Extracranial Vertebral Artery Dissections: A Review of 13 Cases

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Clinical and radiologic findings in 13 patients (11 women, 2 men) with extracranial vertebral artery dissection are reported. Dissection was spontaneous in 8 patients, occurred after neck manipulation in 2 and after a potential minor injury to the neck in 3. Six had a history of common migraine, 4 were using oral contraceptives at the time of dissection, and 3 had fibromuscular dysplasia. Dissection was bilateral in 8 patients and associated with carotid dissection in 3. It usually presented with neck or occipital pain preceding basilar ischemic symptoms by a few minutes to 1 month. In 3 patients, transient ischemic attacks were the only manifestation of basilar ischemia, and in 1 patient there was no symptom of basilar ischemia despite bilateral vertebral dissection. In 19 of the 21 dissected vertebral arteries, the angiographic appearance was that of an irregular stenosis, which was associated in 6 arteries with pseudoaneurysmal formation. In 2 patients, 1 vertebral artery was occluded but the contralateral artery showed the typical irregular stenosis. The dissection involved only the third segment in 33%, only the second segment in 24%, and 2 or more segments in 38%. Eleven patients were treated with anticoagulants and 2 with aspirin; 11 recovered without sequelae and 2 had residual deficit. No recurrence was observed (mean follow-up 34 months). At control angiography (n = 12) or ultrasonic study (n = 1), 63% of dissected vertebral arteries had returned to normal, 26% showed marked improvement, and 11% were occluded. Our patient characteristics are compared with those of previously published cases. The validity of the distinction between spontaneous dissection and dissection associated with minor trauma is discussed. (Stroke 1987;18:1037-1047)

Extracranial vertebral artery dissection is recognized as a cause of stroke with increasing frequency.1-3 However, the clinical and radiologic features of this entity and its natural history are less well known than those of carotid dissection.4

The diagnosis of dissection relies on angiography, which reveals irregular stenosis with or without pseudoaneurysm,5-7 double lumen,12 or occlusion5 of 1 or both extracranial vertebral arteries. Of these three features, the only pathognomonic one is the double lumen. Stenosis is a less specific cause, but becomes suggestive when it is irregular and involves segments of the vertebral artery usually not involved by atheroma. Occlusion is the least specific since it can be due to thrombosis of any cause.3 We were interested in studying nonocclusive forms of extracranial vertebral dissection and report here on 13 such patients with special emphasis on the role of trauma, the long-term outcome of these patients, and the evolution of the arterial lesions.

Subjects and Methods

Thirteen consecutive patients (mean age 38.2 years, range 26-48) with angiographically diagnosed extracranial vertebral artery dissection were observed from November 1980 to November 1986 in one neurology department. Patients whose angiogram showed only vertebral artery occlusion were excluded. Cases 3 and 4 have already been published,2 and 4 others (Cases 1, 6, 7, and 8) have been described angiographically.10

Patients were specifically asked about history of migraine (defined according to the Ad Hoc Committee on Classification of Headache11), high blood pressure, diabetes mellitus, hypercholesterolemia, smoking, and past or current use of oral contraceptives. Particular attention was paid to any unusual neck motion or trauma that could have happened in the previous weeks, days, or hours as well as to all sports activities or strenuous exercises performed in the same period.

The clinical findings are reported in Table 1. All patients had blood cell count, sedimentation rate, protein electrophoresis, fasting serum glucose and cholesterol, coagulation study (prothrombin time, activated partial thromboplastin time, platelet count), electrocardiogram, chest x-ray, brain computed tomography (CT) scan, and cerebral angiography. The location of the arterial stenosis on the angiograms was defined according to the following segmentation of the vertebral artery: first segment, V1, from its origin to its entry into the foramen transverse of C6; second segment, V2, from the foramen of C6 to that of C2; third segment, V3, from C2 to the dura; and fourth segment, V4, intracranial vertebral artery. The angiographic findings are reported in Table 2.

All patients had regular clinical follow-up until November 1986. Evolution of the arterial lesions was
Table 1. Clinical Findings of Presently Reported Cases

