In the past decade, vertebral artery surgery has been stimulated by many diagnostic and therapeutic developments. In 1976, the feasibility of vertebral artery anastomosis at the C1–C2 level was first demonstrated (Carney et al., 1976). By 1978, reports of vein bypass to the vertebral artery at the skull base began to appear (Carney and Anderson, 1978).

The skull base has been called a neurovascular-neurosurgical no-man's land. The factors that combined to bring about the emergence of vascular surgery of the vertebral artery were multidisciplinary; neurosurgeons approached this region from above, and vascular surgeons from the neck. Neurotologic surgeons were already "at home" at the skull base but not yet active in neurovascular reconstruction.

The technical ability to reconstruct the vertebral artery at the skull base demolished the nihilistic attitude toward hindbrain ischemia. Radiological and clinical observations involving all age groups began to appear with greater frequency, treatment became more aggressive, and our perspective broadened. Thus, vertebral artery surgery has become more sophisticated over the past decade and has radically changed the way we treat vertebral artery disease. Brain function is now directly related to brain perfusion. New techniques for measuring brainstem perfusion with high resolution (see Figure 30–1) provide not only an objective hemodynamic basis for selecting patients for vertebral artery surgery and for evaluating its hemodynamic results, but also a method for calibrating neurofunctional testing as a reflection of brain perfusion. Thus, the electroencephalogram (EEG) has become the instrument of choice for monitoring cerebral perfusion in the operating room, and a tool for the measurement of brain perfusion as well as the standard with which other tests are compared.

The major roadblock to relying upon brain perfusion clinically was philosophical: the physiological standard would displace the anatomical "gold standard" of angiography, disability/dysfunction would become an indication for surgery, the measure of successful therapy would be not death or paralytic stroke but the relief of disability, and, finally, sensory and perceptive disorders could be evaluated for a hemodynamic etiology. Now it is accepted that brain function and perfusion, and not the angiogram, are the measures of adequate blood flow, and the disability, impairment of function, and focal ischemia are the measures of need. Congenital vascular patterns and trauma affect all ages; atherosclerosis is not the only disease that involves the vertebral artery.

The early misconceptions also created significant obstacles to the understanding of vertebral circulation. Hindbrain ischemia generally produces disability rather than death; but even when death occurs, the neck is usually not examined in the course of an autopsy, thus vascular pathology in that region goes undetected. Disequilibrium, the most sensitive manifestation of brain-stem dysfunction, can force a person to crawl or lie supine in order to improve brain perfusion and protect vital
functions, yet the episode leaves no "proof" of its passing. Furthermore, the manifestation of hindbrain ischemia often does not direct attention to the brain, like blindness directs attention to the eye, or deafness to the ear, or respiratory arrest to the lungs. Only a high degree of clinical astuteness prompts recognition of the true underlying pathology, i.e., abnormalities in the vertebral blood flow. This hemodynamic approach to the entire brain provides us with both a new nonneurologic language and with substantial useful tools for a sound approach to brain ischemia.

FUNCTIONAL ANATOMY OF THE VERTEBRAL ARTERY

The vertebral artery is characterized by its small diameter (0.5 to 5.5 mm) relative to its great length (15 to 35 cm). It is often asymmetrical and has many segmental branches before the paired arteries finally fuse into the basilar artery. The vertebral artery (see Figure 30-2) arises from the subclavian artery, courses within the bony canals of the cervical vertebrae anterior to the cervical nerve roots, and is surrounded by veins and nerve elements. It reverses direction on the atlas before piercing the dura mater to enter the cranium.

The volume of brain perfused by the vertebral artery varies individually from the region of a single end-artery to the entire brain. Compared with the carotid artery, it is a high-resistance vessel but vertebral revascularization, especially by a distal bypass, commonly results in dilatation of the vessels and extension of the vascular bed perfused.

The vertebral artery is best divided into segments according to the surgical exposure required. The anterior approach provides access to the vertebral artery within the neck. The proximal vertebral artery extends from its origin, which is variable, to the point of entry into the vertebral foramen, usually at C6. It is fixed in its course to the cervical spine by the vertebral nerve and by sympathetic fibers. It may be best approached through a supraclavicular incision. The midvertebral segment of the artery may be approached by a longitudinal incision...
Head turned rt.

Figure 30-2. The effect of head rotation upon the vertebral artery. Head rotation, indicated by large curved arrows, carries the transverse process of the atlas forward which may stretch the artery, disrupt intima and cause thrombosis or dissection. The major contribution to head rotation occurs between C1 and C2 with a lesser contribution from the lower spine. See arrows 1 and 2 demonstrating the minimal rotation of C5 on C6. (From Krueger, B.R. and Okazaki, H.: Vertebral basilar infarction following chiropractic manipulation. Mayo Clin. Proc., 55:322–32. 1980.)

paralleling the course of the sternocleidomastoid muscle. The distal vertebral artery, the term designating the vessel at the level of the axis and the atlas, can be exposed through a high transverse cervical incision. Incisions from one section to the other may be continuous. The intracranial vertebral artery, which includes the suboccipital vertebral artery, must be approached posteriorly.

HEMODYNAMIC BASIS OF VERTEBRAL ARTERY SURGERY

All components that effect blood flow to the brain must be regarded as parts of a single system (Carney and Anderson, 1981; Carney et al., 1981; Yates and Hutchinson, 1961). Evaluation of brain ischemia therefore involves the assessment of the hemodynamic levels, i.e., the system, the "precircle" arteries, the circle of Willis, and the end-arteries. Generalized brain ischemia may be due either to low cardiac output or to combined carotid and vertebral disease. About one third of patients presenting with symptoms of hindbrain perfusion deficit in reality suffer solely from a generalized brain ischemia due to low cardiac output.

The Precircle Level

Arterial obstruction proximal to the circle of Willis may produce ischemia either in the territory directly supplied or in a remote site. Ordinarily, the reserve flow from one carotid to the other will compensate, but this is less likely
to occur between the carotid and vertebral systems, where such compensatory flow is dependent upon the posterior communicating arteries, the flow capacity of the patent vessels, and the resistance of the affected vascular bed. Normally, the pressure gradient between primary arteries and the circle is low but the flow capacity is high. In contrast, the gradient between the circle and the vascular bed of the end-arteries is high and the flow capacity is limited.

In a case of carotid obstruction, perfusion of the cerebral hemisphere may remain normal because of increased flow through the vertebral system, yet the brainstem itself may be ischemic (Bohmfalk et al., 1979). This hindbrain ischemia may be relieved either by carotid endarterectomy to improve carotid flow or by vertebral artery reconstruction to increase vertebral artery flow. Vein bypass to the vertebral artery at the skull base results in maximal increase in blood flow and flow capacity of the vertebral basilar system.

