Endovascular treatment of vertebral artery dissections and pseudoaneurysms

Van V. Halbach, M.D., Randall T. Higashida, M.D., Christopher F. Dowd, M.D., Kenneth W. Fraser, M.D., Tony P. Smith, M.D., George P. Teitelbaum, M.D., Charles B. Wilson, M.D., and Grant B. Hieshima, M.D.

Departments of Radiology and Neurological Surgery, University of California Hospitals, San Francisco, California

Sixteen patients with dissecting aneurysms or pseudoaneurysms of the vertebral artery, 12 involving the intradural vertebral artery and four occurring in the extradural segment, were treated by endovascular occlusion of the dissection site. Patients with vertebral fistulas were excluded from this study. The dissection was caused by trauma in three patients (two iatrogenic) and in the remaining 13 no obvious etiology was disclosed. Nine patients presented with subarachnoid hemorrhage (SAH), two of whom had severe cardiac disturbances secondary to the bleed. The nontraumatic dissections occurred in seven women and six men, with a mean age on discovery of 48 years. Fifteen patients were treated with endovascular occlusion of the parent artery at or just proximal to the dissection site. One patient had occlusion of a traumatic pseudoaneurysm with preservation of the parent artery. Four patients required transluminal angioplasty because of severe vasospasm produced by the presenting hemorrhage, and all benefited from this procedure with improved arterial flow documented by transcranial Doppler ultrasonography and arteriography.

In 15 patients angiography disclosed complete cure of the dissection. One patient with a long dissection of extracranial origin extending intracranially had proximal occlusion of the dissection site. Follow-up angiography demonstrated healing of the vertebral artery dissection but persistent filling of the artery above the balloons, which underscores the need for embolic occlusion near the dissection site. No hemorrhages recurred.

One patient had a second SAH at the time of therapy which was immediately controlled with balloons and coils. This patient and one other had minor neurological worsening resulting from the procedure (mild Wallenberg syndrome in one and minor ataxia in the second).

Symptomatic vertebral artery dissections involving the intradural and extradural segments can be effectively managed by endovascular techniques. Balloon test occlusion and transluminal angioplasty can be useful adjuncts in the management of this disease.

Key Words: dissecting aneurysm • vertebral artery • endovascular therapy • balloon test • coils • stroke

Vertebral artery dissections are increasingly recognized as a source of stroke and subarachnoid hemorrhage (SAH). Rarely, these lesions can present with signs and symptoms of an intracranial mass effect. Patients with spontaneous dissections affecting the extracranial segment of the vertebral artery commonly present with neck pain, transient ischemic attacks (TIA's), or stroke, most commonly lateral medullary syndromes, although embolic events have been reported in all posterior circulation territories. Traumatic dissecting aneurysms or pseudoaneurysms of the extradural segment can have a similar presenting course, but additionally can be associated with massive external hemorrhage or neck hematomas. In contrast, histological studies have shown the intradural segment of the vertebral artery to have a thinner media and adventitia, and dissections involving this segment are more likely to extend subadventitially, producing SAH. Rebleeding in dissecting aneurysms of the vertebral artery has been reported to occur in a range from 24% to 30%, and carries a high mortality. Recent autopsy data suggest that in patients who died from SAH the etiology was a vertebral artery dissecting aneurysm in 4.4% to 7.7% of cases.

A wide variety of treatment regimens have been advocated for dissections involving the extradural vertebral artery, including conservative therapy, antplatelet regimens, anticoagulation therapy, and surgical resection of the involved artery. Intradural
vertebral artery dissections have been treated conservatively in a few cases;\(^{13}\) however, the majority have undergone surgical therapy. Since dissecting aneurysms of the vertebral artery are usually fusiform, surgical recommendations have included proximal clip occlusion of the affected artery when the opposite vertebral artery was equal in size or larger.\(^{12}\) When the affected vertebral artery is dominant, wrapping is often advocated.\(^{6,35,43,50}\) Occlusion of the dominant vertebral artery and even both vertebral arteries can be tolerated in some instances;\(^{20,46}\) therefore, balloon test occlusion has been advocated to evaluate tolerance prior to surgical ligation.\(^{47,52}\) We report our experience with the endovascular treatment of vertebral artery dissections in 16 patients.