<table>
<thead>
<tr>
<th>Case</th>
<th>Sex</th>
<th>Age</th>
<th>History</th>
<th>Neck trauma</th>
<th>Neck or head pain</th>
<th>Ischemic signs</th>
<th>Treatment</th>
<th>Immediate outcome</th>
<th>Follow-up Length (months)</th>
<th>Recurrence</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>F</td>
<td>48</td>
<td>High BP</td>
<td>No</td>
<td></td>
<td>VB TIA</td>
<td>AC (3 yr)</td>
<td>Favorable</td>
<td>69</td>
<td>No</td>
</tr>
<tr>
<td>2</td>
<td>F</td>
<td>36</td>
<td>Migraine, past user of OC</td>
<td>No</td>
<td></td>
<td>+ L lateral medullary (NDMS)</td>
<td>ASA</td>
<td>Favorable</td>
<td>57</td>
<td>No</td>
</tr>
<tr>
<td>3*</td>
<td>F</td>
<td>40</td>
<td>High BP, current user of OC</td>
<td>No</td>
<td>+</td>
<td>VB TIA, bruit, Horner's syndrome</td>
<td>AC (7 mo)</td>
<td>ASA</td>
<td>51</td>
<td>No</td>
</tr>
<tr>
<td>4†</td>
<td>F</td>
<td>42</td>
<td>Current user of OC, FMD</td>
<td>No</td>
<td>+</td>
<td>VB TIA, carotid NDMS</td>
<td>AC (1 yr)</td>
<td>ASA</td>
<td>53</td>
<td>No</td>
</tr>
<tr>
<td>5</td>
<td>M</td>
<td>36</td>
<td></td>
<td>Dubious</td>
<td></td>
<td>Locked-in syndrome (MS)</td>
<td>AC (5 mo)</td>
<td>Sequelae</td>
<td>50</td>
<td>No</td>
</tr>
<tr>
<td>6</td>
<td>F</td>
<td>32</td>
<td>Pregnancy, past user of OC, FMD</td>
<td>No</td>
<td>+</td>
<td>L lateral medullary (NDMS)</td>
<td>AC (9 mo)</td>
<td>Favorable</td>
<td>39</td>
<td>No</td>
</tr>
<tr>
<td>7</td>
<td>F</td>
<td>26</td>
<td>Past user of OC, smoking abuse</td>
<td>No</td>
<td>+</td>
<td>Diffuse brainstem (no definite syndrome)</td>
<td>AC (6 mo)</td>
<td>Favorable</td>
<td>38</td>
<td>No</td>
</tr>
<tr>
<td>8</td>
<td>F</td>
<td>27</td>
<td>Past user of OC, postpartum migraine</td>
<td>Neck manipulation</td>
<td>+</td>
<td>VB TIA, brainstem (no definite syndrome)</td>
<td>AC (9 mo)</td>
<td>Favorable</td>
<td>36</td>
<td>No</td>
</tr>
<tr>
<td>9†</td>
<td>F</td>
<td>42</td>
<td>Current user of OC, premenstrual headaches</td>
<td>No</td>
<td>+</td>
<td>No VB symptoms, carotid TIA, NDMS</td>
<td>AC (6 mo)</td>
<td>Favorable</td>
<td>20</td>
<td>No</td>
</tr>
<tr>
<td>10</td>
<td>F</td>
<td>43</td>
<td>Migraine, current user of OC, smoking abuse</td>
<td>No</td>
<td>+</td>
<td>Lower brainstem (MS)</td>
<td>ASA</td>
<td>Sequelae</td>
<td>12</td>
<td>No</td>
</tr>
<tr>
<td>11</td>
<td>F</td>
<td>47</td>
<td>Migraine</td>
<td>Neck manipulation</td>
<td>+</td>
<td>Vestibular syndrome (ms)</td>
<td>AC (6 mo)</td>
<td>Favorable</td>
<td>9</td>
<td>No</td>
</tr>
<tr>
<td>12</td>
<td>M</td>
<td>38</td>
<td>Migraine, FMD</td>
<td>Dubious</td>
<td></td>
<td>+ L lateral medullary (NDMS)</td>
<td>AC (2 mo)</td>
<td>Favorable</td>
<td>6</td>
<td>No</td>
</tr>
<tr>
<td>13</td>
<td>F</td>
<td>40</td>
<td>Migraine, high BP</td>
<td>Dubious</td>
<td>+</td>
<td>VB TIA, upper brainstem (NDMS)</td>
<td>AC (3 mo)</td>
<td>Favorable</td>
<td>6</td>
<td>No</td>
</tr>
</tbody>
</table>

BP, blood pressure; TIA, transient ischemic attack; VB, vertebrobasilar; AC, anticoagulant; OC, oral contraceptives; NDMS, nondisabling major stroke; ASA, aspirin; MS, major stroke; FMD, fibromuscular dysplasia; ms, minor stroke.

*Concomitant bilateral internal carotid artery dissection.
†Concomitant unilateral internal carotid artery dissection.

