Extracranial Vertebral Artery Disease

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As early as 1844, Quain described the anatomy and operative surgery of the extracranial vertebral artery in lithographic drawings. In 1893, Matas described the contributions made in the early 1800s by other surgeons such as Dietrich, Velpeau, and Maisonneuve for the treatment of penetrating trauma to the vertebral artery. In 1831, Dietrich first proposed ligating the distal vertebral artery in the occipital-atloid region. In 1833, Velpeau ligated the vertebral artery at its proximal portion. Twenty years later, Maisonneuve successfully ligated the vertebral artery at the transverse foramen of the sixth cervical vertebra for a stab wound to the neck. The patient later died from a cerebral septic embolism.

As the discovery of extracranial vertebral artery disease became more extensive, new methods of treatment evolved. Pathologic injury to the vertebral artery, caused by erosion of its wall by a tuberculous abscess, was repaired by ligation by Smythe in New Orleans in 1864. Alexander also used ligature of the vertebral arteries to treat epilepsy, sometimes ligating both arteries at the same time. Elective ligation of the vertebral artery was also used to treat aneurysms. In 1888, Matas was the first surgeon who did not rely on ligation of the vertebral artery as treatment but fully excised an aneurysm between the occiput and the atlas through a posterior approach.

For the next 50 years, few advances were made in the medical treatment of extracranial vertebral artery disease until Moniz performed the first vertebral angiogram in 1927 (Moniz won the Nobel Prize not for this discovery but for the prefrontal lobotomy). Radner first reported selective angiography of the vertebral artery. This technique allowed researchers to correlate occlusive disease with symptoms. In 1946, Kubik and Adams first described basilar artery insufficiency caused by thrombosis of the basilar artery. Ten years later at the Mayo Clinic, Millikan and Siekert reported studies of cerebrovascular disease and the syndrome of intermittent insufficiency of the basilar arterial system. They introduced the use of anticoagulation drugs in the treatment of thrombosis of the basilar artery and noted a substantial reduction in the incidence of brainstem infarctions.

With this revolution in the diagnosis of diseased arteries, more aggressive surgical techniques were developed. The cause of brain ischemia was assumed to be hypoperfusion, with the solution being revascularization. In 1958, Crawford and coworkers presented their results of surgical treatment of brainstem ischemia by reconstructing the vertebral artery after removing atherosclerotic plaque. The next year, Cate and Scott first described the technique of trans-subclavian endarterectomy of the subclavian-vertebral artery.

In 1961, angiography allowed Reivich and colleagues to describe the process of reversed flow in the vertebral artery with proximal left subclavian stenosis in two patients with associated neurological dysfunction. This phenomenon was called subclavian steal syndrome. Angiography also allowed visualization of other causes of extracranial vertebral artery disease, including extrinsic compression of the vertebral artery by osteophytes, constricting bands, and rotational obstruction, all of which were diagnosed and treated by surgical decompression.

Angiography also provided the first extensive cooperative study of the incidence of extracranial arterial stenosis caused by atherosclerotic lesions in patients with cerebrovascular insufficiency. In 1968, stenosis was defined as a compromised lumen of more than 50% by the Joint Study of Extracranial Arterial Occlusion. Of 4748 patients, 80% had four-vessel angiograms that were categorized by location of the arterial stenosis. For the first time, this study provided a frequency distribution of surgically accessible sites with stenosis caused by atherosclerosis of the extracranial vertebral artery.

As microsurgery evolved in the 1970s, various reconstructive techniques also developed. Wylie and Ehrenfeld first treated pathology of the proximal vertebral artery by the transposition technique, with anastomosis between the vertebral artery and the common carotid artery (CCA). Berguer and associates used vein grafts in this region of the vertebral artery to connect the subclavian artery to the proximal vertebral artery. In January 1977, Carney and Anderson performed the first vein bypass from the CCA to the distal vertebral artery at the level of C1 and C2. Subsequently,
Carney used the supply from the subclavian artery, external carotid artery (ECA), internal carotid artery (ICA), and occipital artery to supply the distal vertebral artery.19

The treatment of penetrating vertebral artery injuries also changed significantly with the advent of the Vietnam War. Surgeons no longer relied solely on ligation of the artery as treatment; in many cases, they actually reconstructed the vertebral artery.19 In the civilian population, vertebral artery injuries were less common; Perry and coworkers20 reported no such injuries among 508 penetrating arterial injuries.

Angioplasty was first introduced by Dotter and Judkins21 in 1964 using flexible dilators, but it was not until the 1980s that angioplasty was successfully performed in the subclavian and vertebral arteries. In 1980, Bachmann and Kim22 first reported dilatation of the subclavian artery for the treatment of subclavian steal syndrome. In 1986, Higashida and colleagues23 reported successful percutaneous transluminal angioplasty of the vertebral arteries.

Today, many of the medical, surgical, and endovascular techniques described so far are still in use. The most rapid developments have been made in endovascular techniques. As the evolution of neurovascular surgery in the 1960s was used to remedy occlusion of the carotid and vertebral arteries, it is not difficult to imagine that we may be witnessing a new evolution in the management and diagnosis of extracranial vertebral artery disease.

**CLINICAL SIGNIFICANCE**

The external vertebral arteries provide blood flow to a large distribution via the basilar artery and posterior cerebral arteries. Therefore, symptoms can arise from the occipital or temporal lobes, cerebellum, pons, and brainstem with its cranial nerves. The term intermittent basilar artery disease.

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The presence of at least two symptoms is required to diagnose vertebrobasilar insufficiency.

Table 102-1 ■ Symptoms of Vertebrobasilar Insufficiency

<table>
<thead>
<tr>
<th>Motor or sensory symptoms, or both</th>
<th>Alternating paresthesias</th>
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<tr>
<td>Dysarthria</td>
<td>Homonymous hemianopsia</td>
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<tr>
<td>Imbalance</td>
<td>Diplopia</td>
</tr>
<tr>
<td>Dizziness or vertigo</td>
<td>Other cranial nerve palsies</td>
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<tr>
<td>Tinnitus</td>
<td>Dysphagia</td>
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The most common symptom that physicians (including neurologists and neurosurgeons) have difficulty relating to VBI is vertigo or dizziness. First, this symptom is often associated with other diseases. In addition, many articles have ruled out the occurrence of vertigo or dizziness as evidence of transient ischemic attacks. The dogma that vertigo or dizziness alone is not a presenting symptom of VBI was thereby established.27 These patients are often treated empirically or referred to an otolaryngologist for further evaluation.

Studies by Grad and Baloh27 and Kumar and associates28 raised significant questions about how extensive an evaluation is needed in patients with isolated symptoms of vertigo. Kumar's group found that vertigo, dizziness, and imbalance—individually or collectively—can be exclusive symptoms of VBI and that vestibular test results (recruitment and hyperactivity) show a clinically significant sensitivity for distinguishing labyrinthine or brainstem-cerebellum (posterior neuraxis) lesions. In general, the vagueness of the presenting symptoms of VBI requires a team approach to analyze these patients. Because the final determination of the cause of VBI requires invasive and expensive tests, a clear-cut approach to making the diagnosis is essential.

**ANATOMY OF THE EXTRACRANIAL VERTEBRAL ARTERY**

The vertebral artery varies in diameter from 0.5 to 5.5 mm and in length from 5 to 35 cm. It is a high-resistance artery, and both sides accommodate blood flow rates of about 120 mL/min. The left vertebral artery is larger than the right in about 75% of cases.