**The Circle of Willis**

The circle of Willis is incomplete when any of the carotid or vertebral arteries have poor or no communication with it or whenever there is no connection between the segments of the circle itself. A hypoplastic vertebral artery ending in a posterior inferior cerebellar artery is an example of such an incomplete circle. It is also a true end-artery, and its occlusion may result in focal infarction of the brain stem and cerebellum. A hypoplastic vertebral system is also incomplete because the basilar artery terminates in the superior cerebellar arteries and has no primary communication with the circle. A hypoplastic system is one of high resistance because its diameter is narrow but its length is unchanged. Hypoplastic systems are commonly found in patients with longstanding symptoms. The primary function of the circle of Willis is to maintain blood flow to the brain during the intermittent obstruction of the cervical vessels produced by head motion. In this process, the principle of competitive flow applies, i.e., large primary vessels communicate freely with one another and have the capability of bidirectional flow. The cardinal characteristic of the circle is the ability of the primary channels to alter flow rapidly in response to slight changes in pressure. The circulation through the circle is dynamically balanced at "dead points," which may rapidly change with alteration in flow. This capability to immediately increase flow through the primary limbs of the circle was designated as "cognate" by Green and associates (1944), as "primary" by Nornes et al. (1979) and as "reserve" by Carney and Anderson (1981). Collateral flow is blood flow delivered through secondary channels and usually it is not immediately available. An artery, carotid or vertebral, directly supplying the circle is a primary limb of a high-flow reciprocating system. Competitive flow within this system is characterized by a low-pressure gradient, high-flow capacity, and ability to rapidly equilibrate flow and pressure. Even in the presence of total occlusion of a major limb, e.g., carotid occlusion, the resultant resting pressure gradient between limbs may be small. Only under the stress of increased flow demand or contralateral carotid compression, can the hemodynamic impact of minor obstructions in primary vessels be demonstrated.

Blood flow within an end artery to the brain is determined by a significant pressure gradient, it tends to be unidirectional, and it has a low flow capacity. Competitive flow within the end arteries after microvascular reconstruction may cause thrombosis at the junction of the end-artery with the circle of Willis or at the site of proximal stenosis where the pressure and flow are balanced: the dead point. For any end-artery reconstruction to remain open, the graft inflow must sustain the pressure within the vessel grafted. To prevent occlusion of the proximal end-artery, end-artery pressure must exceed that in the circle of Willis.

In general, reconstruction of a large vessel results in a better hemodynamic response than does that of a small vessel, and proximal reconstruction is more beneficial than end-artery reconstruction. Small-vessel reconstruction fares better in the presence of a high gradient and in the absence of significant competitive flow. Small vertebral arteries occlude more often than do large ones. Marked improvement of flow in one vertebral artery may be followed by a reduction in flow and diameter of the contralateral vessel. In an asymmetrical vertebral system, reconstruction of the larger vessel results in the greatest improvement in brain perfusion.

The extracranial completion of an incomplete circle of Willis can be achieved by creating a wide open communication between the carotid and vertebral systems in the neck. An anastomosis in the neck base will work well
providing that there is no distal obstruction. In the presence of carotid siphon stenosis, vein bypass from the common carotid to the vertebral artery at the skull base may result in such high bypass flow that the internal carotid flow becomes severely reduced and thrombosis occurs.

The Post-Circle End-Artery

An end-artery, as distinguished from other small vessels, arises from the circle of Willis, the basilar artery, or the vertebral artery, and supplies a defined territory of the brain. Its presence is constant and the blood flow through it is determined by a high-pressure gradient. End-artery occlusion often results in the infarction of a specific segment of the brain. Hypotension within an end-artery is commonly due to extracranial arterial obstructions, but could be also caused by obstruction within the end-artery itself. If an end-artery is small, the resistance to flow will be high. Although some end-arteries are accessible for microvascular reconstruction, others are not.

End-arteries that arise from the basilar artery and supply the brain stem differ from the major cerebral and cerebellar vessels in that they supply regions so small that their occlusion may produce an infarct of only 3 to 5 mm in diameter, an arrangement useful in protecting against extensive infarctions. In the presence of basilar artery disease, these vessels show increased vulnerability to reduced systemic pressure and to decreased arterial blood flow (Camp, 1984; Kistler et al., 1984).

Vascular Obstruction by Head Motion

In extreme head positions, one or more arteries, carotid and/or vertebral, normally obstruct. If the cardiac output is normal and if the circle of Willis is intact, there will be sufficient blood flow in the remaining vessels to maintain brain perfusion and normal brain function. The vertebral artery is particularly vulnerable to obstruction by normal head rotation (see Figure 30–3). To accommodate for this vulnerability, the vertebral system has a feature unique in the body: two vessels fuse in the direction of flow to form one vessel, the basilar artery. Nowhere else in the body does this occur. By this mechanism, the contralateral vertebral artery, if it is patent, can rapidly compensate for the transient obstruction.

Pathology of Head Motion

Hyperextension of the head produces stress at the occipito-atlantoid articulation. This is true even in the normal person, but is a greater problem in patients with anomalies of the skull base, or variant vascular patterns. In some patients the posterior, inferior cerebellar artery will prolapse through the foramen magnum into the neck and become particularly vulnerable to trauma.

Hyperextension of the neck stretches the proximal vertebral artery and produces maximum stress at the point where the vessel is fixed to the spine by the vertebral nerve. This may result in shearing of the intima, dissection (Simeone and Goldberg, 1968), and thrombosis. Extension injuries could also produce soft-tissue injuries on the anterior aspect of the cervical spine whereas flexion trauma may cause compression fractures of the anterior aspects of the bodies of the lower cervical vertebrae (see Figure 30–4).

The greatest contribution to head rotation occurs at the C1–C2 articulation, lesser
amounts take place in the lower cervical spine. Hypermobility which may predispose to vertebral artery injury, may occur with congenital deformities of the skull base, with traumatic disruption of ligaments in cases of rheumatoid arthritis. Congenital fusions, fractures or arthritis may restrict neck motion in one area, only to result in compensatory increase in motion in another region, resulting in arterial injury. Compression of the proximal vertebral artery by tendons of the scalenus anticus muscle may also cause obstruction whenever the head is turned extremely to the contralateral side. Kovacs (1955) pointed out that the posterior placement of the vertebral foramina in relation to the body of the vertebra increases vulnerability to compression of the vertebral artery by the subluxation of the apophyseal articulation. Brain (1963) emphasized the role these joints play in the production of symptoms. Studying the matter by cineangiography, he noted that whenever the head is rotated to the opposite side, the vertebral artery at the level of the atlas moves forward, angles acutely in relation to the transverse process of the axis, and is drawn up and out of the vertebral canal of the lower cervical vertebra. Trauma to the spine, which fixes the artery to the canal, eliminates this mobility. Norrell (1975) classified cervical spine trauma according to the mechanism of injury: extension, flexion, extension with rotation, and lateral flexion. These are normal mechanisms of motion made pathological by varying circumstances. Most head positions when stressed obstruct the vertebral artery and may produce symptoms of ischemia. When sufficient force is applied rapidly, the integrity of the arterial wall suffers and rupture or dissection can occur. A stressed head position can cause transient ischemic and arterial trauma, especially to the vertebral artery. This mechanism is primarily responsible for stroke in the young (Klein et al., 1976; Latchaw et al., 1974; Yates, 1959; Zimmerman et al., 1978). Abnormalities of the occipital-atlantoid and atlantod-axial articulations (Wackenheim, 1974), especially when combined with trauma, may contribute to vertebral artery injury and thrombosis. Computed tomographic scans can provide high quality pictures of the cross-sections of the spine for identifying skull base pathology. Conventional x-rays of this region are of limited value.

**PATHOLOGY OF VERTEBRAL ARTERY DISEASE**

Disease of the vertebral artery is neither confined to any one age group nor to a single
Intracranial atherosclerosis is seldom localized enough to permit the performance of an endarterectomy, and extensive involvement of the basilar artery usually also prevents the performance of such a procedure.

Fibromuscular dysplasia occurs less frequently, but may involve any segment of the vertebral artery and could develop at any age. High quality selective angiography with magnification is necessary to identify this lesion.