Assessed with angiography in 10 patients, with angiography and ultrasonography in 2 patients, and with ultrasonography alone in 1 patient. The method of ultrasonography (continuous-wave Doppler and duplex scanning) of the vertebral arteries has previously been described,12 and detailed ultrasonic data on Patients 6, 7, and 8 are reported in another paper.13

**Patient 1**

A 48-year-old woman experienced bilateral blurred vision, dizziness, loss of balance, and decreased sensation in her left hand and foot on December 3, 1980. These symptoms disappeared within a few hours. Right brachial arteriography on December 5, 1980, showed an irregular stenosis of the second and proximal third segments of the right vertebral artery with an aneurysmal dilatation at the C2 level. The distal right vertebral artery was not opacified. The right carotid artery was normal. Anticoagulants were started on December 6, 1980, and stopped 8 days later. On the following day, she had numbness of the right side of her face and decreased sensation of her left limbs. These symptoms disappeared over a few hours and she was again given anticoagulants. Transfemoral arteriography on December 23, 1980, showed that the right vertebral artery was normal except for a mild residual stenosis at the C4–C5 level. Internal carotid and left vertebral arteries were normal. In October of 1983,
Table 2. Angiographic Findings of Presently Reported Cases

<table>
<thead>
<tr>
<th>Case</th>
<th>Vessel locus lesion</th>
<th>Vascular lesion</th>
<th>Control angiography</th>
<th>Time between angiograms</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>RVA: V2, V3 (distal RVA not opacified)</td>
<td>Irregular stenosis (C5-C1), aneurysmal dilatation (C2)</td>
<td>Mild residual stenosis (C4-C5)</td>
<td>18 days</td>
</tr>
<tr>
<td>2</td>
<td>LVA: V3, V4</td>
<td>Irregular stenosis, aneurysmal dilatation (C2)</td>
<td>Normal</td>
<td>4 yr, 8 mo</td>
</tr>
<tr>
<td>3*</td>
<td>RVA: V3, LVA: V2, V3</td>
<td>Severe stenosis (C1), Irregular stenosis (C6-C3), aneurysmal dilatation (C3-C2), sacciform aneurysm (C1)</td>
<td>Normal</td>
<td>3 mo</td>
</tr>
<tr>
<td>4†</td>
<td>LVA: V3</td>
<td>Severe stenosis (C2)</td>
<td>Normal</td>
<td>2 mo</td>
</tr>
<tr>
<td></td>
<td>RVA: V3, LVA: V3</td>
<td>Irregular stenosis</td>
<td>Normal</td>
<td>16 mo</td>
</tr>
<tr>
<td>5</td>
<td>RVA: V2, V3 (distal RVA not opacified)</td>
<td>Irregular stenosis (C6-C1), aneurysmal dilatation (C1)</td>
<td>Occlusion (US)</td>
<td>9 mo</td>
</tr>
<tr>
<td>6</td>
<td>LVA: V2, V3</td>
<td>Irregular stenosis (C6-C1)</td>
<td>Normal</td>
<td>3 mo</td>
</tr>
<tr>
<td>7</td>
<td>RVA: V2</td>
<td>Irregular stenosis (C5-C2)</td>
<td>Normal (US)</td>
<td>3 mo</td>
</tr>
<tr>
<td>8</td>
<td>RVA: V2, LVA: V2, V3</td>
<td>Occlusion (C6-C2)</td>
<td>Normal</td>
<td>3 mo</td>
</tr>
<tr>
<td>9†</td>
<td>RVA: V2, LVA: V2</td>
<td>Stenosis (C3), Irregular stenosis (C6-C2), severe stenosis (C2)</td>
<td>Normal</td>
<td>5 mo</td>
</tr>
<tr>
<td></td>
<td>RVA: V3, LVA: V2, V3</td>
<td>Irregular stenosis (C1), aneurysm (C1), Irregular stenosis (C5-C4), severe stenosis (C1)</td>
<td>Mild residual stenosis</td>
<td>7 days</td>
</tr>
<tr>
<td>10</td>
<td>RVA: V3</td>
<td>Irregular stenosis (C1), aneurysm (C1)</td>
<td>Normal</td>
<td>1 yr</td>
</tr>
<tr>
<td>11</td>
<td>RVA: V1, V3, V4</td>
<td>Irregular stenosis (C2 to intracranial VA), aneurysmal dilatation (C7)</td>
<td>Mild stenosis of intracranial VA</td>
<td>7 mo</td>
</tr>
<tr>
<td>12</td>
<td>LVA: V4</td>
<td>Occlusion (intracranial VA)</td>
<td>Occlusion</td>
<td>7 mo</td>
</tr>
<tr>
<td>13</td>
<td>LVA: V3</td>
<td>Irregular stenosis (C1)</td>
<td>Normal</td>
<td>2 mo</td>
</tr>
<tr>
<td></td>
<td>LVA: V3</td>
<td>Irregular stenosis</td>
<td>Slight irregularities</td>
<td>2 mo</td>
</tr>
</tbody>
</table>

RVA, right vertebral artery; LVA, left vertebral artery; US, ultrasonography.
*Concomitant bilateral internal carotid artery dissection.
†Concomitant unilateral internal carotid artery dissection.

while still on anticoagulants, she had a right capsular hemorrhage. Anticoagulants were stopped. No further symptom has occurred since then.