The extracranial vertebral arteries can be divided into three regions: proximal, middle, and distal (Fig. 102-1). This division is helpful because the associated pathology appears to differ among the segments. In many cases, the surgical approaches are similarly divided by these anatomic regions.

**Proximal Vertebral Artery.** The proximal vertebral artery extends from the superior portion of the subclavian artery to enter the transverse foramen of C6. It
PATHOPHYSIOLOGY OF EXTRACRANIAL VERTEBRAL ARTERY DISEASE

The pathophysiology of extracranial vertebral artery disease is not as well understood as that of the carotid system. Vertebrobasilar symptoms arise from interruption of the blood supply to the brain and brainstem. The interruption can be the result of hypoperfusion caused by hemodynamic changes or by thromboembolic sources. Ischemia from hemodynamic mechanisms rarely causes infarction initially; rather, the symptoms are short-lived and repetitive. They are, however, still dangerous if the hypoperfusion persists. In contrast, emboli are more likely to cause dangerous infarctions and leave patients with permanent deficits. Most vertebrobasilar symptoms are likely caused by emboli, although exact percentages are lacking in the literature.

**Hemodynamic Causes**

Hemodynamic changes can result from either an interruption of the source of blood supply or blockage of the conduit that provides the blood flow. The former occurs in cardiac insufficiency or postural hypotension from systemic disease. The latter is related to obstruction of blood flow in the arterial system, but obstruction of blood flow in one vertebral artery may be insufficient to cause hemodynamic changes. Anatomic variations in the other artery—such as a hypoplastic vertebral artery, termination of one artery into the posterior inferior cerebellar artery, or complete occlusion of the contralateral vertebral artery—can also occur. There also may be associated pathology in the carotid system or an incomplete circle of Willis. The mere presence of an obstruction or stenosis does not necessarily mean that blood flow in that particular artery will be significantly reduced. In 1938, Mann and colleagues first demonstrated that a decrease in the flow rate of the carotid artery does not become significant until a critical narrowing occurs (called critical stenosis). May and coworkers confirmed this finding in later studies and showed that critical stenosis depends on the baseline rate of blood flow. For example, if symptoms appear in a low-flow system when the rate of blood flow is reduced 25%, the critical stenosis is about 85%. For a high-flow system, however, the critical stenosis for the same percentage of change in the rate of blood flow may be as low as 35%. This fact is often not fully appreciated, especially when a similar criterion for critical stenosis is used for both the carotid and vertebral arteries, even though the flow rate in the CCA approaches 300 mL/min, compared with about 120 mL/min in the vertebral artery. Hence, the criterion for critical stenosis of the carotid artery cannot be applied to the vertebral artery.

Examples of diseases in the extracranial vertebral arteries that cause hemodynamic changes include atherosclerosis, compressive syndromes, traumatic or spontaneous dissections of these vessels, and subcla-
vian steal syndrome. Atherosclerosis is the most common form of vertebral artery disease. Although it is a source of thromboembolic plaque, it can cause significant hypoperfusion by obstructing blood flow. Details of the incidence, clinical manifestations, and prognosis of hypoperfusion caused by this disease process are still lacking. One of the most extensive studies on the incidence of extracranial disease in symptomatic patients was presented by Hass and associates from the Joint Study of Extracranial Arterial Occlusion. They found that the most common site of plaque formation in the vertebrobasilar arterial system was the origin of the proximal vertebral artery (right vertebral artery, 18.4%; left vertebral artery, 22.3%). The second most common site was the middle vertebral artery. In this region, it is believed that the blood flow rate is damped as it passes through the foramen. Atherosclerosis occurs less frequently intracranially in the midbasilar artery and at the entry of the vertebral artery through the dura.

Spontaneous dissections are associated with systemic diseases affecting the arterial walls. In both the carotid and vertebral arteries, fibromuscular dysplasia is the most common cause of spontaneous dissection. It tends to affect areas where there is significant movement of the cervical spine and therefore occurs in the middle and distal segments of the vertebral artery. The formation of pseudoaneurysms is also quite common, although these lesions are often asymptomatic.

Trauma is the third most common cause of vertebral artery disease. Both blunt and penetrating trauma can dissect the vertebral artery. Blunt injury occurs from cervical spine fractures and dislocations that may result in occlusion, pseudoaneurysm, or arteriovenous fistula (AVF) of the vertebral artery, especially in the middle portion. This type of injury can also be created iatrogenically from chiropractic manipulation. The most frequent site of thrombosis is at the level of C2 in the distal vertebral artery. This tendency may reflect the posterior placement of the vertebral foramina with respect to the vertebral body. The vertebral artery has an increased vulnerability for compression by subluxation of the cervical apophyseal joints. Blunt injury to the vertebral artery may be more common than has been quoted in the literature because these patients seldom undergo angiography unless they show symptoms of vertebral insufficiency. Penetrating trauma to the vertebral artery is less common than blunt trauma. In 1971, Perry and coworkers examined 508 penetrating arterial injuries in the civilian population and found no involvement of the vertebral artery in any of the cases. Only during periods of war and with the advent of shrapnel on the battlefield did the incidence of these injuries increase.

Compression of the vertebral artery can cause VBI. The anterior scalene muscle has been found to compress the vertebral artery at the level of C6. Osteophytes and disk spurs, found between levels C6 and C2, can encroach on and compress the middle vertebral artery, causing vascular symptoms. Usually, rotation or extension of the neck triggers symptoms. Dynamic angiography has been recommended for patients who show vertebral artery symptoms on flexion, extension, or rotation of the neck.

Subclavian steal syndrome was first described by Reivich and colleagues in 1961 when they discovered reverse flow in the vertebral artery. It is caused by stenosis or occlusion in the subclavian or innominate artery proximal to the vertebral artery. If the pressure in the subclavian artery distal to the obstruction is low enough, it acts as a "sink" for the flow of blood from the vertebral artery and drains blood from the contralateral vertebral artery and even as far as the circle of Willis (Fig. 102-2). Hence, patients can experience vertebrobasilar symptoms and may also have cerebrum, cerebellum, and brainstem symptoms. Most of these symptoms are caused by use of the extremities when the demand for blood flow is increased and the pressure sink becomes more pronounced. In many cases, patients rarely experience symptoms at rest.

**Embolic Causes**

The pathology that causes hemodynamic changes and the sources of emboli often overlap. Embolism arising from within or outside the vertebral system seeks the terminal branches of the basilar artery or the posterior cerebral arteries. Consequently, symptoms can manifest as simple cranial nerve palsies or brainstem vascular syndromes (e.g., Wallenberg's syndrome, Weber's syndrome). Acute visual field defects or symptoms of occipital lobe infarction can also be presenting symptoms.

Emboli or thrombi may originate from the vertebral arteries themselves or from the subclavian or aortic arches. They also can come from pathologic heart valves, abnormal cardiac wall behavior, or arrhythmias. Atherosclerotic plaque is usually the source of emboli or thrombi from the aorta or subclavian or vertebral arteries. If not treated with anticoagulation, thrombi
from the spontaneous rupture of vessels or after trauma to the extracranial vertebral arteries will obstruct the smaller branches of the vertebral arteries.

