</tr>
<tr>
<td>13</td>
<td>44, M</td>
<td>hemianopia</td>
<td>IV</td>
<td>rt MMA, rt OA, rt artery of falx cerebelli</td>
<td>rt deep pst temporal vein</td>
<td>rt artery of falx cerebelli</td>
<td>rt MMA - NBCA, rt OA - NBCA, rt ascending PhA - NBCA</td>
<td>neuro intact, no residual DA VF</td>
</tr>
<tr>
<td>14</td>
<td>61, F</td>
<td>rt proptosis, rt chemosis, rt visual loss</td>
<td>IIB</td>
<td>rt artery of foramen rotundum, bilat ascending PhA</td>
<td>rt CS (rt inf petrosal sinus occl)</td>
<td>rt ascending PhA</td>
<td>none</td>
<td>improved ocular findings, no residual DA VF</td>
</tr>
<tr>
<td>15</td>
<td>66, F</td>
<td>hemorrhage, hemianopia</td>
<td>III</td>
<td>rt MMA, rt MHT</td>
<td>rt deep pst temporal vein</td>
<td>rt MMA</td>
<td>rt MMA - PVA</td>
<td>baseline visual field loss, no residual DA VF</td>
</tr>
<tr>
<td>16</td>
<td>77, F</td>
<td>rt proptosis, rt chemosis, rt visual loss</td>
<td>IV</td>
<td>rt artery of foramen rotundum, rt accessory meningeal artery, bilat ascending PhA, rt inferolat trunk, rt MMA, lt MHT, rt artery of falx cerebelli, rt pst meningeal artery</td>
<td>rt deep sylvian vein</td>
<td>rt ascending PhA</td>
<td>rt MMA - NBCA, rt accessory meningeal artery - NBCA</td>
<td>improved ocular findings, no residual DA VF</td>
</tr>
<tr>
<td>17</td>
<td>41, M</td>
<td>hemorrhage, seizure</td>
<td>IIB</td>
<td>rt OA, rt ascending PhA, rt MMA, rt MHT, rt artery of falx cerebelli, rt OA</td>
<td>rt TS</td>
<td>rt MMA</td>
<td>none</td>
<td>neuro intact, no residual DA VF</td>
</tr>
<tr>
<td>18</td>
<td>57, M</td>
<td>rt proptosis, rt chemosis</td>
<td>IIB</td>
<td>rt artery of foramen rotundum, rt accessary meningeal artery, rt artery of foramen rotundum</td>
<td>rt CS</td>
<td>rt accessory meningeal artery</td>
<td>none</td>
<td>improved ocular findings, no residual DA VF</td>
</tr>
<tr>
<td>19</td>
<td>46, F</td>
<td>tinnitus</td>
<td>IIB</td>
<td>Cl &amp; C2 branch of rt VA, rt ascending PhA, rt MMA, rt OA</td>
<td>rt SS (rt TS occl)</td>
<td>rt ascending PhA</td>
<td>none</td>
<td>neuro intact, no tinnitus or residual DA VF</td>
</tr>
<tr>
<td>20</td>
<td>50, M</td>
<td>hemorrhage</td>
<td>IV</td>
<td>bilat OA, lt MMA</td>
<td>lt medial frontal cortical vein</td>
<td>ethmoidal division of rt OA</td>
<td>none</td>
<td>neuro intact, no residual DA VF</td>
</tr>
<tr>
<td>21</td>
<td>47, F</td>
<td>tinnitus</td>
<td>IV</td>
<td>lt MMA, lt OA, lt ascending PhA</td>
<td>lt SS (lt SS occl, rt TS occl)</td>
<td>lt MMA</td>
<td>none</td>
<td>neuro intact, no residual DA VF</td>
</tr>
</tbody>
</table>

* ant = anterior; CS = cavernous sinus; inf = inferior; MHT = meningohypophyseal trunk; MMA = middle meningeal artery; neuro = neurologically; OA = occipital artery; occl = occlusion; OphA = ophthalmic artery; PhA = pharyngeal artery; pst = posterior; SS = sigmoid sinus; TS = transverse sinus.