Dynamic Obstruction

The loss of elasticity in the vascular wall results in dilation and elongation of the arterial system, which may give rise to recurrent obstruction aggravated by stressed head or arm positions. Because the obstruction is intermittent, collaterals do not develop. Extrinsic fixation, constriction, and entrapment of the proximal vertebral artery by sympathetic
fibers, by the vertebral nerve, or by the ansa subclavia serve as a point of fixation of the artery to the spine. With advancing age, dilatation and elongation of the arterial system elevate the aorta in the chest and the subclavian artery in the neck. The latter rotates as it rises because it is fixed to the first rib by the scalenus anticus muscle and it is tethered by the vertebral artery. The vertebral artery is, in turn, attached to the spine by nerve fibers. The dilated subclavian artery can be palpated and is often seen above the level of the clavicle so it can be readily delivered into the surgical field. The vertebral artery at C1-C2 is tightly secured by dense fibrous attachments. Nerves, veins, and/or small muscular arterial branches may cross the artery in an aberrant fashion, producing obstruction if the head is kept in a particular position. The artery may also penetrate a nerve and be caught in a snare that may tighten or loosen as the head is rotated. The turbulence thus created results in localized dilation of the involved artery segment which may cause platelet aggregation and embolism. Redundant loops below the atlantoaxial joint tend to have acute angulations which can lead to dynamic obstruction.

Mechanical head motion or trauma can result in the disintegration of the wall of the vertebral artery. The medial layer of the vertebral artery is elastic; the intima is not. The stress of stretch, distention, or direct compression may fracture the intima and leave the media intact. This traumatic etiology of dissecting aneurysm is well documented in the neurosurgical literature. Acute dissection and occlusion of the distal basilar and intracranial vertebral arteries during angiography have occurred (Takahashi, 1974), and were presumably caused by the rapid dilation of the arteries due to injection under pressure. The separation of intima from media is commonly seen during angiography. Furthermore, repeated trauma could compromise the integrity of the wall, making it friable and vulnerable to intimal separation. Acute dissection of the vertebral artery may be associated with delayed onset of symptoms caused by the extension of thrombosis into the posterior inferior cerebellar and basilar arteries. Fusion of the superior cervical ganglion with the anterior ramus of the second cervical nerve at the skull base may result in the erosion of the anterior wall of the vertebral artery. The damage to the arterial wall may make ligation the only possible course of action. Recurring occipital lobe embolism and severe disabling hindbrain ischemia can also be precipitated by minor accidents. If anticoagulants fail to prevent recurrence of emboli, ligation of the vertebral artery must be considered.

Dissection after reconstruction of a vertebral artery at skull base is associated with a state of predissection. In such cases, the artery is edematous and the flaky intima easily strips from the media with even minor surgical manipulation. The lack of adherence of the intima to the media is striking, and the inability to identify this problem preoperatively is frustrating. The operating microscope might be a most valuable asset in such a situation. One clinical finding is constant: pain localized over the artery. On angiography, close inspection may reveal loss of sharp wall definition.

Atherosclerotic stenosis with gradual progression will produce occlusion of the vertebral artery with reserved patency of the distal segment. Thrombosis arising out of ulceration, dissection, or trauma, however, will often propagate intracranially, but the onset of symptoms may be delayed. Although the principal source of embolism to the hindbrain is the vertebral artery itself, embolus to the forebrain often originates in the heart. Entrapment of the vertebral artery may also give rise to platelet aggregates at the site of stricture, which embolize to the occipital lobe causing acute visual field defects.

Although traumatic aneurysms of the vertebral artery are seemingly less common today than they were at the time of Matas (1893), they continue to occur. Whereas berry aneurysms are seldom seen on the vertebral artery, aneurysms and tortuosity of the vertebral artery may cause compression of the medulla or of a nerve root. Aneurysms of the subclavian artery may also encroach upon the vertebral artery and require vertebral reconstruction along with the repair of the subclavian artery. Arteriovenous fistula may be congenital or caused by fibromuscular dysplasia but more commonly by trauma. Congenital malformations of the head and neck may also be fed by branches of the vertebral artery.

The association of hindbrain ischemia with "whiplash" injury and with "thoracic outlet" symptoms is common. The onset of the thoracic outlet syndrome is often precipitated by whiplash injuries, and these patients have EEG findings that are frequently observed with hindbrain ischemia. Proximal vertebral artery obstruction caused by muscles and tendons were noted by Husni and associates (1966) and
Hardin (1965). The arterial pathology of the thoracic outlet syndrome is readily demonstrated by angiography (Bogousslavsky and Regli, 1985); however, compression of the brachial plexus and scalenus muscle is more difficult to appreciate because the outlines of soft tissues cannot be visualized by x-ray. In some cases thoracic outlet syndrome represents a hyperextension stretch injury to the brachial plexus. Stroke-associated thoracic outlet syndrome is an uncommon but a well-documented entity (DeVilliers, 1966).

SIGNS AND SYMPTOMS OF HINDBRAIN ISCHEMIA

Symptoms of hindbrain perfusion deficit are determined by the location and sensitivity of the ischemic area, as well as by the severity of the ischemia. In the presence of diffuse brain ischemia, symptoms are generated by the most sensitive site, often by the vestibular system. Diffuse ischemia with hindbrain symptoms must be differentiated from local ischemia with the same symptoms. Diffuse perfusion deficit must be approached with conservatism; focal ischemia must be dealt with aggressively. Often an associated second lesion of hemodynamic significance is present, usually obstruction of another vessel. Low-output syndromes, dehydration, or an unsuspected malignancy could also cause similar clinical pictures.

Focal Signs

Focal neurological signs neither identify nor localize vertebral artery obstructions. Although regional ischemia of the occipital lobe or the cochlear nucleus of the brain stem may be demonstrated, the site of arterial obstruction could well be the contralateral vertebral artery or even the carotid artery. Proximal obstruction can produce infarctions in the territory of end-arteries with discrete clinical manifestations, the Wallenberg's syndrome. Fisher and associates (1965) reported that of 16 fatal cases of lateral medullary infarction, the vertebral artery was occluded in 12 and the posterior inferior cerebellar artery supplying the region in two. Lateralizing signs of ischemia, especially in the territory of the basilar artery, are only randomly related to the side of vertebral obstruction (Hoobler, 1942). Williams and Wilson (1962) described the full scope of hindbrain symptoms and pointed out that one half of these patients have visual problems and two thirds have vertigo. They recognized that symptoms are primarily related to ischemia and not to infarction because the symptoms are generally reversible. Their clinical evaluation, however, involved speculation rather than proof about the immediate cause.

Disequilibrium

Central vestibular testing is directly concerned with brainstem and vestibular function and its modification by stressed head position and vestibular stimulation, which may be dependent upon blood flow within the vertebral artery. This technique has been found most useful in documenting brain-stem dysfunction. Brain-stem-evoked auditory responses have not been particularly helpful because the cochlea is less sensitive than the vestibular apparatus, and, to eliminate artifacts, the study must be done with a neutral head position and complete muscle relaxation. For us, brain-stem-evoked auditory potentials have not been useful in detecting vertebral artery obstruction produced by head position.

Loss of Hearing

Sudden deafness may be caused by reduced oxygenation of the perilymph. The level of oxygenation can be increased by artificially increasing the blood pressure (Fisch et al., 1984). While use of mixtures of carbon dioxide and oxygen have also been helpful in reversing this type of neurological deficit (Buonanno et al, 1984), they may also aggravate brain ischemia by increasing intracranial steals (Fisch et al., 1984). The onset of sudden deafness commonly occurs during sleep, which suggests that the reduction of blood pressure and/or cardiac rate may be a contributing factor to decreased brain-stem perfusion.