**DIAGNOSTIC EVALUATION**

The symptoms associated with disease of the extracranial vertebral artery are multiple and often vague. Distinguishing patients with true VBI is therefore a significant challenge. Once such patients have been diagnosed, the treatment must be individualized to best suit the specific patient. A statement by one of the senior authors of this chapter 20 years ago still holds true today: "The surgeon operating on the vertebral artery must address not only the surgical technique, but also the diagnostic approach, the hemodynamic documentation." [19]

The diagnostic approach to extracranial vertebral artery disease consists of ruling out patients who present with VBI-type symptoms caused by disorders other than vertebrobasilar artery insufficiency, identifying the cause and thus identifying patients with extracranial vertebral artery disease, and determining whether the cause is embolic or hemodynamic. Many systemic and neurological diseases can cause VBI-type symptoms. Meninge's disease, infection or dysfunction of the vestibular and labyrinthine structures, demyelinating diseases, seizures, tumors of the cerebellopontine angle, spinal column dysfunction, and compression of structures in the posterior fossa from either masses (intra-axial cerebellum tumors) or bony encroachment (Chiari's malformation) can all manifest with VBI-type symptoms. Reduced cardiac output can also cause symptoms of VBI. Cardiac disease such as dysrhythmias, cardiac insufficiency, and infarction can result in poor cardiac output. Thromboembolic causes from cardiac valvular disease, bacterial endocarditis, dysrhythmias, and hematologic diseases (thrombocytosis, bleeding disorders, sickle cell) can cause symptoms of VBI. Other systemic diseases such as diabetes can cause autonomic dysfunction that causes orthostatic hypotension. Severe cases of hypovolemia associated with poor autonomic function can manifest with symptoms of VBI. Therefore, a careful medical and diagnostic workup is necessary when evaluating these patients.

**History and Physical Examination**

The first process in any evaluation is to obtain a good history of the patient's presenting symptoms. The history must identify the onset of symptoms, their duration, and the predisposing conditions that elicit or relieve symptoms. VBI is a vascular phenomenon, and the onset of symptoms is sudden. Hypertension, smoking, and, in women, contraceptive medications can be contributing factors. The patient's work history and any family history of migraine headaches or cardiac or neurological diseases need to be known. Patients on medications, particularly antihypertensive medica-

**Routine Laboratory Evaluation**

A routine metabolic and blood workup should be obtained. Patients on medications that require therapeutic levels should be monitored, because many VBI symptoms can be related to overmedication (e.g., antihypertensives). If the patient's work history indicates exposure to unusual chemicals known to be toxic, the appropriate level should be determined. A 12-lead electrocardiogram, a 24-hour Holter monitor and, if possible, an echocardiogram (if indicated) can be the first steps in evaluating the heart.

**Audiometric and Vestibular Tests**

In some cases, the presentation of vertigo or dizziness with no other findings requires consultation with an otolaryngologist to rule out labyrinthitis or vestibular causes. By the time patients are seen by a neurologist or neurosurgeon, most of them have already been treated with medications for labyrinthitis or Meninge's disease. Before more invasive procedures such as cerebral angiography are recommended, these patients should undergo audiometric and vestibular tests. Audiometric tests include a pure-tone audiogram and a speech discrimination test to indicate hearing loss. A vestibular test can indicate recruitment and hyperactivity, which can be strong indicators of a centrally located lesion (sensitivity of 92%). [28]

**Brain Imaging Techniques**

The brain and the posterior fossa must be imaged as part of the evaluation. Computed tomography is an excellent imaging technique for ruling out mass lesions or hemorrhages. Magnetic resonance imaging (MRI) is highly sensitive and can detect demyelinating disease, stroke, and mass lesions. MRI of the arterial system, or magnetic resonance angiography (MRA), is a good noninvasive screening technique for evaluating the intracranial and extracranial arteries. Its ability to accurately identify stenosis is limited, however. The use of contrast enhancement can increase the utility of magnetic resonance angiography. [32]

For patients with VBI, metabolic changes occur immediately. MRI and computed tomography cannot detect such changes acutely (<24 hours). Single photon emission computed tomography evaluates the metabolic function of the brainstem and cerebellum, as does xenon computed tomography. Both modalities, however, are of limited use in the posterior fossa because of imaging difficulties. Diffusion-weighted imaging and perfusion imaging, two new magnetic resonance tech-
nologies, are becoming increasingly available for the evaluation of acute ischemic stroke patients. Diffusion-weighted imaging provides early information about the location of acute focal ischemic brain injury, and perfusion imaging can document the presence of disturbances in microcirculation perfusion.

Cerebral Angiography

Cerebral angiography is considered the gold standard for evaluating the intracranial and extracranial vessels of the brain. Unlike MRA, it is an invasive procedure and carries a low risk of stroke (1% overall incidence of neurological deficit, and 0.5% incidence of persistent deficit).

In cases of extracranial vertebral artery disease, the aortic arch must be visualized, as well as the four major intracranial arteries. VBI symptoms are caused predominantly by intracranial disease, and good visualization of the intracranial vessels is essential. Similarly, subclavian steal syndrome manifests with VBI symptoms, although the pathology is located in the subclavian artery.

Compared with MRA, cerebral angiography is a dynamic study. As the contrast medium diffuses, a quantitative sense of the hemodynamics can be obtained. In the hands of an experienced neuroradiologist or neurosurgeon, the blood flow in the basilar artery can be determined as low or high. Similarly, retrograde flow, as occurs in subclavian steal syndrome, can be seen. Dynamic angiography can also be used to monitor vascular changes associated with head position. This feature is needed in patients with an occlusion or reduced blood flow in the vertebral artery from an obstruction caused by soft tissue (ligament or muscle), neuronal tissue, or bone.

Hemodynamic Evaluation

Once an obstructive lesion has been identified, it is important to determine whether the VBI symptoms are from poor perfusion caused by the obstruction or by emboli. Cerebral angiography can give some sense of the cause, but it is far from reliable. Several methods have been used to evaluate the hemodynamics. Ultrasonography of the vertebral arteries has been used, but insonation is difficult, and its sensitivity is questionable. Interequipment, interinstitution, and technician variability make this method unsatisfactory. Intracranial hemodynamic changes have also been monitored with transcranial Doppler ultrasonography, but this technique is also difficult to use in the posterior fossa.

Since the 1980s, flow quantification using phase-contrast MRI of the blood vessel has been studied. Although static MRI or conventional angiography is useful for determining the anatomy of the vessel, phase-contrast MRI provides actual flow rates of blood in the vessel (in milliliters per minute). Both in vitro and in vivo flow studies have shown that velocities and volumetric flow rates can be estimated accurately for the carotid, vertebral, and major cerebral arteries. Normal values for flow rates in these vessels have been estimated.

Therefore, phase-contrast MRI provides a noninvasive method for analyzing the cause of VBI symptoms. Rates of blood flow in both the vertebral and basilar arteries can be estimated using this technique. Based on knowledge of the normal range of flow rates in these vessels, it can be determined whether obstructive lesions are significant enough to cause hypoperfusion of the vertebral artery. This knowledge is essential in planning treatment.

Other Evaluation Techniques

Once the diagnostic data are analyzed, one should have a clear indication of whether the cause of VBI is hemodynamic changes or emboli. If the treatment plan involves surgery or endovascular management that will change hemodynamics, further investigation is required. Several alternatives are available, but the optimal choice must provide reperfusion with the smallest risk to the patient. In the past, this decision was based solely on the surgeon’s bias and training.

The use of mathematical models provides a unique method of testing alternative surgical strategies before they are implemented. Many models have been presented in the literature, but the most common difficulties are their lack of patient specificity and their inaccuracy. We routinely use such models for planning surgery by simulating alternative procedures and evaluating the flow rate distribution after each one. The extracranial vertebral arteries are not isolated hemodynamically. Reconstruction of the vessels affects blood flow in the entire extracranial and intracranial system for both carotid and vertebral arteries. Therefore, careful planning is necessary.