**Clinical Material and Methods**

Sixteen patients with vertebral artery dissections were treated by endovascular techniques between July, 1987, and August, 1992. Patients with vertebral fistulas were excluded from this study. The patient's age, sex, presenting symptoms, dissection site, side affected, embolic device utilized, and length of follow-up evaluation are summarized in Table 1. The age at onset of symptoms ranged from 21 to 76 years (mean 50.3 years). Trauma produced three dissections in this series, two were iatrogenic injuries suffered at other hospitals (anterior cervical dissection in one, posterior fossa tumor approach in another). The nontraumatic dissections occurred in seven women and six men; their mean age at onset of symptoms was 48.3 years. Nine patients presented with SAH, two of whom developed severe cardiac disturbances secondary to the bleed (Cases 8 and 13). Three patients with trauma (Cases 4, 12, and 14) developed massive neck and chest hematomas requiring intubation. Two patients (Cases 3 and 11) had symptoms of mass effect produced by a partially thrombosed dissecting aneurysm pressing into the lower medulla. One patient (Case 2) had recurrent TIA's, and surgical exploration revealed a dissection of the intradural vertebral artery. The remaining patient (Case 5) suffered a series of strokes and TIA's despite antplatelet and anticoagulation therapy.

**Radiographic Evaluation**

All patients were evaluated with four-vessel digital subtraction angiography. In two patients, an initial arteriogram obtained elsewhere included only injection of the dominant vertebral artery, and the ruptured dissecting aneurysm on the proximal nondominant vertebral artery was not visualized. Compression views (Huber maneuver) were utilized in selected cases to evaluate potential collateral vessels from the anterior circulation (posterior communicating arteries). One patient (Case 6) had occlusion of the contralateral vertebral artery; however, test occlusion of the remaining dominant left vertebral artery was tolerated without incident. All endovascular procedures were performed with local anesthesia and light neuroleptic analgesia to allow continuous neurological monitoring. When the dissection involved the dominant vertebral artery or severe spasm was noted in the contralateral vertebral artery, a test occlusion with a nondetachable 0.85-mm silicone balloon\(^*\) was performed with the occlusion site adjacent to or just proximal to the dissection site. In four patients (Cases 1, 7, 13, and 15), severe vasospasm secondary to SAH was noted in the contralateral vertebral artery. Successful angioplasties of this vessel and other affected anterior and middle cerebral arteries were performed utilizing a special silicone angioplasty balloon. In all cases, transcranial Doppler ultrasound studies revealed a return to normal velocity of the dilated vessel, and angiography showed marked improvement in flow (see Illustrative Cases). One patient (Case 8) who developed rebleeding during treatment required delayed angioplasty.

**Endovascular Treatment**

Endovascular occlusion of the affected vertebral artery was performed in 15 cases. In one patient (Case 12), the affected vertebral artery was preserved. When the dissection site involved the extradural vertebral artery, the site chosen for test and permanent occlusion was just proximal to the origin of the dissection to minimize displacement of associated thrombus. When the dissection involved the intradural segment, the occlusion device was positioned at the dissection site to reduce the risk of occlusion of surrounding perforators and eliminate the risk of recanalization from collateral vessels. Detachable silicone balloons were utilized in seven patients (Cases 1, 2, 3, 5, 7, 10, and 11) as the sole embolic agent, and were combined with fibered platinum coils in three patients (Cases 8, 9, and 16). When occlusion was performed with two balloons, as