Respiratory Arrest

Respiratory arrest, a common emergency, is rarely considered to be caused by a brain-stem ischemic attack. Angiographic evaluation of such patients and their aggressive management can be rewarding (Nenci et al., 1983; Glenn et al., 1980). Similarly, apnea and cardiac rhythm disturbances occurring during sleep, although studied extensively, have sel-
dom been looked upon as manifestations of brain-stem ischemia.

**Provoking Respiratory Dysrhythmia**

Provocation of respiratory arrest or dysrhythmia by carotid compression is occasionally observed in the neurovascular laboratory. Kramer (1912) noted that injections of alcohol or chloroform into the carotid artery resulted in mild and transient manifestations, tachycardia and tachypnea; but injection into the vertebral artery resulted in hypotension, bradycardia, and respiratory arrest. Hardin (1965) presented four cases of patients presenting with acute respiratory arrest and paraplegia that recovered following emergency vertebral artery reconstruction. Thus, it appears that the control of respiration, heart rate, and blood pressure is mediated by the brain stem.

Nagashima (1970) evaluated the responses of the vestibular system during the temporary occlusion of the vertebral artery during surgery in the awake patient. He classified the resulting symptoms into three categories: (1) disorders of vision, (2) disorders of consciousness, and (3) visceral symptoms. His observations on abnormal ocular movements, ambylophia, alteration of visual fields, and disturbances of perception agreed with those of Hauge (1954). The level of consciousness acutely fell in five of the 25 patients studied but returned to normal after the occlusion was removed. In five patients, hypotension and bradycardia were preceded by nausea and vomiting. In one case, the vision suddenly blurred when the right vertebral artery was occluded for 30 seconds, after which nausea and vomiting occurred.

**Neurovascular Stress Testing**

The cardiovascular system in general and the cerebral circulation in particular have great reserve capacities. Both systems must function well to achieve effective brain perfusion, and both systems need to be tested under stress. The value of stress testing of the cardiovascular system has long been recognized (see Figure 30–6). The same is true of the blood supply to the brain. Common methods of stress testing of the cerebral circulation include the tilt table (Carney and Anderson, 1978; Furlan and Breuer, 1984), stressed head positions (Causse and Causse, 1979), inhalation of 5 percent carbon dioxide (Fisch et al., 1984; Furlan and Brewer, 1984), the treadmill (Carney and Anderson, 1978), central vestibular testing (Kumar, 1981), and carotid compression (Eikelboom, 1981). Other forms of commonly occurring stress include the induction of anesthesia, surgical occlusion of a vessel, and hypotensive anesthesia with or without cardiopulmonary bypass and angiography.

**Measurement of Brain Perfusion**

Static and dynamic computed tomographic (CT) brain scans or their functional equivalent should be performed on patients considered for hindbrain revascularization. Diagnosis and localization of brain ischemia are based upon the objective findings of infarction, hypodensity, and focal atrophy seen on conventional CT scan and upon ischemia determined by dynamic contrast-enhanced CT studies. The newer techniques, like stable xenon-enhanced CT dynamic scanning, position emission scanning, and magnetic resonance imaging appear even more promising. It is important to remember that these studies are only performed when the patient is supine with the head in neutral position. If the patient is young and if symptoms occur only under stress, the dynamic CT studies may be normal. In this case, functional equivalent studies, such as central vestibular studies under stress, will often be diagnostic.

**Selective Angiography with Magnification**

Selective angiography of the carotid and subclavian arteries is employed with rotational views of the vertebral artery using magnification. We seldom use digital subtraction angiography because of the necessity of high resolution of the vessel at the skull base. The anatomical findings on angiography should be correlated with the results of general and topical hemodynamic evaluation.

**TREATMENT ALTERNATIVES TO VERTEBRAL ARTERY SURGERY**

Treatment for annoying and disabling hindbrain ischemia should begin by discontinuing or reducing the dose of antihypertensive medication, minimizing postural hypotension, and regulating diet. For some patients, pressure gradient supports are beneficial. Surgical intervention and treatment performed either
selectively or during crisis requires detailed neurovascular workup.

Hypervolemic Hemodilution

Plasma volume expansion, bed rest, and neck support are usually effective in controlling acute crises where respiratory arrest is not a threat. Hypervolemic hemodilution is the single most effective nonsurgical therapy available for episodes of acute brain ischemia (Carney and Anderson, 1981b). Cardiac dysrhythmia must be controlled, and if necessary physiologic pacing is used.

Carotid Surgery

In the presence of large posterior communicating arteries, endarterectomy of a stenosed carotid artery may be effective in relieving hindbrain ischemia. The same may be true when the obstruction of the internal carotid artery is dynamic rather than static. Relief of hypoglossal carotid entrapment with obstruction may substitute for vertebral artery reconstruction (Carney and Anderson, 1981a).

Thrombolysis

Thrombolytic therapy has been used for acute carotid and vertebral artery thrombosis in selected cases. Nenci and associates (1983) reported four cases of acute brain-stem stroke treated with streptokinase and urokinase. Three of the patients whose embolism to the distal end of the basilar artery or stenosis of the basilar artery were confirmed by angiography responded well to therapy. The fourth patient, who had bilateral cerebral artery occlusion, died. This therapeutic approach may be useful in the early treatment of respiratory failure caused by acute brain-stem injury. It may also
have a place in treating delayed and late thrombosis at the site of surgical reconstruction.

Balloon Occlusion and Embolization

Interventional radiologists have used balloon catheters (DeBrun et al., 1978), both for trial occlusion of the subclavian and vertebral arteries (George and Laurian, 1979) before surgery and also for permanent occlusion as definitive treatment of arteriovenous fistulas in the neck after bypass to the distal vertebral artery. In the latter case, the vertebral artery is occluded at a site proximal to the bypass but distal to the fistula. Both embolization and balloon inflation, distal and proximal, can then be performed. Additional bypass to the vertebral artery distal to the arteriovenous communication permits occlusion without compromising brain perfusion.

Balloon Angioplasty

Percutaneous balloon dilatation of lesions in the proximal vertebral artery has recently been reported (Motarjeme et al., 1982). Percutaneous angioplasty of the basilar artery has not been reported. Intraoperative angioplasty of the basilar artery has unsatisfactory results. Disruption of the arterial media by the sharp edge of a calcified plaque, however, can result in intracranial aneurysm and hemorrhage (Sundt et al., 1980). Postoperative balloon dilatation of the stenosed bypass to the distal vertebral artery, however, may be considered in the future. The presence of the distal vertebral artery bypass permits access by catheter to the intracranial artery for balloon placement, angioplasty, embolization, or chemotherapy.

Spinal Fusion, Decompression

In some patients with impaired vertebral artery flow, operating on the vertebra may be preferable to direct surgery of the vertebral artery. Persons with congenital or acquired hypermobility at the skull base of the atlantoaxial articulation should be evaluated for treatment to restrict this abnormal mobility. Bypass to the distal vertebral artery can be occluded by the mechanical rotation of the head. The unstable spine is better served by stabilization and cervical spinal stenosis by decompression. However, posterior decompression may change the relative position of the neck and aggravate head hyperextension. Hindbrain ischemia due primarily to vertebral artery obstruction distal to C-1 will not be benefitted by surgery proximal to C1.

Surgical fusion of the atlas and axis has been considered in lieu of or in combination with vascular reconstruction. Verbiest (1969) was of the opinion that fusion with unilateral distal vertebral artery reconstruction is not necessary. Extensive resection of the transverse process of C1 may result in symptomatic instability of this joint. Further experience is needed in this area.