By the time a treatment plan is chosen, the evaluation should indicate the cause of the VBI symptoms. In cases of hemodynamic compromise, removing the obstruction by surgery or endovascular angioplasty or bypassing the lesion may be indicated. For emboli, medical management is recommended initially in most cases. If the symptoms persist, surgical options can be used. Removing or bypassing the lesion with ligation of the offending vessel is often used.

MEDICAL MANAGEMENT

The use of medical management for vertebrobasilar ischemia dates to the 1950s, with the use of heparin, and to the 1970s, with the use of oral anticoagulation. This type of medical treatment for vertebrobasilar ischemia was associated with good outcomes. These early studies were flawed, however, because no angiographic studies were performed to identify the cause and significance of the extracranial disease. Without these data, the conclusions are anecdotal because the causes of extracranial vertebral artery disease are so variable.

Medical therapy of the extracranial vertebral arteries is used to prevent thrombus formation anywhere in
the vertebral arteries or to prevent emboli from plaque. Vertebral artery dissections with the potential to form thrombi have been treated successfully with anticoagulation therapy. Once a thrombus has formed and caused hypoperfusion, however, it can be treated with angioplasty with anticoagulation or with local infusion of streptokinase or urokinase to dissolve the clot. The administration of tissue plasminogen activator has met with great success in dissolving clots.

Thrombi and emboli can also come from systemic sources. Typically, they migrate to the anterior circulation but can make their way to the posterior circulation. Medical therapy and, in particular, anticoagulants are used to prevent this thromboembolic formation from systemic sources. Medical therapy is also used to reduce the risk and complications of stroke, which include hypertension and high cholesterol levels.

Several antiplatelet trials have shown that aspirin reduces the relative risks of stroke, myocardial infarction, and vascular death by about 25%. Ticlopidine is more effective than aspirin but has important side effects. Clopidogrel is as effective as ticlopidine, with fewer side effects. In 1996, the European Stroke Prevention Study showed that dipyridamole effectively prevents stroke and, when combined with aspirin, is equivalent to ticlopidine or clopidogrel. These results can be applied to the medical treatment of extracranial vertebral artery disease.

Warfarin has also been used to prevent stroke and myocardial infarction. In 1995, the Warfarin-Aspirin Symptomatic Intracranial Disease Study showed a significant difference in stroke rates in patient with intracranial disease taking warfarin versus aspirin (stroke rate, 10.4/100 patient-years versus 3.6/100 patient-years). In many cases of extracranial vertebral artery disease, there is an associated intracranial component. The use of warfarin is encouraged in these cases.

ENDOVASCULAR MANAGEMENT

Endovascular management of the external vertebral artery is in its infancy, and only selected procedures are performed. Vertebral AVFs have been treated by embolization with latex balloons. Beaujeux and colleagues treated 46 AVFs that occurred between C1 and C2 in 21 patients, between C2 and C5 in 5 patients, and below C5 in 20 patients. More recently, electrical detachable coils were used to treat an AVF; the 5-month follow-up showed obliteration of the fistula.

Although percutaneous transluminal angioplasty has been widely used to treat the ICA with or without the placement of stents, vertebral artery angioplasty is becoming more common. Its main application to the external vertebral artery is for the treatment of atherosclerotic plaque, which most often occurs at the origin of the vertebral artery. The plaque is often fibrous with a smooth surface (ulcerated in <4% of cases), making it ideal for percutaneous transluminal angioplasty. Restenosis poses a major problem; in one series, it was reported in 3 of 34 arteries. The use of stents can alleviate this complication. Storey and associates reported three patients who failed medical therapy and conventional angioplasty of the proximal vertebral artery and developed restenosis within 3 months. Stents were placed, and a 9-month follow-up showed no restenosis and no symptoms. This method is becoming the standard technique for treating stenosis of the proximal vertebral artery.

CASE HISTORY 1

A 53-year-old right-handed man was seen in the emergency room after complaining of the sudden onset of horizontal diplopia, dizziness, and ataxia. His medical history was significant for heart disease and hypertension, for which he was treated medically. The neurological examination was significant for nystagmus. The cranial nerves were intact, as were his sensation to pinprick and motor function. The cerebellar examination was significant for right-sided dysmetria. T1-weighted MRI showed hypodense areas in the right lateral medulla. The patient was placed on warfarin, but the dizziness continued intermittently for 3 months. After an extensive workup for other causes of his dizziness, the patient underwent four-vessel cerebral angiography, which showed 90% stenosis at the proximal vertebral artery (Fig. 102-3). The patient underwent angioplasty and stent placement. He was asymptomatic during a 3-month follow-up.

SURGICAL MANAGEMENT

Unlike those for the carotid artery, no clinical trials have shown the beneficial effect of surgery for high-grade stenosis of the vertebral artery. If an extensive evaluation shows hypoperfusion or the patient is refractory to medical management, either endovascular or surgical management is indicated. The surgical approach to each anatomic segment of the extracranial vertebral artery is different. However, treatments intended for a given segment can sometimes be used for a preceding segment. The following discussion of the surgical management of extracranial vertebral artery disease is divided into the different surgical procedures that can be performed in each anatomic segment.

Surgery of the Proximal Vertebral Artery

Several operations have been devised to treat lesions of the proximal artery. Transposition of the proximal vertebral artery onto the CCA is the most common procedure performed in this section of the artery. Bypasses using vein grafts are also performed from the adjacent subclavian artery or CCA to the proximal vertebral artery. Endarterectomy of the subclavian artery or the proximal vertebral artery can also be performed. Vertebral artery angioplasty with stent placement, however, is becoming the first choice of nonmedical management.

Approach to the Proximal Vertebral Artery. The standard approach to the proximal vertebral artery is a supraclavicular approach (Fig. 102-4A). The patient's head is placed in a headrest. Downward traction of the arm provides better exposure. We prefer to keep the head midline for this approach. A supraclavicular inci-
sion is made about 2 cm above and parallel to the clavicle and extends from the suprasternal notch to 7 to 8 cm laterally. The skin is retracted superiorly and inferiorly, leaving the platysma intact. The platysma is divided horizontally. The superficial veins flank the edges of the sternocleidomastoid muscle as the external jugular vein comes from the lateral edge and crosses the muscle at the middle level (see Fig. 102-4B). The sternocleidomastoid muscle has two origins: the clavicular head from the superior surface of the medial third of the clavicle, and the sternal head from the anterior surface of the manubrium of the sternum.

The clavicular head is divided, leaving a cuff on the clavicle, and the muscle is retracted superiorly and laterally. The omohyoid muscle can also be divided (see Fig. 102-4C). The dissection is kept medial to expose the carotid sheath. The anterior scalene muscle lies laterally, with the phrenic nerve lying on top of it. This muscle is usually far lateral to the exposure and rarely requires division. The carotid sheath is separated from the overlying fascia and opened. Inside can be found the CCA, the internal jugular vein, and the vagus nerve. The jugular vein and vagus nerve are retracted laterally, and the CCA is retracted medially. From this point, dissection proceeds below the deep fascia layer caudally.

If the right side is exposed, several steps are needed. The lymphatic drainage on the right side of the neck is different from that on the left. Delicate lymphatic trunks empty into the right subclavian and jugular veins, which are usually smaller than the lymphatic ducts on the left. Because they do not coagulate completely, it is better to identify and ligate them. The right recurrent laryngeal nerve exits the vagus nerve and loops below the right subclavian artery as it approaches the trachea and larynx. Consequently, medial retraction of the trachea can cause ipsilateral paresis of the vocal cord.