---

**TABLE 1**  
**Summary of vertebral artery dissections in 16 cases**

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>Presenting Symptoms</th>
<th>Side</th>
<th>Location</th>
<th>Embolic Device</th>
<th>Angioplasty</th>
<th>Follow-Up Period (mos)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>46</td>
<td>F</td>
<td>SAH</td>
<td>rt</td>
<td>l</td>
<td>balloons</td>
<td>yes</td>
<td>63</td>
</tr>
<tr>
<td>2</td>
<td>49</td>
<td>F</td>
<td>TIA's</td>
<td>rt</td>
<td>l</td>
<td>balloons</td>
<td>no</td>
<td>51</td>
</tr>
<tr>
<td>3</td>
<td>36</td>
<td>F</td>
<td>mass effect</td>
<td>rt</td>
<td>l</td>
<td>balloons</td>
<td>no</td>
<td>38</td>
</tr>
<tr>
<td>4</td>
<td>60</td>
<td>M</td>
<td>external bleed</td>
<td>lt</td>
<td>E</td>
<td>coils</td>
<td>no</td>
<td>34</td>
</tr>
<tr>
<td>5</td>
<td>21</td>
<td>M</td>
<td>stroke/TIA's</td>
<td>lt</td>
<td>E</td>
<td>balloons</td>
<td>yes</td>
<td>24</td>
</tr>
<tr>
<td>6</td>
<td>74</td>
<td>M</td>
<td>SAH</td>
<td>lt</td>
<td>l</td>
<td>coils</td>
<td>no</td>
<td>22</td>
</tr>
<tr>
<td>7</td>
<td>51</td>
<td>F</td>
<td>SAH</td>
<td>rt</td>
<td>l</td>
<td>balloons</td>
<td>yes</td>
<td>19</td>
</tr>
<tr>
<td>8</td>
<td>48</td>
<td>M</td>
<td>SAH</td>
<td>lt</td>
<td>l</td>
<td>balloons &amp;</td>
<td>yes</td>
<td>19</td>
</tr>
<tr>
<td>9</td>
<td>45</td>
<td>F</td>
<td>SAH</td>
<td>rt</td>
<td>l</td>
<td>balloons &amp;</td>
<td>yes</td>
<td>19</td>
</tr>
</tbody>
</table>

---

*SAH = subarachnoid hemorrhage; TIA = transient ischemic attack; L = intradural; E = extradural.

---

*Silicone balloons obtained from Interventional Therapeutic Corp., South San Francisco, California.

---

V. V. Halbach, et al.
Endovascular treatment of vertebral artery dissections

in Cases 2, 5, and 10, they were inflated with slightly hyperosmolar nonionic contrast material (concentration 200 mg% iopamidol or 300 mg% metrizamide). The balloons were delivered through two separate catheters and detached simultaneously to prevent balloon migration should one inadvertently deflate. When a single balloon was used for occlusion (Cases 3, 9, and 11), hydroxyethyl methacrylate was exchanged for the contrast material and allowed to solidify for 60 minutes prior to detachment. 20

Fibered platinum coils† were utilized in six patients (Cases 4, 6, 8, 9, 14, and 15), delivered through a variable-stiffness microcatheter with the aid of a coil pusher.‡ Electrolytically detachable coils were used in four patients (Cases 12, 13, 15, and 16), which permitted short segment occlusion of the dissection site.

All patients underwent close neurological evaluation following treatment. Neurological examinations were documented at 24-hour intervals until discharge. Late follow-up studies at 3 months, 6 months, and yearly intervals were also obtained. Following endovascular therapy, patients were observed in an intensive care unit, kept flat in bed for 24 hours, with volume expanded. Over the ensuing days, gradually increasing activity was permitted. Posttreatment arteriograms were obtained in all cases to assess complete occlusion. Late follow-up angiograms were obtained in 11 patients. The remaining five patients were evaluated with magnetic resonance (MR) imaging and/or angiography. Plain films were obtained in all cases to document the location of the embolic material.

Treatment Results and Complications

Four patients underwent successful transluminal angioplasty of severe vasospasm prior to treatment. Of the 15 patients who underwent deconstructive therapy (closure of the affected vertebral artery), complete occlusion of the dissection site was documented on late follow-up imaging studies in 14. The two patients with symptoms of mass effect had gradual alleviation of their presenting symptoms over 8 weeks and follow-up MR imaging revealed thrombosis and shrinkage of the aneurysm. One patient (Case 10), with a left vertebral artery dissection arising extracranially and extending intracranially, underwent balloon occlusion just proximal to the origin of the dissection site. A follow-up angiogram at 10 months demonstrated collateral filling of the distal vertebral artery above the balloons. The reconstituted vertebral artery remains normal in appearance, and clinically the patient is well 17 months following SAH.