VERTEBRAL ARTERY SURGERY: GENERAL CONSIDERATIONS

Indications

The general indication for vertebral artery reconstruction is underperfusion of the brain caused by a hemodynamically significant obstruction of the vertebral artery (see Table 30-1). Reconstruction is thereby expected to relieve disability, enhance function, and prevent brain damage. Pain caused by bone erosion, pressure on neural tissue, or ischemia owing to arteriovenous malformation is also a valid indication for surgery. The specific hemodynamic objective of vertebral artery surgery is to enhance perfusion to a particular area of the hindbrain as a whole or to the forebrain by increasing collateral flow through the circle of Willis. Advanced cortical atrophy, hydrocephalus, brain tumor, and intracranial aneurysm are some of the principal contraindications to reconstruction of the vertebral artery. Other conditions that may compromise results include cervical spinal stenosis, abnormalities of the skull base or cervical vertebrae. spinal cord pathology, left ventricular dysfunction, cardiac dysrhythmia, and systemic hypotension. Overlooked obstruction of the carotid artery (Carney, 1981) and vertebral artery stenosis distal to the site of reconstruction could also cause unsatisfactory results after technically successful vertebral surgery.

Preparation for Surgery

For elective surgery the patient should be neurologically stable, i.e., with absent or controlled symptoms when provoked by passive tilt. For the acute case of hindbrain ischemia, hypervolemic hemodilution combined with
Table 30-1. Potential Indications for Reconstruction of the Distal Vertebral Artery at the Base of the Skull

- Obstructive pathology at the C1-C2 level
- Obstruction of the vertebral artery proximal to C2
- Acute thrombosis of the proximal vertebral artery with patent distal segment
- Chronic occlusion of the proximal vertebral artery with patent distal segment
- Failure of proximal reconstruction
- Ligation of proximal vertebral artery
- Aneurysm of the vertebral artery
- Arteriovenous fistula involving the midvertebral artery
- Irradiation of the lower neck
- Previous carotid subclavian bypass
- Increased hazard of proximal reconstruction: the anterior spinal artery arising from the subclavian artery
- Inoperable carotid lesions: to increase collateral flow to the forebrain as an alternate to extracranial-intracranial bypass
- Hypoplastic systems with incomplete circle of Willis
- Interventional access to the posterior fossa: for balloon placement, angioplasty, embolization
- Safeguard against neurological damage under cardiopulmonary bypass, hypotensive anesthesia, general anesthesia
- Reestablishment of vascular continuity disrupted by tumor and/or resection
- Preservation as a conduit when the internal carotid artery is occluded or likely to become occluded by ligation or thrombosis
- Completion of the circle of Willis—extracranial: when there is no effective posterior communication between isolated and hypoplastic systems and other vessels
- Control of recurrent occipital lobe embolism from the vertebral artery proximal to C2: anticoagulant failure

In summary, the selection of procedures for reconstruction of the vertebral artery at the base of the skull depends on the site and nature of the pathology, the findings at surgery, and the ability of the surgeon. The principal decision relates to the level at which the artery is to be approached. There is no one standard procedure and combinations of techniques are applied in various circumstances. The view of clinical experience with vertebral artery ligation may be helpful. Dandy (1944) reported a case of sudden death after momentary pinching of a vertebral artery in the operating room. Bakay and Sweet (1953) measured the intrarterial pressures in the posterior inferior cerebellar artery and also in the exposed vertebral artery between the occiput and the atlas before and after intracranial ligation of the vertebral artery for aneurysm. They concluded that the ligation of one vertebral artery is unlikely to reduce distal pressure and proximal ligation is therefore probably of little benefit in treating an aneurysm arising from the basilar artery. Shintani and Zervas (1972) noted that in 100 cases of ligation reported in the literature, the overall mortality rate was 12 percent.

Rainer and associates (1970) reported two deaths in 54 cases of vertebral artery surgery owing to brain damage. One patient died in surgery and the other was lost postoperatively. They therefore recommended the use of temporary shunting while the vertebral flow is interrupted during surgery.

Cormier and Laurian (1976) observed a 1.5 percent mortality rate in a total of 172 patients, in whom 119 vertebral arteries were reconstructed. No further details were given. Edwards and Mulherin (1980) reported neither mortality nor stroke in 204 reconstructions of the vertebral artery. Use of shunts was abandoned early in their series. Senter and Long (1983) reported use of a shunt during the reconstruction of the midvertebral artery. I have not employed shunts for either carotid or vertebral reconstruction for a decade, and others have taken similar positions. On the other hand, I give careful attention to plasma volume expansion and monitoring in the operating room. If a patient is very high risk, intolerance to the induction of anesthesia warrants cancellation of surgery.

**Selection of Procedures**

The procedure used is determined by the site of pathology, the nature of the pathology, the findings at surgery and the ability of the surgeon. The principal decision relates to the level at which the artery is to be approached. There is no one standard procedure and combinations of techniques are applied in various circumstances (Carney et al., 1981). Just as coronary bypass and carotid procedures have been performed simultaneously, reconstruction of
the proximal vertebral artery at the time of coronary bypass is also possible (Moran, 1980). Reconstruction of the vertebral artery at the skull base, however, is too complex to combine with cardiac surgery.

RECONSTRUCTION OF THE PROXIMAL VERTEBRAL ARTERY

Exposure

The proximal vertebral artery is most conveniently exposed through a transverse supraclavicular incision extending from the midline over the lateral edge of the clavicular head of the sternocleidomastoid muscle. The clavicular head can be divided or retracted medially. If only retracted, then the lateral edge must be mobilized widely. The scalene fat pad is incised medially and retracted laterally. The scalenus anticus muscle with the overlying phrenic nerve is exposed high to permit radical excision (see Figure 30-7). Attention is then directed caudally toward the first rib for completion of the resection. Muscle bands extending between the cords of the brachial plexus or other encroaching bands are excised.

The subclavian artery is mobilized first laterally, then medially. The thoracic duct, if injured, is ligated. An attempt is made to preserve the branches of the subclavian artery. Commonly, the arch of this artery rides high in the neck, far above the entrance of the vertebral artery into the foramen of C6. The overlying nerve fibers are frequently divided. The vertebral artery is mobilized to the vertebral foramen. Occasionally, tendinous attachments of the scalenus muscles encroach upon the vertebral artery and are resected. If the nature of the procedure requires ligation of the vertebral artery, this should be carried out near its origin from the subclavian artery to gain maximal length. The vertebral vein is ligated when encountered. If the subclavian artery appears to be friable or lies deep and the operative plan does not call for its exposure, then it should be avoided because minor injury may lead to further damage, requiring complex repair or even replacement by prosthesis. Unusual situations may require extension of the

Figure 30-7. The normal anatomy of the proximal vertebral artery is illustrated, showing the basic relationships of soft tissues. The cisterna chyli and vertebral nerve are not shown.
incision into the thorax or mobilization of the vertebral artery above the C6 level.

**Transsubclavian Endarterectomy**

Cate and Scott (1959) described an endarterectomy of the origin of the vertebral artery through the subclavian artery (see Figure 30-8). No incision was made in the vertebral artery.

**Endarterectomy with Patch Angioplasty**

Vein patch angioplasty (see Figure 30-8B) is greatly facilitated by the use of fine monofilament suture. If the origin of the vertebral artery is very low in the thoracic inlet, the site of planned arteriotomy may be difficult to reach. Transthoracic vertebral patch angioplasty (see Figure 28-9) has recently been recommended concomitantly with replacement of the proximal subclavian artery (Robicsek, 1985).