† Occluded venous sinuses are listed in parentheses.
No complication occurred in this series. All 21 patients had a Glasgow Outcome Scale score of 5 when discharged from the hospital. The average clinical follow up was 18.6 months (range 2–46 months). No patient developed symptoms referable to the DAVF during the follow-up period. In 12 patients without manifestations that could be clinically monitored (for example, tinnitus or cavernous sinus syndrome), follow-up diagnostic angiograms obtained at 6 to 12 months postembolization revealed no evidence of DAVF recurrence.
Discussion

Our patients were treated with a novel transarterial flow-arrest embolization technique in which diluted NBCA was slowly injected through a wedged catheter, previously advanced via a feeding artery, across the fistula and into the parent venous structure, and thus eliminating the pathological arteriovenous connection. As initially described for the treatment of brain arteriovenous malformations,5 with this technique the arterial pedicle beyond the tip of the wedged microcatheter becomes a functional extension of the catheter, establishing an effective embolization point closer to the fistula, thereby facilitating both delivery of the liquid embolic agent across the fistula site and permeation of the perifistulous collateral network. Recanalization does not occur if the glue cast traverses the fistulous connection and oc-

Fig. 2. Case 4. Angiography studies obtained in a 78-year-old man presenting with an intracranial hemorrhage. A: Left vertebral artery (VA) injection, lateral view, demonstrating a DAVF draining directly into an ectatic superior vermian vein. Note the associated aneurysmal venous varix (arrow). B: Contrast injection delivered through a microcatheter (large arrow) wedged within the left posterior meningeal artery (arrowheads), lateral view, revealing the pathological shunting point (small arrow). C: Left posterior meningeal artery microinjection, frontal view, demonstrating venous congestion of the right cerebellar hemisphere (arrows). D: Lateral roadmap image of the glue cast following embolization of the DAVF through the left posterior meningeal artery. The NBCA permeated the immediate parent venous structure (arrowheads). E: Postembolization left VA injection, lateral view, exhibiting DAVF occlusion. A complete six-vessel postembolization angiography study demonstrated occlusion of the DAVF (not shown).
cludes the immediately receptive venous apparatus. Use of this embolization technique has not been previously described in the treatment of DAVFs.

In our view, in certain situations the permeation of DAVFs with a liquid polymer delivered transarterially is preferable to transvenous coil embolization for the following reasons: 1) the fistula site is definitely occluded, reducing the likelihood that shunt flow may be diverted into alternative venous pathways and predisposing it to intraparenchymal hemorrhage; 2) treatment is not limited by venous access problems, including thrombosed or stenotic dural sinuses and high-grade lesions draining directly into cortical veins; 3) cure does not necessarily require the sacrifice of functional venous pathways that may drain normal brain parenchyma; 4) specific complications of transvenous embolization may be avoided in certain circumstances (for example, catheterization of the inferior petrosal sinus, which is often performed for transvenous access to the cavernous sinus and tentorial DAVFs, may cause sixth nerve palsy or venous rupture producing subarachnoid hemorrhage); and 5) development of de novo DAVFs in separate locations after transvenous coil embolization may occur due to venous hypertension–induced angiogenesis from coil occlusion of major dural sinuses. Recently, there have been multiple reports in which authors specifically describe intracranial DAVFs as being completely treated with the use of transvenous parent sinus occlusion, but later complications occurred by the development of a de novo DAVF at a separate intracranial location.