One patient (Case 12) suffered a traumatic dissection in her dominant vertebral artery. Electrolytically detachable coils were navigated into the dissection site, occluding it, with preservation of the vertebral artery.

Two clinical complications were observed in this series. The first patient (Case 9) was a 45-year-old woman with an SAH from a dissection involving the origin of the right posterior inferior cerebellar artery (PICA). Balloon and coil occlusion resulted in mild gait difficulties and intermittent diplopia which resolved over the ensuing 2 months. Follow-up angiography revealed occlusion of the dissection and origin of the PICA but excellent collateral filling from the anterior inferior cerebellar artery. The second complication, discussed below, occurred in Case 8 when the patient rebled during balloon occlusion. Urgent occlusion of the dissection site with coils produced a partial Wallenberg syndrome which dramatically improved over the ensuing months. The reblooding during the procedure aggravated the patient's severe vasospasm, producing hemiparesis and declining mental status which improved with emergency angioplasty.

Illustrative Cases

Case 12

This 76-year-old woman suffered from multiple-level cervical spondylitis. During an anterior CS–7 discectomy at another hospital, severe intraoperative hemorrhage was encountered which was controlled with surgical packing. Postoperatively, a massive neck hematoma and bloody pleural effusion developed, requiring reintubation and transfusion of 11 units of blood over 24 hours. The patient was transferred to the University of California San Francisco (UCSF) Hospitals, where an emergency arteriogram demonstrated a traumatic dissecting aneurysm arising from an ectatic loop of the dominant left vertebral artery (Fig. 1A). The right vertebral artery was hypoplastic. The aneurysm was initially treated with two electrolytically detachable platinum coils and a small residual neck remnant was treated with an additional coil 2 days later. Computerized tomography (CT) showed the coils within the foramen transversarium (Fig. 1B). The patient's hematocrit stabilized and a follow-up arteriogram (Fig. 1C) obtained 2 weeks later confirmed complete closure of the dissection site and wide patency of her ectatic vertebral artery. She made a complete recovery.

Case 13

This 48-year-old woman developed severe headache, crushing substernal chest pain, and loss of consciousness; a subsequent ventricular tachycardia required cardioversion. She was initially thought to have a myocardial infarction; however, cardiac enzyme levels proved to be normal. A CT scan demonstrated SAH. Her initial neurological examination exhibited fixed and dilated pupils in an unresponsive patient without spontaneous respiration. An intracranial pressure (ICP) bolt revealed pressures of 18 to 20 cm H2O. An initial arteriogram of both internal carotid arteries and injection of the dominant vertebral artery were negative except for basilar vasospasm. Forty-eight hours after the initial SAH, a second hemorrhage occurred with the ICP increasing to 50 cm H2O. The patient was referred to the UCSF Hospitals for further workup and care.

Neurological examination on admission showed a

† Coils obtained from Cook Corp., Bloomington, Indiana, and Target Therapeutics, Inc., San Francisco, California.
‡ Tracker microcatheter and coil pusher obtained from Target Therapeutics, Inc., San Francisco, California.
Case 15

This 54-year-old man with a long history of hypertension and systemic atherosclerosis suffered an SAH. Angiography revealed a fusiform aneurysm arising from the dominant left vertebral artery above the origin of the PICA (Fig. 3A). Repeat angiography revealed severe vasospasm of the contralateral intradural segment of the right vertebral artery (Fig. 3B). Angioplasty of this segment was performed without incident (Fig. 3C). The left vertebral artery then tolerated a test occlusion and the fusiform aneurysm was occluded with electrolytically detachable coils (Fig. 3D). The patient was discharged 1 week later in excellent condition.
Endovascular treatment of vertebral artery dissections

Case 8

This 48-year-old man with a history of hypertension lost consciousness and became apneic, requiring cardio-pulmonary resuscitation. An initial cardiac workup including coronary angiography showed no abnormal findings. His neurological condition improved; however, he complained of severe headache, prompting a CT scan which revealed an SAH. An arteriogram revealed a dissecting aneurysm involving the dominant left vertebral artery (Fig. 4A and B). He was transferred to our institution for further evaluation.