**Bypass to the Proximal Vertebral Artery**

Berguer and associates (1976) reported on a bypass to the proximal vertebral artery from the subclavian artery. Autogenous vein is generally used as the graft, but the use of prosthetic material has also been reported (Bohl et al., 1977). The distal anastomosis is usually per-

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**Figure 30-8.** Vertebral endarterectomy performed through the interior of the subclavian artery (A) and by the direct route combined with patch arterioplasty (B).
formed end-to-side to leave the course of the vertebral artery undisturbed. In my experience, bypass to the proximal vertebral artery has rarely been necessary. However, its use permits bypass to the vertebral artery within the cervical spine when the proximal segment is not suitable for use.

Decompression

When only one vertebral is patent or if there is marked sensitivity to intermittent dynamic occlusion, decompression may be the procedure of choice. The proximal vertebral artery is mobilized from its origin to the foramen transversarium at C6. If the vertebral nerve fibers overlying the vertebral artery are divided, a mild Horner’s syndrome will result but it usually disappears within a year. However, the vertebral nerve may be spared in cases where the artery is divided and primarily anastomosed. Radical excision of ganglia or fibers for microscopic confirmation of structure is not only unnecessary but worsens the sympathetic sequel. Bleeding from the vertebral vein near the vertebral foramen can be most troublesome. To assure long-lasting good results, all ligaments, muscles, and bands overlying the proximal vertebral artery are excised up to the foramen.

Segmental Resection

Segmental resection and end-to-end anastomosis may be applied in some cases of obstruction owing to entrapment. The artery should be of excess length and adequate diameter and the conditions for the performance of a convenient and unencumbered anastomosis should be present. If this is not the case, an alternative technique should be employed.

Subclavian-to-Proximal Vertebral Arterial Anastomosis

Obstruction of the proximal vertebral artery is often associated with an elongated and high-lying subclavian artery. Ligation and division of the vertebral artery near its origin permit moving the vessel anterolaterally to a more easily accessible surgical field (see Figure 30-9). If needed, shortening of the vertebral artery is accomplished by resection (see Figure 30-10). The origin of the thyrocervical trunk may be incorporated in the reconstruction, and used as the proximal site for the anastomosis.

Common Carotid-to-Proximal Vertebral Arterial Anastomosis

This procedure does not require the resection of the scalenus anticus muscle because the vertebral artery may be approached medial to the muscle. Introduced by Wylie in 1970, it has received enthusiastic support from Edwards (Edwards and Mulherin, 1980) and has gained in popularity over the years. The procedure also has the limitation that its use requires simultaneous occlusion of both the carotid and the vertebral arteries. Edwards and Mulherin (1980), though, reporting on 204 cases, did not observe any neurological sequelae due to this maneuver.

Inferior Thyroid-to-Vertebral Arterial Anastomosis

If there is a high-grade atherosclerotic obstruction of the proximal vertebral artery, the interior thyroid artery is usually well developed because it serves as a collateral channel. It can be anastomosed end-to-end to the proximal vertebral artery (Carney et al., 1981).
External Carotid-to-Midvertebral Arterial Anastomosis

Corkill and associates (1977) reported the anastomosis between the external carotid artery and the midvertebral artery for proximal disease. This technique is an alternative to distal vertebral artery bypass.

Superior Thyroid-to-Vertebral Anastomosis

Kojimi and associates (1983) reported microvascular anastomosis of the superior thyroid artery to the midvertebral artery in order to bypass atherosclerotic obstruction of the origin of the vertebral artery.

Ligation of the Proximal Vertebral Artery

Ligation of the vertebral artery may be required incidentally to procedures upon the subclavian artery or to control recurring hind-brain embolism (see Figure 30–9A). Confirmation of a patent contralateral vertebral artery is highly desirable. The use of heparin may decrease the likelihood of delayed thrombosis, and reconstruction at the skull base can be performed at the later date.

Replacement of the Subclavian Artery

Replacement of the subclavian artery with a prosthesis may be necessary if the performance of an anastomosis to it is part of the operative plan and the subclavian artery is of poor quality. Replacement of the vertebral artery with a Dacron prosthesis can be performed too, with patency persisting more than three years.

Contraindications

Contraindications particular to reconstruction of the proximal vertebral artery include (1) obstruction distal to the site of reconstruction, (2) opacification of the anterior spinal artery by a radiculomedullary branch arising from the subclavian artery (Carney et al., 1981), and (3) presence of a carotid-subclavian bypass. Ligation of the anterior spinal artery
should be avoided because it may result in paraplegia. The patient with borderline pulmonary function may be severely compromised by phrenic nerve injury or the loss of the scalenus muscle as an accessory muscle of respiration.

RECONSTRUCTION OF THE MIDVERTEBRAL ARTERY

Hutchinson and Yates (1957) first reported compression and obstruction of vertebral arteries by osteoarthritic spurs which resulted in death from stroke. Vascular reconstruction of the proximal artery was recommended by Crawford et al. (1958), whereas others pursued the issue by decompressing the vertebral artery within the cervical spine high at the skull base (Hardin, 1965).

Decompression

In the 1960s, Creighton Hardin, general and vascular surgeon, noted the filling of the basilar artery by right carotid injection in one case that sparked his interest in the vertebral system. In this case, the left vertebral artery was hypoplastic and the right vertebral artery was severely compressed by osteophytic spurs. The dominant right vertebral artery was decompressed on January 15, 1959, and the site was marked with silver clips. Postoperative films of the cervical spine revealed the clips at the incorrect vertebral level, and the larger spur was removed one week later. The bony mass reduced the arterial diameter by one half and was densely adherent to the arterial wall. Hardin in 1963 reported 15 additional cases using an incision paralleling the posterior edge of the sternocleidomastoid muscle. Bakay and Leslie (1965) also reported three similar cases and added interbody fusion of the vertebrae to the procedure. In Nagashima's (1970) experience with 20 additional patients, metal needle markers were used to localize the vertebral level by x-ray before injecting a small amount of dye to mark the tissue. He made a transverse cervical incision and carried dissection to the spine. The longus colli muscle was then resected and the osteophytes were excised with a dental burr, Hall air drill, or curette, but fusion was not performed. Head rotation during surgery resulted in near-total occlusion of the vertebral artery in seven of these patients and severe compression in the remaining 13. Verbiest (1969) combined decompression and mobilization of the vertebral artery during anterior decompression of nerve roots, removal of cervical disk, or fusion. He noted that in some cases scalenus anticus syndrome, if present, also cleared after the decompression of the nerve roots or the brachial plexus.

An argument against vertebral artery decompression is that the structural changes in the arterial wall are permanent. Gortvai (1964) noted that even after straightening the course of the artery, a crease remained in the adventitia where compression had been the most severe. He speculated that this crease may have represented either an atheromatous plaque or local fibrosis. Nagashima (1970), using the operating microscope, also noted that even after removal of bone and cartilage the vertebral artery still remained kinked because of a marked perivascular fibrosis.

The vertebral artery can erode bone and, in turn, osteophytes can erode the arterial wall. Such erosion by bony compression may necessitate ligation of the vertebral artery and may also be the source of thrombosis and embolism (Verbiest, 1969). The primary pathology of the midvertebral artery has been the compromise of vertebral artery blood flow with stressed head positions, but other neurological lesions, such as tumors of nervous origin and aneurysms, are also occasionally encountered. Aneurysms may be either small or extensive with venous communications. In the 1960s, the primary reason for utilizing decompression of the anterior aspect of the midvertebral artery was that it was the sole therapy a surgeon could offer. Two criticisms of the procedure included (1) the changes noted in the arterial wall extended through its entire thickness and could not be relieved by decompression, and (2) the arterial lumen was not explored and the state of the lumen not determined.