If the left side is exposed, the thoracic duct is encountered as it arches from the side of the esophagus laterally to the angle between the internal jugular and subclavian veins. The proximal portion of this duct is ligated twice (see Fig. 102-4C), and smaller branches are also ligated. The left recurrent laryngeal nerve can be retracted with greater ease because it loops around the aortic arches and approaches the trachea much lower.

The vertebral artery can now be identified (see Fig. 102-4D). It is the first branch of the subclavian artery and exits from its posteroinferior surface. This feature distinguishes it from the thyrocervical trunk, which has multiple branches and exits from the anteroinferior surface. Alternatively, the vertebral artery can be located superiorly as it exits the transverse foramen of C6. The transverse process of C6 can be identified adjacent to its foramen. The artery arises from the apex of two muscles as they attach to the carotid tubercle: the anterior scalene muscle and the longus colli. The vertebral vein, which overlies the artery, can be divided or retracted. The vertebral vein is formed at the lower end of the canal of the transverse foramina from a venous plexus within the canal around the vertebral artery. The vein is anterior to the artery and often adheres to it.

It is important to identify and preserve the sympathetic chain. The vertebral artery is looped and dissected from C6 to the subclavian artery. Care is exerted to avoid destroying the sympathetic trunks and stellate or intermediate ganglia that lie on it. The anterior surface is freed.
FIGURE 102-4. Supraclavicular approach to the proximal vertebral artery. A, The incision is placed 2 cm above and parallel to the clavicle. B, Exposure of the sternocleidomastoid muscle and the external jugular vein. C, The clavicular head is divided, leaving a cuff on the clavicle, and the muscle is retracted superiorly and laterally. The omohyoid muscle can also be divided to expose the vascular contents and thoracic duct. D, Exposure of the vertebral artery.
Transposition of Proximal Vertebral Artery to Common Carotid Artery. This procedure, first described by Wylie and Ehrenfeld in 1970, is used because of the ease of exposure. Its limitation, however, is the requirement for simultaneous occlusion of both carotid and vertebral arteries (Fig. 102–5A). Using the standard approach described earlier for isolation of the proximal vertebral artery, the CCA is prepared for the vertebral artery. The CCA is already isolated during the dissection of the vertebral artery. Adventitia is cleared from the carotid artery. The patient is given a bolus of 3000 to 5000 U of heparin. Five minutes later, the vertebral artery is clamped at the level of C6 with a temporary clip. The proximal vertebral artery just above the stenosis is occluded with a hemoclip and cut above it. The artery is freed from the surrounding sympathetic trunk and moved medially toward the CCA. If the vertebral artery is not lax enough, it may be necessary to remove it from the C6 transverse process. A fish-mouth opening is made in the proximal end of the vertebral artery.

A partially occluding clip is placed on the carotid artery at the selected level and used to rotate the vessel medially. This maneuver allows the anastomosis to be performed on the posterolateral wall of the CCA in line with the trajectory of the vertebral artery. With 7–0 monofilament nylon suture, the superior and inferior ends of the fish-mouth opening are sutured to the corresponding ends of the hole in the carotid artery. One suture is used to form a running anastomosis on the back wall and is tied to the opposite end on completion. The front walls are then sutured. Before the last suture is tied, the lumina of both arteries are flushed with heparinized saline. First the vertebral artery and then the CCA are back-flushed. The final suture is tied, and all clamps are removed. If blood continues to ooze, gentle pressure is placed over the anastomosis with Gelfoam. After copious irrigation and when hemostasis is obtained, the neck opening is ready to be closed.

The sternocleidomastoid muscle is reapproximated. A suction drain is placed in the neck and should be removed in 24 hours.

Vein Graft Bypass from Subclavian Artery or Common Carotid Artery. When transposition is infeasible because of the length of the proximal vertebral artery or an endarterectomy cannot be done, a vein graft bypass is indicated. Berguer and Feldman anastomosed a saphenous vein graft to the subclavian artery distal to the site of origin of the vertebral artery and then attached it by an end-to-side anastomosis to the subclavian artery (see Fig. 102–5B). Although this procedure does not interrupt carotid blood flow, it requires two anastomoses and is time consuming. The proximal vertebral artery can be bypassed from the subclavian artery to the thyrocervical trunk (see Fig. 102–5C) or CCA.

For any bypass to be successful, one of the three brachiocephalic arteries must be free of significant stenosis. Usually, the left carotid and innominate arteries are less likely to be stenotic. If the carotid artery is stenotic or otherwise compromised, the subclavian artery can be used. The desired segment of the subclavian artery is in the area of the anterior scalene muscle or more distally. The vein is usually autogenous saphenous, although prosthetic materials have been used. As described previously, this approach uses an end-to-side anastomosis.

Subclavian-Vertebral Endarterectomy. In 1959, Cate and Scott described an endarterectomy of the origin of the vertebral artery through a subclavian approach (see Fig. 102–5D). They chose this approach because the vertebral artery is too fragile to accommodate vertical dissection. The dissection requires exposure of the proximal and distal subclavian artery. As described earlier, a more extensive approach is required distally. The anterior scalene muscle is divided, and care is exerted to preserve the phrenic nerve. The thyrocervical trunk and internal mammary artery need not be sacrificed but can be clamped with temporary aneurysm clips. Again, the thoracic duct must be ligated on the left, as described previously.

Heparin (5000 U) is given. Five minutes later, the proximal and distal portions of the subclavian artery are clamped. A horizontal incision is made in the subclavian artery below its junction with the vertebral artery. The plaque is removed from the subclavian artery and followed into the stoma of the vertebral artery. If intimal flaps remain at the margins, they are tacked up with 6–0 monofilament suture. In this region, the plaque is usually short and does not require this procedure. The incision in the subclavian wall is closed with 6–0 monofilament nylon after back-flushing from the vertebral artery and from the proximal and distal subclavian artery. Hemostasis is obtained, and the wound is closed.

An alternative approach is to apply a vein patch obtained from the saphenous or jugular vein to a vertical incision made in the vertebral artery that extends into the subclavian artery. After the plaque is removed, the vein patch is used to close the incision.

Although subclavian-vertebral endarterectomies have been used successfully since the 1960s, they are still associated with several technical problems. The endarterectomy is a difficult approach to use with a low-lying subclavian artery. Some have advocated the use of intrathoracic approaches. With other safer alternatives now available, the procedure is seldom used today.
compression, this technique can be used instead of removing the ganglia, which will worsen the Horner’s syndrome.

**Other Procedures.** If high-grade stenosis of the proximal vertebral artery is present, the inferior thyroid artery can become well developed. Carney proposed an end-to-end anastomosis of the inferior thyroid artery to the proximal vertebral artery.

**CASE HISTORY 2**

A 65-year-old right-handed man was referred to our institution for persistent vertigo and dizziness. The patient had undergone multiple neurovascular surgeries, including a left carotid endarterectomy 14 years earlier and a left carotid-to-subclavian artery bypass 12 years earlier. His medical history was significant for hypertension, severe hypercholesterolemia, benign prostate hypertrophy, gastroesophageal reflux, and a questionable myocardial infarction.

He was alert, oriented, pleasant, and in no acute distress. He had a carotid endarterectomy scar on the left. The rest of his physical examination was normal. Cranial nerves II through XII were intact. His pupils were equally round and reactive. His motor strength was 5/5 in all extremities, and his facial expressions were symmetrical. There was no evidence of decreased sensation. His reflexes were symmetrical, and no Babinski’s reflex was present. His gait was steady, but he was unable to tandem walk. There was no dysmetria, but the Romberg test was positive.