On examination, the patient had a Grade II (Hunt & Hess) neurological condition with bilateral sixth nerve palsies and decreased sensation in his left leg. A repeat arteriogram (Fig. 4C) showed severe vasospasm involving the basilar, posterior cerebral, middle cerebral, and anterior cerebral arteries. A balloon was navigated through the left vertebral artery in an attempt to occlude the dissection site. While the balloon was being advanced toward the dissection site, the patient complained of severe headache. The balloon was immediately inflated and the headache stabilized. An angiogram revealed that the balloon had entered the dissection site and was in the subarachnoid space now, tamponading the bleeding site (Fig. 4D). The heparinization was immediately reversed and a second catheter was navigated to the dissection site and multiple coils placed across the dissection site, including the origin of the PICA. The balloon was then detached. The patient complained of new arm numbness. A CT scan confirmed an SAH, and over the next few hours the patient had decreasing mental status and new left hemiparesis. An emergency ventriculostomy revealed elevated pressures, but no improvement in neurological condition. An angioplasty of both internal carotid arteries and middle cerebral arteries produced immediate improvement in the patient's mental status and hemiparesis. Six days later, he developed increasing confusion requiring angioplasty of the right vertebral, basilar, right posterior cerebral (Fig. 4E and F), and both internal carotid and middle cerebral arteries. He improved dramatically except for unchanged incoordination of the left arm and leg and decreased sensation in the left foot. Over the ensuing 3 months, his strength returned to normal in the left arm and leg but he still has minimal dyscoordination of his right arm and decreased temperature and pain sensation in the left side of his body. He has returned to his former job as a construction worker. Skull films revealed that the balloon has migrated through the subarachnoid space to the C2-3 level (Fig. 4G).

Discussion

Presentation and Treatment Considerations

Dissecting vertebral artery aneurysms are rare but are increasingly recognized as a source of morbidity and mortality. Numerous studies have subclassified vertebral artery dissections according to their origin as either intracranial or extracranial. Spontaneous extracranial dissections most commonly occur in the distal one-third, usually at the C-1 and C-2 levels, where mechanical tension and stretch are frequently implicated. The dissection plane in these regions usually occurs between the intima and media and can produce spinal cord and brain ischemia by several mechanisms. The dissection site can serve as a source of thrombus formation producing TIA's, stroke, and even death. Spontaneous healing will occur in the majority of cases, as shown by Mokri, et al. Various therapeutic regimens have been advocated such as conservative therapy, wearing a soft cervical collar, antiplatelet and/or anticoagulation therapy, and surgical resection of the affected artery. Trauma can produce dissections by a variety of mechanisms. Nonpenetrating trauma, sometimes mi-
nor, has been implicated in dissection of the C-1 and C-2 segments, including tennis, yoga, use of a trampoline, and chiropractic manipulation. 12,16,37,38,41 Penetrating injuries can damage all three layers of the vertebral artery, often leading to giant neck hematomas, massive external bleeding, 22 or embolic stroke. 22 When all three layers of the vessel have been transgressed, only soft thrombus makes up the walls of the resulting pseudoaneurysm. Prior experience 21,22 has shown that traumatic pseudoaneurysms are easily managed with endovascular therapy, usually with trapping procedures. Occlusion of an acute pseudoaneurysm using embolic material, with preservation of the parent artery, is exceedingly difficult as the embolic material usually migrates into the soft thrombus containing the false aneurysm. The one exception is when the pseudoaneurysm is surrounded by bone or ligaments. The patient in Case 12 sustained an iatrogenic injury that damaged the dominant vertebral artery. The artery was preserved because the majority of the wall of the aneurysm was contained by surrounding bone. Therefore, traumatic fresh pseudoaneurysms of the second (intraforaminal) portion of the vertebral artery may be managed by such techniques.