With the development of bypass procedures to the vertebral artery at the skull base, the need for arterial decompression within the cervical spine has diminished while decompression of cervical nerves impinged by bone remained a mainstay of neurological and orthopedic surgery.

RECONSTRUCTION OF THE DISTAL VERTEBRAL ARTERY

Exposure

An anterolateral or anterior approach may be used to reach the vertebral artery at the skull
base. The former is designed to expose the vertebral artery between the C1 and C2 segments by a route anterior to the internal jugular vein. The incision for the anterolateral approach, as described by Henry (1957) and also used by Verbiest (1969), can be extended first posteriorly toward the occiput, then medially and caudally. It permits wide access to the anterolateral structures of the neck.

The incision for the anterior approach (Nanson, 1966) mobilizes the earlobe and extends anterior to the ear. The dissection can then be extended to the base of the skull. This incision also provides good access to the parotid gland and facial nerve. Displacing the mandible forward improves access to the vertebral artery, the anterior cervical spine, and the high carotid. Both approaches work well, but the anterolateral one seems less cumbersome. In general, the less the experience of the operator, the wider the field that needs to be exposed. After the incision is made, the superior flap is carefully mobilized from the sternocleidomastoid muscle to avoid the deep lobe of the parotid gland and facial nerve. The greater auricular nerve is usually sacrificed and rarely reconstructed. The mastoid process is mobilized so that it can be divided with an osteotomy. Once the mastoid process is divided, the deep ligamentous attachment of the mastoid can be sectioned. This maneuver leads directly to the mastoid attachment of the posterior belly of the digastric muscle whose caudal edge is then mobilized to the hyoid before being elevated off the prevertebral fascia. The spinal accessory nerve lies posterior and lateral to the internal jugular vein. The superior cervical ganglion which sits astride the prevertebral fascia and medial to the jugular vein is not visualized unless it is fused with the anterior ramus of the second cervical nerve. The latter surrounds the vertebral artery and attaches it to the spine at the level of the atlantoaxial articulation. It is a sensory branch that can be divided with impunity. In a lean patient, aggressive superior traction may be followed by palsy of the mandibular branch of the facial nerve. Aberrant nerve fibers will often ensnare the vertebral artery, resulting in convolutions appreciated only at surgery.

In the course of the operation the transverse process of the atlas can be easily seen and palpated in the field. The caudal attachments include the levator scapulae, the scalenus medius, and the anterior intertransversalis muscles. Some of these muscle attachments must be divided. The atlantoaxial articulation can be palpated through the prevertebral fascia, which is then incised. The superior cervical ganglion lies medial to the anterior ramus of the second cervical nerve, which embraces the vertebral artery and holds it close to the vertebra. This ramus is divided and retracted. The venous plexus, if present, must also be controlled. The encircling sheath is incised, and the artery is mobilized to permit application of vascular clamps after exposing 1.5 cm of its length.

In some patients, the anterior capsule of the atlantoaxial articulation is worn thin by prominent flaring of the bony surfaces; in others, adhesions surround the artery. Entry into the capsule in such cases may reveal sharp edges of dense bone, which should be ground smooth. Occasionally, the bone overlying the foramen transversarium of C2 and C3 needs to be removed to gain access to the medial bight of the vertebral artery. Resection of the transverse process of C1 is well tolerated. Access to the artery high at C1 is much more readily secured than at the medial bight. Resection of extensive posttraumatic dystrophic calcification compromises joint stability.

TECHNICAL CONSIDERATIONS

When the lateral bight of the vertebral artery at C2 forms a free loop, mobilization can be accomplished with a minimum of dissection. The medial angle of the vertebral artery at the level of the axis, however, is more often the site of significant pathology. The bight of the vertebral artery may be buried deep in a bony cavern lined by veins or bony lakes. When the fibrous sheath extends to this area, it may be covered by Batson's venous lakes, and could cause severe blood loss if injured.

Bypass Grafts

The best substitute for artery is artery. The stress applied to a graft at the skull base is three-dimensional, and the position of the graft is important. The more posteriorly it is located, the less severe are the stresses involved. The more anterior is the position, the greater are the arcs cut by flexion and rotation. It is doubtful that the compound motion at the skull base would be tolerated by a synthetic prosthesis.

The vein is far from being a perfect graft material. Intimal hyperplasia can occlude a well-constructed vein graft. Thin-walled veins
tend to dilate excessively, tear easily, and are prone to kinking. Generally, thick-walled veins work best and are easiest to gauge for 4 to 6 mm vertebral arteries. Discrepancy in size is more of a problem if the arteries are hypoplastic. A smaller diameter arterial anastomosis with the use of a microvascular technique may be preferred in such situations. Many neurosurgeons have anastomosed the external carotid directly to the vertebral artery; even the superior thyroid artery has been used for this purpose.

Clark and Perry (1966) mobilized the vertebral artery from C6 to C1 to gain a long enough segment to anastomose the vertebral artery to the external carotid artery at the level of the bifurcation. That reconstruction remained patent for more than a decade (Perry, personal communication, 1985). Shorter lengths of the vertebral artery have been used by others (Carney et al., 1981; Diaz et al., 1983).

The use of artificial prostheses has been unrewarding. Occlusion often occurred with Dacron prostheses and with reconstructions originating from the external carotid artery.

PROCEDURES AT THE SKULL BASE
Decompression and Mobilization

An occasional patient may have severe local obstruction of the vertebral artery caused by compression by arterial branches or neighboring nerves. Division of the obstructive tissues and arterial mobilization may be curative. If there is prominent flaring and spurring of the C1-C2 joint surfaces, it is recommended that the articulation be ground smooth.

Carotid-to-Vertebral Arterial Anastomosis

In 1966, Clark and Perry mobilized the proximal vertebral artery to the skull base to obtain a segment sufficient to anastomose the vertebral to the external carotid artery at the level of the bifurcation (see Figure 30–11). When direct anastomosis is planned between these vessels, the vertebral artery can be passed deep to the internal jugular vein to shorten the distance to the carotid.

Segmental Resection

Acute angulation or constriction may compromise blood flow at a specific point. In such cases, mobilization alone may not be enough and segmental resection and end-to-end repair may be applicable. Redundancy of the vertebral artery may facilitate the performance of such an operation. The medial angle of the vertebral artery at C2 is mobilized and the segmental branches are ligated and divided. Unroofing the foramen transversarium at C3 usually permits the surgeon to obtain an ade-
quate length of the artery. Segmental resection allows restoration of flow without a graft, which is desirable in the younger patient. A three-year follow up with angiography has revealed good patency and excellent configuration of the anastomosis (see Figures 30–12 and 30–13).

Bypass to the Vertebral Artery at the Skull Base

When the proximal vertebral artery is patent and obstruction due to redundancy occurs at the C2 level, segmental resection is often adequate to restore normal flow. However, if vertebral artery obstruction exists proximal to C2 and cannot be corrected by reconstruction of the proximal segment, then reconstruction at the skull base by vein bypass is the procedure of choice (Carney et al., 1977).

Distal Anastomosis

End-to-End Anastomosis. Because this technique requires ligation and division of the proximal end of the vertebral artery, it is easier technically to perform the anastomosis if the distal end of the artery is delivered out of the depth of the wound and into the surgical field. Hemodynamically, this type of anastomosis is less likely to lend itself to the occlusion because it eliminates competitive flow and provides better laminar flow within the vertebral artery, which should diminish the chance of failure. It is the procedure of choice when the proximal vertebral artery is to be excluded, as is the case with arteriovenous malformations. Its disadvantage is that the vertebral blood flow is interrupted during the performance of both the distal and the proximal anastomoses (see Figure 30–14).