The patient underwent an extensive evaluation by the ear, nose, and throat service to rule out a vestibular cause for the symptoms. The evaluation was negative and led to MRI and MRA. Angiograms showed occlusion of the left subclavian-to-carotid artery bypass and occlusion of the left origin of the subclavian artery. The origin of the right vertebral artery was 70% stenotic (Fig. 102-6A).

Based on these data, the patient’s symptoms were attributed to stenosis of the right vertebral artery. The treatment of choice, angioplasty of the lesion and stent placement, was unsuccessful. The patient then underwent a right vertebral artery-to-carotid artery transposition in a similar manner as described previously. His hospital course was uneventful. His symptoms resolved, and 6-month follow-up studies showed the transposition to be patent (see Fig. 102-6B).

**Surgery of the Middle Vertebral Artery**

Surgical reconstruction of the middle vertebral artery is rarely undertaken, although it is possible to bypass diseased segments. Most surgeons use angioplasty...
as the first choice of treatment or may bypass at the level of the distal vertebral artery. Single or minor extrinsic lesions can be removed to relieve compression on or kinking of the vertebral artery. In some cases of extensive proximal artery disease, it may be necessary to revascularize the middle vertebral artery. The proximal artery or a portion of the middle vertebral artery is ligated. After the vertebral artery is dissected, any of the previously described techniques can be used to attach the middle vertebral artery.

**Approach to the Middle Vertebral Artery.** The middle vertebral artery can be accessed from an anterior or anterolateral approach. The incision can traverse a skin crease or be made longitudinally along the anterior border of the sternocleidomastoid muscle (Fig. 102–7A), depending on whether the pathology involves one or two levels. Initially, a cervical radiograph is used to define the level of interest and should be repeated intraoperatively before the transverse process is drilled. The skin is retracted, and the platysma is left intact until it is completely exposed. An incision is then made longitudinally along the anterior border of the sternocleidomastoid muscle (see Fig. 102–7B). By blunt dissection, a plane is developed between the strap muscles, trachea, and esophagus, which are retracted medially, and the sternocleidomastoid muscle and carotid sheath, which are retracted laterally. The longus colli muscle can be seen on the anterior vertebral body (see Fig. 102–7C). For a more lateral approach, the carotid muscle can be seen on the anterior vertebral body (see Fig. 102–7D). A limited endarterectomy is performed with a possible vein graft or transposition from the middle vertebral artery to the CCA.

The sympathetic ganglia seen on the lateral aspect of the longus colli need to be preserved (see Fig. 102–7C). As noted, the vertebral artery can be located at the transverse foramen of C6 between the longus colli muscle medially and the anterior scalene laterally. Using a periosteal elevator, the dissection proceeds subperiosteally to remove the muscles from their attachment to the anterior surface of the transverse process. The muscles are reflected laterally with sutures. The transverse process is removed using a high-speed drill or curet (see Fig. 102–7D). Immediately below the transverse process, anterior to the vertebral artery, is the venous plexus, which is coagulated meticulously. Care is taken to preserve radiculomedullary arteries that exit from the vertebral artery between C1 and C5 and supply the spinal cord.

**Decompression of the Middle Vertebral Artery.** Several surgeons have operated in this segment of the vertebral artery to treat external compressive lesions (Fig. 102–8A). The anterior approach is used. Once the transverse process is reached, the level of the compression can be identified and decompressed. Plain anteroposterior cervical radiographs identify the level. Osteophytes are drilled off or removed with curets. The periosteum must be removed; otherwise, adhesions to the artery can persist. The artery is dissected circumferentially and displaced laterally. In some cases, degenerative changes of the zygapophyseal joint can result in protrusion and compression of the artery, and it must be removed. At the end of the procedure, the artery should be free of all restrictions.

**Vein Grafts.** Vein grafts can be used to connect the middle vertebral artery to the CCA (see Fig. 102–8B), subclavian artery, or ICA. In 1966, Clark and Perry used a saphenous vein graft to connect the ECA to the vertebral artery at C2-3. The advantage of the ECA as the donor supply is that the proximal and distal anastomoses do not interfere with the cerebral circulation. A disadvantage occurs when the source of blood is from the ICA or CCA. Harvesting the vein and the potential for graft occlusions may pose a problem.

Synthetic grafts have also been used with similar donor sources. However, they have other disadvantages, including infection and pseudoaneurysm formation. Further, the use of grafts is limited over regions of constant movement because of rigidity and the corresponding wear caused by traction.

Pritz and associates reported using the trunk of the ECA to connect to the middle vertebral artery after an aneurysm was found at C4. The vertebral artery was ligated distal to the aneurysm, and the trunk of the ECA was connected to the vertebral artery. In some cases, if the ECA is too short to reach the vertebral artery, use of the occipital artery can be considered.

**Middle Vertebral Artery Endarterectomy.** In the case of limited focal stenosis of the middle vertebral artery, a selective vertebral endarterectomy can be performed (see Fig. 102–8C). The artery is removed from the transverse foramen and incised vertically. The procedure is performed in the standard fashion with the artery clamped proximally and distally. The plaque is removed in its entirety. When the proximal artery is occluded into the middle segment of the vertebral artery, this approach is used to access the vertebral artery. A limited endarterectomy is performed with a possible vein graft or transposition from the middle vertebral artery to the CCA.

**Transposition of Middle Vertebral Artery to Common Carotid Artery.** Transposition of the vertebral artery to the ICA, ECA, or CCA is feasible but can be surgically challenging. The main concern is to dissect enough of the vertebral artery to reach the donor site (see Fig. 102–8D). It is easier to use a vein graft or to go higher to the distal vertebral artery.

**CASE HISTORY 3**

A 53-year-old right-handed man suffered "black-out" episodes when he turned his neck to the right while driving. The patient had no significant medical history, except for a 30-year smoking history and neck pain during the past 5 years. His neurological examination was unremarkable.

Plain cervical radiographs showed degenerative disease of the spine with a large osteophyte at C6. After an extensive evaluation, the patient underwent aortic arch and dynamic four-vessel angiography. The right vertebral artery was occluded at the C6 foramen when the patient's head was turned to the right (Fig. 102–9).

The patient underwent an anterolateral approach. Cer-
FIGURE 102-7. Anterior approach to the middle vertebral artery. A, The incision is placed along the medial border of the sternocleidomastoid muscle. B, The skin is retracted, and the platysma is left intact initially until it is completely exposed. Then an incision is made longitudinally along the anterior border of the sternocleidomastoid muscle. C, By blunt dissection, a plane is developed between the strap muscles, trachea, and esophagus, which are retracted medially, and the sternocleidomastoid muscle and carotid sheath, which are retracted laterally. The longus colli muscle can be seen on the anterior vertebral body. D, The vertebral artery is exposed by drilling away the bone surrounding the transverse foramen.
vical radiography was used to identify the center of the incision at C6. The osteophyte was drilled off and removed with curets. The artery was dissected circumferentially and displaced laterally. The patient did well postoperatively and had no symptoms immediately after surgery.

**Surgery of the Distal Vertebral Artery**

The distal vertebral artery is vulnerable to blunt trauma, and injury to the intima can result in thrombosis, embolization, and dissection. This region has a high incidence of AVF formation and aneurysmal degeneration. Patency of the distal segment is often maintained through cervical collaterals. In many cases, if angiography reveals collaterals from the occipital artery, this artery is left intact.