**Patient 2**

A 36-year-old woman suddenly developed pain and stiffness in her neck on December 3, 1981. Her stiff neck persisted over the next few days and, on December 15, she noted difficulty in swallowing, decreased temperature sensation over her right limbs, left ptosis, and about 1 hour later, severe vertigo and aphony. Bilateral brachial arteriography on January 7, 1982, revealed a severe stenosis of the third and fourth segments of the left vertebral artery with an aneurysmal dilatation at the C2 level. The right vertebral and internal carotid arteries were normal. She was given 1 g aspirin/day for 1 year. She recovered completely over the next few weeks and has been symptomless since then. In October of 1986, digitized intra-arterial arteriography showed a normal left vertebral artery.

**Patient 3**

A 40-year-old woman had acute onset of headache, neck pain, vomiting, and a bruit in her left ear on January 20, 1982. Ten days later she had a brief loss of consciousness. Four days after her loss of consciousness, she experienced a sudden episode of bilateral blurred vision, which lasted 5 minutes. On admission the next day, examination was normal except for a left Horner’s syndrome and a bruit over the left side of her neck. Transfemoral arteriography on February 10, 1982, revealed an irregular stenosis of the second segment of the left vertebral artery (from C6 to C3), with aneurysmal dilatation at the C3–C2 level, a sacciform aneurysm at the C1 level, and a severe stenosis of the third segment of the right vertebral artery at the C1 level. Both internal carotid arteries were severely narrowed, the right one from 1 cm above the origin up to the siphon and the left one at the C1-C2 level, with aneurysmal dilatation just below the entrance into the carotid canal. The renal arteries were normal. The patient was given anticoagulants for 7 months followed by 1 g aspirin/day. The bruit and headache disappeared gradually over 2 weeks. On May 5, 1982, digitized intravenous arteriography was normal except for the persistence of a sacciform aneurysm of the left internal carotid artery at the C1 level. The left vertebral artery was not properly opacified, but continuous-
wave Doppler of this artery was normal. She has been symptomless since then.

**Patient 4**

A 42-year-old woman experienced a 10-minute episode of bilateral blurred vision on February 20, 1982. A similar episode occurred the next day and was accompanied by severe neck pain, which lasted a week. On February 28, she suddenly heard a pulsatile bruit in her right ear and developed weakness and numbness of the left side of her body, which gradually improved. Fifteen days after onset, clinical examination was normal except for a slight impairment of joint position sense on her left side. Transfemoral arteriography on May 10, 1982, showed a severe stenosis of the third segment of the left vertebral artery at the C2 level. The right internal carotid artery was severely narrowed from 1 cm above the origin up to the entrance into the carotid canal, with an aneurysm at the C1 level. The left internal carotid artery was not selectively catheterized but looked extremely irregular on global arteriography. The right vertebral artery was normal. The right renal artery had a beaded appearance, suggestive of fibromuscular dysplasia. On the day after angiography, she suddenly developed a moderate dysphasia, which gradually improved. She was given anticoagu-

**Patient 5**

A 36-year-old man with neck pain for 3 days developed a locked-in syndrome over a few hours on March 31, 1982. Several days before the onset of neck pain, he had done heavy work in his home but could not recall any unusual neck movement. Transfemoral arteriography on March 31 showed a severe stenosis of the third portion of both vertebral arteries. Carotid arteries were normal. Anticoagulants were started immediately and replaced 5 months later by 1 g aspirin/day. He gradually improved but remains severely disabled with a right hemiparesis. On digitized intra-arterial arteriography performed on December 5, 1983, the vertebral, carotid, and renal arteries were normal.

**Patient 6**

A 26-year-old woman suddenly experienced severe neck pain on July 8, 1983, followed a few minutes later by paresthesia in both arms, on the upper part of her back, her neck, and her left tongue. She had difficulty moving her upper limbs and moderate dysarthria. All symptoms disappeared spontaneously over the next 3 days. Examination on July 21 disclosed only increased deep tendon reflexes and decreased sensation of her right thumb. Bilateral brachial arteriography on July 26, 1983, showed a severe and irregular stenosis of the second segment of the right vertebral artery and of the second and proximal third segments of the left vertebral artery with reinjection of the distal part of both vertebral arteries via muscular arteries. She was treated with anticoagulants for 6 months and has been symptomless since then. Ultrasonography performed on September 21, 1983, showed normal vertebral arteries.

**Patient 7**

A 27-year-old woman experienced pain in the right side of her neck on November 15, 1983, 2 weeks after a normal delivery. On November 21, she had a neck manipulation and the pain disappeared. She was perfectly well until December 31, 1983, when she suddenly experienced a 1-hour episode of paresthesia over her whole face with vertigo, aphonia, right hemiparesis, and vomiting. She recovered completely, but a few hours later she had a similar episode with horizontal diplopia and difficulty in swallowing. She improved again, but on admission 2 days later, right facial and hand weakness, incoordination of her right limbs, and minor dysarthria were still present. A few hours after admission, she experienced numbness of her face and left arm and a left hemiparesis, which disappeared completely in 1 hour. Transfemoral arteriography on January 6, 1984, showed normal carotid arteries. The right vertebral artery was occluded from C6 to C2 and was reinjected via anastomosis with muscular arteries. The left vertebral artery was moderately and irregularly stenosed from C6 to C2. She was given anticoagulants for 9 months, then 1 g aspirin/day. She recovered
completely over the next few weeks and has been symptomless since then. In April of 1984, digitized intra-arterial arteriography showed normal vertebral and renal arteries.