The treatment of spontaneous (nontraumatic) vertebral dissections remains controversial. With some reports demonstrating dissections at other sites in 36% of patients and bilateral vertebral artery dissections in 21% of cases 36,42 coupled with reports of spontaneous healing in the majority (88%) of patients, 36 it seems that a deconstructive procedure is warranted only in a small minority of patients. However, our experience and that of others 11,43,52 have shown that some patients will experience repeated embolic events despite anticoagulation or antiplatelet regimens. The patient in Case 5 was a 21-year-old medical student who suffered two strokes and continued to have repeated TIA’s despite anticoagulation therapy. Serial MR imaging failed to reveal a dissection (a common occurrence in our series); however, angiography disclosed a dissection with a large intraluminal thrombus. Balloons deposited just proximal to the dissection site were successful in preventing further events. Our policy for the treatment of such extracranial dissections of both the vertebral and carotid arteries, which continue to produce emboli despite maximum medical therapy, is to deposit the balloon just proximal to the dissection site, thus reducing the risk of dislodging or displacing thrombus from the dissection. The vertebral artery, however, has multiple sources for collateral reconstitution through muscular branches from the external, contralateral vertebral, costocervical, and thyrocervical arteries. In Case 10 this was responsible for the only treatment failure in this series. Despite reconstitution of the vertebral artery at a level involved with the original dissection site (which extended intracranially), both MR imaging and angiographic findings suggested complete healing and the patient remains symptom-free 17 months following treatment. Such a case underscores the need for the occlusion site to be as close to the dissection site as possible, and trapping procedures should be considered for long segment dissections.

Intracranial vertebral artery dissections, and the rare extracranial dissection that extends to the intracranial segment, usually present with SAH. 42,52 With the dissection plane extending initially between the intima and media and eventually rupturing through the thinner adventitia. 42,51 The mean patient age at onset of intracranial dissections in the series reported by Yamaura, et al., 51 was 49.7 years, which corresponds closely with the 48.3-year average observed in our nontraumatic patients.

**Endovascular Therapy**

Our endovascular procedure for intracranial dissection varies from the treatment of extracranial dissection. After tolerance to test occlusion has been established, the embolic device is delivered to the dissection site, producing occlusion of the dissecting aneurysm and flow in the involved vertebral artery. Both balloons and coils were successful in achieving this goal. Solitary balloons are solidified with a hardening material, hydroxethyl methacrylate, 20 before detachment to prevent late deflation of the balloon. The electrotyically detachable coils, although initially designed for saccular aneurysms with narrow necks, work exceedingly well at producing occlusion of fusiform aneurysms such as dissecting aneurysms. To date, we have utilized this new tool for the treatment of 17 patients with fusiform aneurysms or fistulas and have encountered no documented distal emboli. These new coils also permit occlusion of a vessel in a relatively short segment (see Cases 13 and 15, above) unlike fibered platinum coils. In dissections involving the vertebral artery distal to the origin of the PICA, the shortest segment occlusion is the most desirable. Anatomical studies 31 have shown the plethora of critical perforator vessels arising from the distal vertebral arteries, especially the nondominant, usually right-sided vertebral artery. Interestingly, the series of Yamaura, et al., 51 (the largest series of surgically treated intracranial vertebral artery dissecting aneurysms), complications occurred in all three patients with clip occlusion of a dissection arising from the vertebral segment above the origin of the PICA. Overall, this complication represented 25% of patients who underwent clip occlusion or trapping procedures. Although not stated by the authors, it may be important to speculate on the possible etiology of these complications. It is possible that damage to the perforators occurred from surgical access or during application of the clip. More likely, progressive thrombosis occurred in the segment distal to the clip as the small perforator vessels provided inadequate supply to keep this segment open. Endovascular treatment may offer a theoretical advantage in permitting aggressive anticoagulation therapy following closure of the dissection site should delayed deficits develop. On the other hand, a surgical clip permits the shortest possible occlusion length, as all balloons and coils require a short segment for their stability.