Before planning any type of procedure, surgeons must consider the biomechanics of the spine at this level. Rotation occurs with the axis line posterior to the neck. Therefore, it is best to place grafts posteriorly to avoid undue torsion. An appropriate length must be used to avoid kinking or torsion.

**Approach to the Distal Vertebral Artery.** The approach to the distal vertebral artery depends on the revascularization technique used. The most common techniques are the anterolateral approach and the posterior approach. The former is used for an ECA-to-distal vertebral artery bypass or an occipital-to-distal vertebral artery bypass. These arteries can be anastomosed directly end to side or end to end, or an interposition graft from a vein or the radial artery can be used to connect the arteries. The latter approach is used to gain greater exposure of the vertebral artery for an occipital artery-to-distal vertebral artery bypass or to decompress an obstructed vertebral artery.

In the anterolateral approach, the exposure is made high in the anterior triangle. In some cases, disarticulation of the jaw provides additional exposure. The incision is made on the medial edge of the sternocleidomastoid muscle and extends in a curvilinear fashion over the mastoid bone (Fig. 102–10A). The incision is brought down to the platysma muscle, which is separated from the subcutaneous tissue. Along the medial border of the sternocleidomastoid muscle, the carotid sheath is entered, and the internal jugular vein is identified. The parotid gland is freed from the sternocleidomastoid muscle and reflected anteriorly. In the posterosuperior corner, the greater auricular nerve crossing the sternocleidomastoid muscle is sacrificed. A self-retaining retractor is placed to retract the internal jugular vein medially and the sternocleidomastoid muscle laterally (see Fig. 102–10B). Below the sternocleidomastoid muscle, the accessory nerve is visible. This nerve is protected by placing a loop around it and retracting it laterally. The belly of the digastric muscle is retracted superiorly, or it can be divided. Below the digastric muscle, the Cl tubercle is palpated. The fascia is cleared, and the fibers of the levator scapulae and splenius cervicis become visible (see Fig. 102–10C).

The C2 tubercle is also palpated, and the levator scapulae is cut to reveal the anterior ramus of the C2 nerve root, which passes laterally to the vertebral artery. Cutting this nerve exposes the vertebral artery (see Fig. 102–10D). Dissection of the overlying tissue reveals a venous plexus surrounding the vertebral artery. Careful coagulation or the use of Gelfoam prevents injury to the artery. Approximately 1 to 2 cm of vertebral artery is exposed. Further exposure can be obtained by removing the lateral wall of the transverse foramen of Cl.

**External Carotid Artery-to-Distal Vertebral Artery Bypass.** This technique requires a carotid bifurcation free of disease and a long ECA trunk. The anterolateral approach is used to isolate both the vertebral artery and the ECA. The major drawback of this procedure is the need to mobilize the ECA to reach the vertebral artery. The ECA is skeletonized, and all its branches are divided and ligated before the appropriate length is selected (Fig. 102–11A). We sometimes leave the occipital branch intact, primarily because musculoskeletal branches from the occipital artery always feed the more distal vertebral artery. The ECA is mobilized laterally either below or above the ICA and connected to the distal segment of the vertebral artery by an end-to-end
FIGURE 102-10. Anterolateral approach to the distal vertebral artery. A, The incision is placed along the medial border of the sternocleidomastoid muscle and extends posteriorly over the mastoid bone. B, The skin is retracted and the platysma is cut, exposing the sternocleidomastoid muscle with the great auricle nerve, which is also cut. The levator scapulae muscle, which covers the anterior ramus of C2, is visible. C, The levator scapulae muscle is cut to reveal the anterior ramus of C2. D, The vertebral artery is revealed. Medial exposure of the carotid sheath shows the common, internal, and external carotid arteries.
Vein Bypass from the Distal Vertebral Artery. Using the anterolateral approach, both the vertebral artery and the carotid artery are exposed. The donor site can be the CCA (see Fig. 102-11B), ICA, or ECA (see Fig. 102-11C).

A saphenous vein of selected length is removed from the leg. The vein is prepared, and its valves removed. The patient is given heparin (5000 U). After 5 minutes, the vertebral artery is gently pulled up with the loop to isolate a 2-cm section. Two Sugita clips are used to isolate the vertebral artery, and an incision is made equivalent to the fish-mouth end of the vein graft. The vein is connected to the vertebral artery with an end-to-side anastomosis using 8-0 polypropylene. If the artery is occluded in the proximal segment, the ECA and vertebral artery can be anastomosed end to end. The J-clamp is removed. If backflow through the graft is good, a temporary clip is used to occlude the vein.

The proximal end of the graft is passed below the CCA or ECA. The CCA (see Fig. 102-11B) is cleared of any surrounding tissue, and a cross-clamp is applied to its proximal and distal portions. Using an aortic punch, a 4- or 5-mm elliptical arteriotomy is made in the posterior wall of the CCA. With 6-0 polypropylene, the vein graft is anastomosed end to side. Again, backflow is allowed from the vein and distal CCA before the final suture is placed. The clamp on the proximal carotid artery is then removed.

The ECA can be anastomosed end to end to the proximal portion of the artery from the vein graft. The distal portion of the ECA is tied off permanently (see Fig. 102-11C).

We do not recommend the use of a vein graft for the ECA-to-distal vertebral artery bypass (see Fig. 102-11C), because rotation of the head often leads to thrombosis of the graft. This is especially true if the graft is passed below the carotid artery.

Occipital-to-Distal Vertebral Artery Bypass. As discussed, collateral blood flow to the distal vertebral artery comes from the occipital artery. In such cases, transposing the occipital artery directly to the distal vertebral artery has minimal effect. In cases of acute occlusion with inadequate collateral blood flow, a simple anastomosis between the occipital artery and the distal vertebral artery from the anterolateral approach can be used (see Fig. 102-11D).

The posterior approach for an occipital artery-to-distal vertebral artery bypass has been advocated by others. This approach is more familiar to neurosurgeons because the patient is in a full prone or three-quarter prone position. The incision is made from the C3-4 spinous process to the inion in a hockey-stick fashion to the mastoid bone. Details of this approach can be found elsewhere. All occipital approaches have the same disadvantages, with the added challenge that the occipital artery tends to be more tortuous as it emerges from the trapezius muscle, and large lengths of it can be difficult to isolate. However, the distal vertebral artery is easily accessed with a large exposure from the dura to C1 by removing the C1 foramen as inferiorly as the exit of C2.

Decompression of the Distal Vertebral Artery. An occasional patient may have severe local obstruction of the vertebral artery caused by compression from arterial branches or neighboring nerves. This can easily be treated by using the posterolateral approach with division of the obstruction.

Sometimes acute angulation or constriction can compromise blood flow. In such cases, the vertebral artery is removed as inferiorly as C3. The redundant section is cut, and an end-to-end anastomosis is reestablished. Constriction can also be caused by head rotation. In some reported cases, occlusion of the distal vertebral artery followed lateral head rotation. Traditionally, these patients were simply fused between the skull, C1, and C2. In younger patients, however, it is best to explore the anatomy and remove the pathology. If the artery is too short, an appropriate bypass should be used.

CASE HISTORY 4

A 77-year-old right-handed man was admitted to another hospital with pulmonary edema and right-sided weakness. In the emergency room he became hypotensive and unresponsive. He was hospitalized, and his neurological examination revealed double vision, nystagmus, dysphagia, and paresthesias of the hands and feet. MRI of the brain showed right medullary and cerebellar infarctions in the territory of the right posterior inferior cerebellar artery. MRA showed narrowing of the basilar artery with small vertebral arteries. His electrocardiogram was abnormal, and congestive heart failure developed. After medical management, he was transferred to a rehabilitation program.