**Patient 9**

A 42-year-old woman with neck pain for 1 month on November 11, 1984, suddenly experienced a right hemiparesis and dysphasia, which disappeared in 2 hours. A few hours later, over 2 hours she developed a dense hemiplegia and severe dysphasia. CT scan showed a hypodensity in the left middle cerebral artery territory. Arch arteriography on November 19, 1984, revealed stenosis of the distal second segment (C3 level) of the right vertebral artery. The left vertebral artery was irregularly narrowed in its second segment and stenosed at the C2 level. This vessel was poorly visualized above C2, but its caliber looked normal. There was a tapered occlusion of the left internal carotid artery. The right carotid artery was normal (Figure 1, left). She was given anticoagulants for 6 months, then 1 g aspirin/day. She recovered completely over a few weeks and has been symptomless since then. At repeat transfemoral angiography on April 16, 1985, the right vertebral artery was normal; the left vertebral artery was still slightly irregular in its second segment, but the severe stenosis previously seen at the C2 level had disappeared (Figure 1, right). The left internal carotid artery remained occluded. Renal arteries were normal.

**Patient 10**

A 43-year-old woman suddenly experienced a right nuchal pain on October 10, 1985, which became bilateral a few days later. On October 22, 1985, she developed dizziness, dysarthria, difficulty in swallowing, and obnubilation over the course of 1 hour. Examination revealed increased deep tendon reflexes, left hemiplegia, left extensor plantar response, decreased sensation on the left side of her face, and...

**Figure 1.** Patient 9. Left: Arch arteriography, November 19, 1984. Stenosis of right vertebral artery at C3 level (large arrow), irregular stenosis of second segment of left vertebral artery with severe stenosis at C2 level (small arrows), tapered occlusion of left internal carotid artery (long arrow). Right: Transfemoral arteriography, April 16, 1985. Slight irregularities of second segment of left vertebral artery and disappearance of severe stenosis at C2 level.
absent gag reflex. Digitized intra-arterial arteriography on October 31, 1985, showed irregular stenosis of the third segment of the right vertebral artery with an aneurysm at the C1 level (Figure 2, top left); the left vertebral artery could not be opacified. Transfemoral arteriography on November 7, 1985, revealed mild residual stenosis of the right vertebral artery (Figure 2, top right), moderate stenosis of the proximal second segment, and severe eccentric stenosis of the third segment of the left vertebral artery (Figure 2, bottom left).

The carotid arteries were normal. She was given 0.5 g aspirin/day for 9 months and recovered incompletely. In November of 1986, she was admitted again because of sudden recurrence of nuchal pain. Examination was comparable with the previous one. A left brachial arteriography revealed a normal left vertebral artery (Figure 2, bottom right).

**Patient 11**
A 47-year-old woman, while pushing the cord of the phone with her foot on January 20, 1986, suddenly experienced a left occipital pain that worsened during the following hours. From January 21 to 27, 1986, she had 4 neck manipulations, but the pain remained unchanged. On February 2, 1986, she experienced oscillopsia and vomiting. Examination 2 days later disclosed a rotatory nystagmus on left lateral gaze. Right brachial arteriography on February 7, 1986, showed an aneurysmal dilatation of the first segment (Figure 3) and an irregular stenosis of the third and fourth segments of the right vertebral artery, which did not fill the basilar artery. A digitized intra-arterial arteriography on February 13, 1986, revealed normal carotid arteries, occlusion of the fourth segment of the left vertebral artery, and retrograde filling of the basilar artery and both posterior inferior cerebellar arteries. She was treated with anticoagulants, recovered completely in a few days, and has been symptomless since then. At the control digitized intra-arterial arteriography in September of 1986, the right vertebral artery was normal except for a mild stenosis of its fourth segment, whereas the left vertebral artery remained occluded.

**Patient 12**
A 38-year-old man had pain and stiffness in the left part of his neck in early March of 1986. The pain lasted for 5 days and reappeared 20 days later. On March 27, while turning his head to look over his shoulder, he felt a "cracking" in his neck, after which the pain disappeared. The following morning, he experienced dizziness, vomiting, and loss of balance when walking. In the evening, he had difficulty in swallowing and a severe inspiratory dyspnea. Examination showed decreased sensation of his right limbs, left Horner’s syndrome, and left pharyngeal paralysis. Bilateral brachial arteriography on April 4, 1986, showed moderate and irregular stenosis of the third segment of the left vertebral artery. The right vertebral artery was very thin and did not fill the basilar artery. The right internal carotid artery was normal. He was given anticoagulants for 2 months, then 1 g aspirin/day. Examination 3 months after the onset disclosed only a left Horner’s syndrome. Transfemoral arteriography on June 11, 1986, revealed a normal left vertebral artery and a beaded appearance of the right renal artery suggestive of fibromuscular dysplasia. The right vertebral artery could not be opacified.