Our most significant complication occurred when a deflated detachable balloon entered the dissection site, producing rebleeding. Prompt recognition and closure
Endovascular treatment of vertebral artery dissections

Fig. 4. Case 8. A and B: Left vertebral angiograms, anteroposterior (A) and lateral (B) projections, demonstrating a dissecting aneurysm arising from the distal left vertebral artery. C: Angiogram obtained during the balloon embolization procedure showing the inflated balloon (thick arrows) outside the confines of the parent vessel. The catheter is outlined by short arrows. D: Right vertebral arteriogram obtained after embolization showing multiple platinum coils occluding the vertebral dissection. The balloon remains adjacent to the vertebral artery (arrow). E: Right vertebral arteriogram demonstrating persistent severe vasospasm involving the basilar artery and posterior cerebral arteries. F: Right vertebral arteriogram obtained mid-angioplasty showing the improved caliber of the basilar and right posterior cerebral artery. G: Lateral plain skull film obtained 1 year after the procedure demonstrating unchanged platinum coils within the left vertebral artery dissection site. The detachable balloon has migrated inferiorly to the C-2 level (arrow).

of the bleeding site resulted in a good outcome; although the patient still suffers mild sensory deficits, the only residual effect is a partial Wallenberg syndrome related to closure of the PICA. Our experience with other vascular perforations occurring during neurointerventional procedures has shown that prompt recognition and closure of the perforation results in good clinical outcome in the majority of patients.

Surgical Therapy

Current surgical recommendations for the treatment of intracranial vertebral dissection includes clip occlusion or trapping procedures if the contralateral vertebral artery is equal or greater in caliber, and wrapping of the dissection if the affected artery is dominant. Only a few patients who have undergone wrapping procedures for vertebral artery dissections have been reported in the literature; therefore, the effectiveness of this procedure is unknown. Data from patients with saccular aneurysms who have undergone wrapping procedures suggest that the rebleeding rate is lower than in patients with an untreated aneurysm but much higher than in those with aneurysms who have undergone surgical clipping. Extrapolation from these data suggests that occlusion of the dissection site, either by surgical clipping or endovascular occlusion, should carry a lower rebleeding rate than wrapping of the dissection site. Although our series is small, no rebleeding events have occurred in our treated patients. Our experience with test occlusion has shown that patients can often tolerate permanent occlusion of the dominant vertebral artery and, as in Case 6, bilateral vertebral artery occlusion without deficits. Angioplasty was a useful adjunct to improve flow in the collateral pathways prior to occlusion.

Both Yamaura, et al., and Su, et al., commented
on the usefulness of test occlusion in surgical planning. It must be emphasized that the site of test occlusion is critical. Proximal test occlusion of the vertebral artery is often tolerated due to the plethora of available surrounding collateral vessels. This, however, does not imply that distal occlusion of vertebral arteries will be tolerated. Therefore, test occlusion should be performed at or just proximal to the intended site of permanent occlusion. Su, et al., suggested that a double lumen balloon can be used in the mid-vertebral artery to evaluate tolerance to test occlusion during somatosenory evoked potentials recording. We believe that because of the extensive distal collateral network, distal test occlusion will be more predictable and we prefer to perform the more complete neurological assessment permitted by neuroleptic analgesia.

Because of the high risk of rebleeding, we prefer to treat ruptured dissecting aneurysms as soon as possible. Currently, those patients treated soon after a hemorrhage are followed closely with transcranial Doppler ultrasound studies and frequent neurological monitoring to detect symptoms or signs suggestive of progressive vasospasm. If severe spasm is noted on the initial angiogram, we prefer to undertake angioplasty of the collateral pathways prior to endovascular occlusion.

Our results in this series compare favorably with the published surgical results for the treatment of vertebral dissections. The majority of our intracranial dissections arose above the level of the PICA, which has been shown in surgical series to be a more difficult region for treatment with clip occlusion.\(^\text{22}\) The addition of a balloon test occlusion, sometimes with transluminal angioplasty of collateral pathways, should more accurately assess which patients can safely undergo occlusion, rather than the anatomical criteria that have previously been suggested.\(^\text{22}\) Endovascular occlusion techniques may offer a therapeutic alternative in the management of this difficult disease.

References


V. V. Halbach, et al.
Endovascular treatment of vertebral artery dissections


Manuscript received September 21, 1992.
Accepted in final form January 27, 1993.
Address reprint requests to: Van V. Halbach, M.D., 505 Parnassus Avenue, L352, San Francisco, California 94143–0628.