The radiographic and clinical outcomes in the present study compare favorably with those of other published series in which investigators used transvenous embolization or open surgical techniques. Although our obliteration rate was higher than those previously reported with earlier transarterial techniques, prior series generally included transarterial coil-, PVA-, and poly-

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**FIG. 3.** Case 14. Angiography studies obtained in a 59-year-old man presenting with an intracranial hemorrhage. A: Left ECA injection, frontal view, demonstrating a DAVF draining into a parasagittal cortical vein (arrow). Marked venous hypertension of the right cerebral hemisphere is visualized. A division of the left middle meningeal artery was feeding the fistula (arrowheads). B: Contrast injection from the microcatheter (large arrow) wedged in the previously noted division of the left middle meningeal artery, frontal view, displays the pathological shunting point (small arrow). C: Left middle meningeal artery glue cast, unsubtracted frontal view. The NBCA permeated dural collateral vessels as well as the immediate parent venous apparatus (arrow). D: Postembolization left ECA injection, lateral view, demonstrating DAVF occlusion. A complete six-vessel angiography study revealed occlusion of the DAVF (not shown).
mer-based methods resulting in occlusion of arteries proximal to the fistula site, permitting later reestablishment of arteriovenous shunting through adjacent collateral networks.

To maximize the efficacy of each transarterial NBCA embolization, a catheter must be securely wedged into position and a true flow-arrest state must be established. Furthermore, the injection dynamic should be predicated on the catheter’s distance from the fistula, the shunting rate, the size of the feeding artery engaged, and the extent of collateralization present. Although all DA VFs in our series were obliterated with the transarterial injection of NBCA, 35% were treated using adjunctive embolizations with PVA or NBCA. All of these patients harbored high-flow shunts in which preemptive devascularization of the collateral inflow before definitive occlusion of the shunt with NBCA was performed to minimize fragmentation and premature polymerization of the definitive glue column, and to prevent uncontrolled systemic venous embolization with NBCA.

Possible complications associated with transarterial NBCA embolization of DA VFs may include ischemic cranial nerve palsies, transcerebral embolization into normal cerebral arteries, and systemic venous embolization with NBCA. Although none of these complications occurred in our series, 30% of cases required more than one attempt to cast glue across the pathological shunting point. Multiple attempts of injecting NBCA were required, particularly when the catheter’s wedged position deteriorated or was not achieved within the initially selected dural branch. Successive attempts, however, are generally possible due to the multiplicity of potential target arteries available for each DA VF (mean number of feeding arteries 4.1, range two–nine arteries). Moreover, although the unsuccessful NBCA depositions were not in and of themselves curative (reflecting proximal occlusion of specific branches participating in the supply of the shunt), they nevertheless served to reduce collateral inflow, making subsequent NBCA embolization attempts more likely to be successful in curing the DA VF. Although not encountered in our experience, rupture of the catheterized vessel during glue injection is possible. Injecting the polymer in a slow, controlled manner likely prevents this complication from occurring.

During the study period, 11 additional patients with DA VFs admitted to our institution were treated with transvenous coiling of the parent venous sinus. Our decision to use a transvenous approach in these select cases was determined by the specific angioarchitecture and flow characteristics of each DA VF. In our experience, a primary transve-