**Patient 13**
A 40-year-old woman noted neck and back pain after washing the walls and ceiling of her kitchen on May 11, 1986. Ten days later, she had a 10-minute episode of vertigo and vomiting. On the following day, she was admitted to our hospital because of clouding of consciousness. Examination also found slurred speech, bilateral extensor plantar responses, and paralysis of vertical gaze. Bilateral brachial arteriography on May 28, 1986, revealed stenosis of the third segment of the left vertebral artery at the C1 level. She was given anticoagulants for 3 months, then 1 g aspirin/day and improved rapidly. On July 23, 1986, examination was normal, and digitized intra-arterial angiography showed only slight irregularities of the left vertebral artery.

**Discussion**
The incidence of extracranial vertebral artery dissection is not precisely known; for both carotid and vertebral dissections, it is estimated to be from "1 to 3 cases per year in large academic-affiliated hospitals." The fact that our 13 patients were seen in 6 years in a large neurology department is in accordance with this figure and suggests that this disease is not uncommon.

To compare our patient characteristics with those of the previously published cases, we reviewed published reports of pathologically confirmed and angiographically diagnosed extracranial vertebral dissections, again excluding patients whose angiogram showed only vertebral artery occlusion. In some reports, authors were uncertain of the nature of the arterial lesion and did not call the process dissection. These cases, however, were clinically and angiographically similar to those reported as dissection and will therefore be taken into account. Thirty-eight cases with 49 nonocclusive forms of extracranial vertebral dissection have thus been selected (Table 3).

The present study confirms that vertebral dissection occurs predominantly in middle-aged adults, but the female preponderance was more marked in our study (85%) than in previous reports (53%).

Particular attention has been paid in our study to some factors that might predispose to arterial dissection: chronic hypertension and atherosclerosis, fibromuscular dysplasia, migraine, and oral contraceptive use. Chronic hypertension was present in 3 of our patients; none had arteriographic evidence of atherosclerosis. Angiographic evidence of fibromuscular dysplasia is reported in about 15% of extracranial carotid dissection and in 0.5–1% of all arteriograms; it was found in 23% of the patients in our series and in 5% in previous reports. The higher prevalence of fibro-
muscular dysplasia in our series could be due to the fact that carotid angiograms were available in all cases and renal angiograms in 6 of 13 cases. A history of migraine was more frequent in our series (46%) than in previous reports of vertebral (8%) or carotid (11%) dissection. There are at least two reasons for the discrepancy between our data and the literature: first, the female preponderance in our population and, second, the fact that all our patients were specifically asked about migraine. The association of migraine with dissection raises the possibility that the migraine-associated dissections were not dissections but rather complicated migraine with vasospastic changes on arteriography. However, this hypothesis is very unlikely. Among 6 such patients in the present series, 3 had an aneurysmal formation (Figure 3) in addition to stenosis, which could not be due to vasospasm. Another who had stenosis without aneurysm had increased diameter of both vertebral arteries on duplex scanning, which later returned to normal. Four women (36%) in our series were taking oral contraceptives at the time of the dissection; 4 additional women were past users of oral contraceptives, and 2 of these had their dissection during pregnancy or post partum. In prior reports, only 10% of women were taking oral contraceptives at the time of dissection. Internal carotid artery dissection after childbirth has been described, but the occurrence of an extracranial vertebral artery dissection during pregnancy or post partum has, to our knowledge, never been reported. The high prevalence of migraine and hormonal factors in our series suggests that they could be implicated in the pathogenesis of dissection. A case–control study is needed to test the association between these two factors and dissection of cervical arteries.

Clinical symptoms and signs were similar to those previously described. Pain, usually located in the occiput and/or posterior neck, was the presenting symptom in about 80% of the cases in our series as well as in the literature, and preceded the first ischemic symptom from a few minutes to 1 month. Basilar ischemic symptoms were present in 33 of 38 published cases (87%) and in 12 of our 13 cases. In 1 of our cases, as well as in 5 reported cases, there were no basilar ischemic symptoms; in 4 cases (References 15, 22, 29, our Case 9), vertebral dissection was discovered when exploring a concomitant carotid dissection. One patient experienced an isolated headache while another patient, dead of peritonitis, had bilateral vertebral dissection discovered at autopsy. In all published cases with basilar ischemia and in 9 of our 12 cases, the clinical presentation was a completed stroke, only rarely preceded by transient ischemic attacks (TIAs) (2 published reports and 2 in our series). Among completed strokes, the roughly 30% prevalence of lateral medullary syndrome found in our series is in accordance with the literature but in disagreement with the statement by Hart and Easton that extracranial vertebral artery dissection "usually presents with features of the lateral medullary syndrome." TIAs were the only manifestation of basilar ischemia in 3 of our cases and in none in the literature, which suggests that some cases may be undiagnosed because of the still-widespread reluctance to perform vertebral angiography. The development of good quality digitized angiography should overcome this reluctance, and four-vessel angiograms should be performed in young or middle-aged adults presenting with "true" basilar TIAs, particularly if preceded by neck or head pain.