The patient was transferred to our institution for further evaluation. Four-vessel cerebral angiography showed both carotid arteries to be normal, with good filling of posterior cerebral arteries via patent posterior communicating arteries. His left vertebral artery was narrow and terminated in the posterior inferior cerebellar artery. His right vertebral artery was 90% stenotic at its origin on the subclavian artery (Fig. 102-12) and fed the basilar artery by musculoskeletal branches from the occipital artery (Fig. 102-13). The basilar artery was poorly visualized, but segmental stenosis of this artery was possible. Phase-contrast MRI showed that blood flow in the basilar artery was 28 mL/minute. Blood flow in the right vertebral artery was 12 mL/minute. Both values were substantially low compared with normal.

Based on these findings, the patient’s symptoms were attributed to hypoperfusion of the basilar artery. Although his vertebral artery filled, it was segmented. Therefore, it was not clear if the entire vertebral artery was patent. Angioplasty of the proximal vertebral artery stenosis was recommended. This procedure failed because the lesion could not be penetrated, and an ECA-to-distal vertebral artery bypass was recommended.

An anterolateral approach followed. Because the patient’s occipital artery supplied musculoskeletal branches that fed the vertebral artery, the only branch from the
ECA that was not ligated was the occipital artery. An end-to-end anastomosis was completed between the ECA and the distal vertebral artery. Intraoperative flow measurements showed that when the ECA proximal to the occipital artery was temporarily occluded, there was no flow in the vertebral artery. The patient tolerated the procedure without difficulty. Phase-contrast MRI showed that the blood flow in the basilar artery was 50 mL/minute. He was returned to his rehabilitation program with no further symptoms.

Postoperative angiography showed patent distal vertebral artery-to-ECA anastomosis (Fig. 102-14). With better flow in the basilar artery, the segmental stenosis of the vertebral artery was more noticeable. The patient is now asymptomatic. If further symptoms are noted or if follow-up phase-contrast MRI shows a substantial decrease in
Basilar artery flow, the patient will be considered for angioplasty of the basilar artery via his new anastomosis.

OUTCOMES AND COMPLICATIONS

Two studies have reported outcomes from medical therapy for extracranial vertebral artery disease. Millikan and colleagues showed a decline in the mortality rate from 43% to 14% when heparin was used systemically to treat extracranial vertebral artery disease. Twenty years later in a 4-year follow-up, Whisnant and coworkers reported that the incidence of brainstem stroke decreased from 35% to 15% when oral anticoagulants were used. In neither study were the patients chosen randomly, nor did most of the patients undergo angiography. A well-controlled, randomized, multicenter study is needed to address the efficacy of medical therapy for extracranial vertebral artery disease. Meanwhile, outcome studies on the use of antiplatelet medications and anticoagulants in the anterior circulation and the use of warfarin for intracranial disease are well documented. The findings can be applied to the extracranial vertebral arteries, but with caution.

There are few studies on the complications and outcomes associated with endovascular management of extracranial vertebral artery disease. During a 12-year period, Beaujeux and associates treated 46 patients with AVF involving the extracranial vertebral arteries, 35 of which were treated by endovascular therapy. Thirty-two (91%) of these patients had complete occlusion, and three patients (9%) had partial occlusion. They reported no complications.

Few studies have reported long-term outcomes for percutaneous transluminal angioplasty of the extracranial vertebral arteries. Higashida and coworkers performed percutaneous transluminal angioplasty for hemodynamically significant stenosis of the proximal vertebral artery in 34 cases and of the distal vertebral artery in 5 cases. For the former, three cases of postprocedural transient ischemic attacks occurred. Short-term follow-up showed restenosis in three patients, two of whom again underwent angioplasty. In 12 months, symptoms in all patients improved, except for one who died of an unrelated aneurysm. For the distal vertebral artery, two patients died from complications during the procedure. The three other were symptom free 12 months later.

In 1996, Storey and colleagues observed restenosis within 3 months of angioplasty of the vertebral artery. Subsequently, they recommended the use of stents. In the next few years, the long-term outcome of angioplasty with and without stenting compared with medical therapy for vertebral artery stenosis will be available with the results of the Carotid and Vertebral Artery Transluminal Angioplasty Study.

The outcome of patients undergoing surgery of the external vertebral artery depends on the cause of the disease, the segment of the vertebral artery affected, and the type of treatment performed. In the hands of an experienced surgeon, surgery is associated with a very low mortality rate, and life expectancy is not substantially different from that of the general population with cerebrovascular disease. These individuals usually die within 10 years of cardiac disease. Berguer detailed the long-term outcome and experience with surgery of the proximal vertebral artery. Of 230 patients, 2 died. Complications included recurrent laryngeal nerve palsy in 2%, Horner's syndrome in 15%, lymphocele in 4%, chylolthorax in 0.5%, and im-
mediate thrombosis in 1%. Symptoms resolved substantially or were cured in 83% of the patients. Patency rates were 95% and 91% at 5 and 10 years, respectively.

For middle vertebral artery disease, decompression for osteoarthritic spurs was associated with excellent results in series by Hardin and colleagues and Nagashima. Diaz and coworkers reported surgical treatment (vertebral endarterectomy and vertebral-to-carotid transposition) in 11 of 12 patients with middle vertebral artery disease. Patency was found in 11 of the 12 patients. One patient had an occluded graft and continued to have transient ischemic attacks. VBI, however, was relieved in all patients. Follow-up ranged from 4 to 34 months.

Berguer and colleagues published the outcomes of 100 consecutive reconstructions of the distal vertebral artery over a 14-year period. They performed 72 distal vertebral artery-to-CCA, -ICA, or -ECA venous grafts. Twenty-three procedures were transposition of the distal vertebral artery to the ECA or occipital artery. In general, however, as more sophisticated endovascular techniques are developed, the use of angioplasty and stent placement to treat occlusive disease will likely become more common. Such a trend has already occurred with the proximal vertebral artery. For the distal vertebral artery, ECA-to-vertebral artery transpositions or vein graft surgeries are the most common procedures used to revascularize this region and will continue to be used. The middle vertebral artery will not be operated on as frequently as the other segments unless compressive lesions are easily accessible. Other cases of segmental occlusion in this region can be approached more distally.

**CONCLUSION**

The literature reports many series using different approaches to the extracranial vertebral artery with great success. Table 102–2 summarizes the endovascular and surgical approaches to the extracranial vertebral artery that we believe should be initiated before more sophisticated techniques are used. We must emphasize, however, the need to analyze the clinical and laboratory data of each patient methodically and to define the cause of the symptoms. If surgical intervention is indicated, the choice of procedure must optimize blood flow while minimizing the treatment risk of death or morbidity to the patient.

There will always be unique cases that require special approaches to the three segments of the extracranial vertebral artery. In general, however, as more sophisticated endovascular techniques are developed, the use of angioplasty and stent placement to treat occlusive disease will likely become more common. Such a trend has already occurred with the proximal vertebral artery. For the distal vertebral artery, ECA-to-vertebral artery transpositions or vein graft surgeries are the most common procedures used to revascularize this region and will continue to be used. The middle vertebral artery will not be operated on as frequently as the other segments unless compressive lesions are easily accessible. Other cases of segmental occlusion in this region can be approached more distally.

**REFERENCES**