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**TABLE 2**

*Summary of cerebral DAVF treatment results reported in the literature*

<table>
<thead>
<tr>
<th>Treatment Modality</th>
<th>Authors &amp; Year</th>
<th>No. of Patients</th>
<th>Embolic Material</th>
<th>DAVF Obliteration</th>
<th>Complications</th>
<th>Mean Clinical Follow Up (mos)</th>
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<tbody>
<tr>
<td>transarterial embolization</td>
<td>Grossman, et al., 1985</td>
<td>7</td>
<td>PVA</td>
<td>2 of 7 complete, 3 of 7 delayed complete, 2 of 7 partial</td>
<td>none</td>
<td>19</td>
</tr>
<tr>
<td></td>
<td>Quisling, et al., 1986</td>
<td>5</td>
<td>PVA</td>
<td>1 of 5 complete, 4 of 5 partial</td>
<td>none</td>
<td>24</td>
</tr>
<tr>
<td></td>
<td>Halbach, et al., 1987†</td>
<td>22‡</td>
<td>IBCA, PVA, other</td>
<td>17 of 22 complete, 5 of 22 partial</td>
<td>1 mild stroke</td>
<td>31</td>
</tr>
<tr>
<td></td>
<td>Halbach, et al., 1987§</td>
<td>17‡</td>
<td>IBCA, PVA</td>
<td>10 of 17 complete, 6 of 17 partial</td>
<td>1 mild stroke, 1 visual field defect</td>
<td>19†</td>
</tr>
<tr>
<td>present study</td>
<td>Halbach, et al., 1989**</td>
<td>21</td>
<td>NBCA</td>
<td>23 of 23 complete, 5 of 11 partial</td>
<td>1 venous infarct</td>
<td>10‡</td>
</tr>
<tr>
<td></td>
<td>Halbach, et al., 1989††</td>
<td>13</td>
<td>coils, IBCA</td>
<td>9 of 13 complete, 4 of 13 partial</td>
<td>1 aphasia, 1 transient vertigo</td>
<td>15§</td>
</tr>
<tr>
<td></td>
<td>Yamashita, et al., 1993‡‡</td>
<td>16</td>
<td>coils, other</td>
<td>14 of 16 complete, 2 of 16 partial</td>
<td>3 worsened chemosis, 3 cranial nerve palsies, 1 epidural hematoma</td>
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<tr>
<td>transvenous embolization</td>
<td>Urtasun, et al., 1996</td>
<td>24</td>
<td>coils, NBCA</td>
<td>17 of 24 complete, 7 of 24 partial</td>
<td>1 transient dioplia, 1 nasal hypophasia, 1 delayed death</td>
<td>11</td>
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<tr>
<td>surgical management</td>
<td>Quinones, et al., 1997</td>
<td>12</td>
<td>coils (transorbital)</td>
<td>12 of 13 complete, 1 of 13 partial</td>
<td>1 stitch granuloma, 1 abduccus palsy</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>Roy, et al., 1997</td>
<td>24</td>
<td>coils, balloons, other</td>
<td>21 of 24 complete, 3 of 24 partial</td>
<td>6 cranial nerve palsies, 2 transient vertigo</td>
<td>13</td>
</tr>
<tr>
<td></td>
<td>Sundt, et al., 1983</td>
<td>24</td>
<td>NR</td>
<td>24 of 24 complete</td>
<td>1 SAH</td>
<td>NR</td>
</tr>
<tr>
<td></td>
<td>Collice, et al., 2000</td>
<td>34</td>
<td>preop PVA</td>
<td>34 of 34 complete</td>
<td>2 venous infarcts, 2 deaths</td>
<td>33</td>
</tr>
</tbody>
</table>

* IBCA = iso-butyl cyanoacrylate; NR = not reported; SAH = subarachnoid hemorrhage.
† Data obtained from Halbach, et al.11
‡ Patients treated with compression therapy alone or subsequent surgery were excluded from this table.
§ Data obtained from Halbach, et al.8
‖ Angiographic follow up was performed.
** Data obtained from Halbach, et al.10
†† Data obtained from Halbach, et al.9
‡‡ Data obtained from Yamashita, et al.28

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nous approach is useful in treating high-flow DAVFs with complex arterial supplies, for which parent venous sinus occlusion is unavoidable and the transvenous approach appears simpler and equally efficacious.

An alternative treatment in patients harboring high-grade DAVFs with direct cortical venous drainage is microsurgical disconnection of the fistula from its recipient venous structure. If a high-grade DAVF cannot be easily accessed via endovascular means or if the risk of embolization is thought to be higher than that for an open microsurgical approach (for example, some ethmoidal and marginal tentorial DAVFs), microsurgery is an effective option for definitive treatment or palliation of symptoms. In summary, using a multimodal approach including transarterial, transvenous, and microsurgical techniques, the majority of DAVFs can be successfully treated with minimal morbidity.

Conclusions

Transarterial NBCA embolization with the aid of a wedged catheter in flow-arrrest conditions is a safe and effective treatment for intracranial DAVFs. It is particularly useful in situations in which access to the draining venous structures is limited.

References


Manuscript received July 3, 2002. Accepted in final form November 25, 2002.

Address reprint requests to: P. Kim Nelson, M.D., Neurointerventional Service, New York University Medical Center, 560 First Avenue, Tisch Hospital, HE-208, New York, New York 10016. email: nelso01@popmail.med.nyu.edu.