Extracranial vertebral artery dissection was more frequently bilateral (61%) and associated with carotid dissection (23%) in the present series than in previous cases (29% and 8%, respectively), but a number of published cases did not have four-vessel angiograms. Since it was decided to exclude vertebral artery occlusion as the only angiographic sign of dissection from both the present study and the review of the literature, it is not astonishing that the major finding was irregular stenosis of the extracranial vertebral artery; it was present in all our cases in at least 1 vertebral artery and in all but 2 published cases. In these 2 cases, 1 had a

![Figure 3. Patient 11. Right brachial arteriography. February 7, 1986. Aneurysmal dilatation of right vertebral artery at C7 level (arrow).](http://stroke.ahajournals.org/attachment/)

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double lumen aspect and 1 an isolated pseudoaneurysm.5 Stenosis with pseudoaneurysm was found in 29% of the cases (6 of 21 dissected arteries) in our series and in 20% of the cases in published reports. The present study confirms the rarity of dissection limited to the first segment (VI) of the vertebral artery. The main discrepancy between our study and previous reports lies in the frequency of dissections restricted to the V3 segment and of extensive dissection involving 2 or more segments. In our series, these findings occurred in 33 and 38%, respectively, compared with 57 and 16% in previous reports. No evidence of distal embolization, particularly to the posterior cerebral artery, was observed in the present series, even in cases with early angiograms. Distal embolization has previously been reported in 2 patients with extracranial vertebral dissection6,25 and in 15% of cases with carotid dissection.4 It therefore seems that in the present series, the dissecting process was more severe — at least angiographically — than in previous ones, with a greater frequency of bilateral and extensive dissection and of carotid involvement.

Follow-up angiography was performed from 7 days to several years after the first angiography in 12 patients with 19 vertebral dissections in the present series and in 16 published cases with 18 vertebral dissections. The results were similar: normalization in 63% (12 of 19) initially dissected arteries compared with 61% of cases, marked improvement in 26% of arteries compared with 33% of cases, and occlusion in very rare instances (11% in our series compared with 6% in published cases). Normalization or improvement is thus extremely frequent in this condition and an excellent argument in favor of the diagnosis. Combining data from our series and the literature, the return to normal was observed in 3 weeks—56 months (mean 8.6 months) and improvement with residual stenosis in 7 days—7 months (mean 2.7 months). This suggests that residual stenosis is just a step toward normalization rather than a persisting sequela or evidence of preexisting arterial disease. However, the above figures merely reflect the dates of angiographic controls and not the true timing of arterial improvement. Marked improvement was observed as early as 7 days after the first angiogram in 1 of our cases, which stresses the point that the first angiography should be performed as early as possible so as not to overlook the diagnosis. The timing of control angiography is open to debate since the natural history remains poorly documented. If only those cases with control angiography performed within 3 months after the first are taken into account (n = 20 vertebral arteries), 60% were already normal and 40% improved; these proportions are similar to those found when all cases (regardless of interval between angiographies) are taken into account. This suggests that control angiography could be performed around the third month.

Secondary thrombotic and embolic complications are the rationale for use of anticoagulants and antiplatelets in dissection.4 Eleven of our patients were given anticoagulants; 10 recovered without significant sequelae and 1 had significant residual deficit. Aspirin was given as the first treatment in the 2 remaining patients; 1 recovered without sequelae and 1 had significant residual deficit. Prior reports with outcome data include 27 patients. Ten patients2,15,18,19,28,30,32,33 were given anticoagulants; in 8, signs resolved satisfactorily, and 2 were left with significant residual deficit. Twelve patients2,6,15,20,22,25,29,31,34 were not given anticoagulants; 6 recovered without significant sequelae, 5 had residual deficit, and 1 died. The remaining 5 patients5,7,16,17,21 were treated surgically; 3 recovered

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**Table 3. 51 Cases of Extracranial Vertebral Artery Dissection**

<table>
<thead>
<tr>
<th>No.</th>
<th>Prior reports</th>
<th>Present series</th>
<th>Spontaneous*</th>
<th>Minor trauma†</th>
<th>Dubious‡</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (range)</td>
<td>38.4 (16–56)</td>
<td>38.2 (26–48)</td>
<td>36.4 (25–48)</td>
<td>33.9 (15–49)</td>
<td>38.1 (29–56)</td>
</tr>
<tr>
<td>Sex ratio F/M</td>
<td>1.1/1</td>
<td>5.5/1</td>
<td>5.5/1</td>
<td>1.6/1</td>
<td>0.5/1</td>
</tr>
<tr>
<td>Migraine</td>
<td>3 (8%)</td>
<td>6 (46%)</td>
<td>4 (21%)</td>
<td>3 (13%)</td>
<td>2 (22%)</td>
</tr>
<tr>
<td>Oral contraceptives</td>
<td>2 (10%)§</td>
<td>4 (36%)</td>
<td>6 (32%)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Fibromuscular dysplasia</td>
<td>2 (5%)</td>
<td>3 (23%)</td>
<td>3 (16%)</td>
<td>1 (4%)</td>
<td>1 (11%)</td>
</tr>
<tr>
<td>Bilateral vertebral artery dissection</td>
<td>11 (29%)</td>
<td>8 (62%)</td>
<td>9 (47%)</td>
<td>8 (35%)</td>
<td>2 (22%)</td>
</tr>
<tr>
<td>Carotid dissection</td>
<td>3 (8%)</td>
<td>3 (23%)</td>
<td>5 (26%)</td>
<td>1 (4%)</td>
<td>0</td>
</tr>
<tr>
<td>Localization¶</td>
<td>(n = 49)</td>
<td>(n = 21)§</td>
<td>(n = 28)</td>
<td>(n = 31)§</td>
<td>(n = 11)</td>
</tr>
<tr>
<td>V1 only</td>
<td>3 (6%)</td>
<td>0</td>
<td>1 (4%)</td>
<td>0</td>
<td>2 (18%)</td>
</tr>
<tr>
<td>V2 only</td>
<td>10 (20%)</td>
<td>5 (24%)</td>
<td>4 (14%)</td>
<td>9 (29%)</td>
<td>2 (18%)</td>
</tr>
<tr>
<td>V3 only</td>
<td>28 (57%)</td>
<td>7 (33%)</td>
<td>11 (39%)</td>
<td>17 (55%)</td>
<td>7 (64%)</td>
</tr>
<tr>
<td>≥ 2 segments</td>
<td>8 (16%)</td>
<td>8 (38%)</td>
<td>12 (43%)</td>
<td>4 (13%)</td>
<td>0</td>
</tr>
</tbody>
</table>

*Prior reports1,2,6,7,22,23,26,29 and present series (Cases 1–4, 6, 7, 9, 10).
†Prior reports1,2,6,7,22,23,26,29 and present series (Cases 8, 11).
‡Percent of women.
¶n, number of dissected vertebral arteries.
§One patient had intracranial dissection on one side and extracranial dissection on the other side.
without sequelae and 2 had residual deficit. The present study and previous ones indicate that anticoagulants are not harmful in this condition and could even be of benefit although no conclusion can be drawn from the comparison of nonrandomized treatment groups. Long-term outcome of patients with extracranial vertebral dissection appears to be favorable. In our series, long-term follow-up lasted 6–69 (average 34) months; no recurrence was observed. Prior reports include 12 patients with follow-up of >1 year (average 21 months); only 1 patient had a recurrent stroke, 1 year after the first stroke, the etiology of which was uncertain.

The relation of trauma to dissection is a complex issue. In the present series, dissection occurred after neck manipulation in 2 patients and after a potential minor injury to the neck in 3. The remaining 8 patients absolutely denied any recent trauma or abnormal movement of the neck. Evidence from different sources demonstrates that vertebral arteries may be compromised by head motion and, because of an impressive temporal relation, vertebral dissection has been linked to chiropractic manipulation and to a variety of circumstances associated with abrupt change in head position. However, the degree of injury to the neck that could induce dissection is difficult to define quantitatively, and the potential role of minor neck traumas such as head turning while driving a car does not explain why such trivial traumas are inconsequential in most people.

We defined 3 categories according to the presence of a potential traumatic event preceding the dissection and its relation with it: 1) spontaneous, when there was no recording of any form of injury to the neck or head; 2) minor trauma, when there was a close temporal relation of dissection with minor trauma (neck manipulation, sport activities, etc.); and 3) dubious, when the temporal relation of dissection with potential trauma was questionable. In Table 3 are presented some characteristics of patients with extracranial dissections according to this classification. Compared with dissection related to minor trauma, spontaneous dissection is more frequent in women (F/M sex ratio 3.5/1 vs. 1.6/1), more frequently involves a long portion of the vessel, and is more frequently associated with carotid dissection. Oral contraceptive use and fibromuscular dysplasia are also more frequent in patients with spontaneous dissection. However, these differences are minimal, and no specific particularity emerges that could discriminate spontaneous dissections from those related to minor trauma. These two variables can therefore probably be considered two portions of the same spectrum and not two different entities.

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