Side-to-Side Anastomosis. The proximal vertebral artery is preserved with this technique. Also, if the bypass occludes later on, the postoperative status is not worse than it was preoperatively. The major advantages to vein bypass are that only a short length of artery needs to be mobilized for reconstruction, the dissection of the medial angle is avoided, and vertebral blood flow is only briefly interrupted. Vascular clamps can be applied and anastomosis accomplished in a mobilized arterial segment not more than 1.5 cm long. Ideally,
Figure 30-13. Angiograms: A: Preoperative. B: Postoperative 42 months. The principal indication for surgery in this patient was recurrent syncope resulting from head hyperextension.

this anastomosis should not compromise the lumen of the vertebral artery or the objective of the operation, i.e., preservation of the "native" vertebral artery, is lost.

The Proximal Anastomosis

Common Carotid Artery. The bypass to the vertebral artery should originate from a source that is adequate and will not be compromised by the position of the head, and does not jeopardize future surgery. The common carotid-to-distal vertebral arterial bypass which originates well below the carotid bifurcation fulfills these criteria. In the case of very low-lying bifurcation, bypass from the subclavian artery or from the internal carotid artery should be considered.

Subclavian Artery. This artery is a good site of origin for a bypass, because with the distal anastomosis at the skull base, it tends to place the graft posteriorly in the neck where it is less vulnerable to rotation. Another practical advantage to this procedure is that the contribution of the bypass to brain perfusion can be measured readily by compressing the graft. However, its use requires a second incision. One must also be careful because the subclavian artery is positioned lower than the vertebral artery so that air can be trapped in the vein graft. Backflow will not occur if the venous valves have not been removed, and passage of a probe is very difficult.

Internal Carotid Artery. After endarterectomy of an occluded internal carotid artery, its proximal segment offers an excellent takeoff site for a vein graft. Similarly, if the internal carotid is to be ligated for some reason, it should be made high at the skull base so that the proximal segment provides a satisfactory conduit to the vertebral artery (see Figure 30-15).

External Carotid Artery. The external carotid artery bypass to the C1-C2 vertebral artery fared poorly, with occlusions occurring in four of the first six reconstructions (Carney and Anderson, 1978). This type of reconstruction exposes the anastomosis to tension when the head is rotated to the opposite side. The bypasses that have remained open have shown considerable slack, and have taken on a "lazy-S" configuration as they cross superficial to the internal jugular vein without producing tension. Direct anastomosis of the external carotid to the midvertebral artery has also been reported (Bladin and Merory, 1975; Corkill et al., 1977; Pritz et al., 1981; see Figures 30-16, 30-17, and 30-18).

Occipital Artery. The occipital artery is a small-diameter vessel which approaches the vertebral artery at a right angle. Size discrepancy may make flow competition great. Attempts to use the occipital artery are often abandoned because of friability of the vessel. Mechanical motion at the skull base could also compromise the quality of vessel wall. Anasto-
Figure 30-14. Mobilization of the entire vertebral artery to gain enough length to perform an end-to-end external carotid to proximal vertebral anastomosis at level of the carotid bifurcation. The principle of directly anastomosing the vertebral artery to the carotid artery involves the exclusion of the subclavian-vertebral artery inflow. When two large size vertebral arteries are present, this technique can be used (A). Cross section of a cervical vertebra on CT-scan. The shaded area of the cross-section of the bony vertebra indicates the limited amount of bone that must be removed to unroof and mobilize the vertebral artery (B). Segmental resection and anastomosis of the vertebral artery at the skull base (C).
jugular vein, but excess tension may compress the vein and also occlude the graft, whereas excess length can easily result in kinking. Grafts with a longitudinal axis fare much better than those with a horizontal axis. In one series, all bypasses from the occipital artery and all bypasses except two from the external carotid failed, irrespective of the level of the carotid bifurcation.

Neurological Deficit

There were no immediate operative deaths in this series. Of the two neurological deficits noted in the region supplied by the carotid artery, one was temporary and the other was permanent. The former patient had a tight stenosis of the carotid siphon and had a transient ischemic episode after surgery. The second patient had a low-lying carotid bifurcation and his vein bypass from the external carotid artery occluded asymptomatically in the postoperative period. Exploration failed to reveal any responsible mechanism for the occlusion. The situation was corrected by leaving the venous anastomosis intact, and placing a 4 mm Dacron prosthesis from the vein segment to the external carotid artery utilizing interrupted suture.

THE TRIGEMINAL ARTERY MODEL

The vein bypass to the distal vertebral artery has many similarities to the trigeminal artery, which connects the carotid to the basilar artery (see Figure 9–8). Complications may appear during the long-term follow-up of the bypass procedures. While the trigeminal artery is not subject to the stress of head motion and its wall is arterial not venous, the quality of the individual vein, its diameter, wall thickness and intima may affect long-term results.

Complications include embolism, thrombosis, and aneurysm (Carney, 1981; Kojimi et al., 1983; McCormick and Greene, 1967) which may be manifested by brainstem transient ischemic attacks, visual symptoms, and even cortical blindness. Clinicians should watch for these complications in patients undergoing bypass to the distal vertebral artery. The effects of alterations in perfusion pressure and volume flow on the arterial system and on neurological functions require further observation.
Figure 30-16. Different varieties of external carotid-to-distal vertebral artery bypass: superficial to the internal carotid artery (A), deep to the internal carotid artery (B), and occipital artery-to-distal vertebral artery bypass (C).

Figure 30-17. Schematic representation of the procedure used in a patient in whom vertebral flow was impeded by a combination of buckling and fibrous obstruction at the skull base (A). Surgical reconstruction consisted of releasing the fibrous compression and performing an external carotid-to-distal vertebral artery bypass using an autogenous saphenous vein graft (B).
Recovery

The patient usually feels better immediately after surgery. Within two to three weeks there is usually a relapse, but improvement is gradual thereafter. Being forewarned of this seems to benefit patients greatly. However, an occasional patient may require repeated hospitalization and administration of plasma volume expanders for relief. The use of a cervical pillow at night and a soft cervical collar is desirable in the early postoperative period.

In general, the time required to obtain relief of symptoms is directly related to their duration. Patients with symptoms of short duration obtain maximal improvement in about 12 weeks after surgery, which corresponds to the time when induration of the tissues of the neck subsides. If symptoms are longstanding, improvement continues more slowly over 24 months.

Although some patients will not experience anesthesia of the area supplied after division of the greater auricular nerve, most will retain some permanent numbness that is aggravated by cold weather. Patients should be informed of that in advance. Injury to the spinal accessory nerve will weaken shoulder stability, which requires six to 12 months to resolve.

THE INTRACRANIAL VERTEBRAL ARTERY

The history of ligation of the vertebral artery through the suboccipital approach goes back to 1888 (Matas, 1893). Before microvascular
surgery was developed, this was the only operative procedure utilized on the intracranial vertebral artery. The objective was, naturally, not revascularization, but rather control of bleeding, obliteration of an arteriovenous fistula, or exclusion of a cervical aneurysm. Waga and associates (1978) controlled a dissecting aneurysm that extended to the posterior inferior cerebellar artery by clipping the intracranial vertebral artery proximal to the aneurysm.

CONCLUSION

In summary, vertebral artery surgery is more complex than that in the carotid artery. Revascularization is now possible at all levels of the head and neck. I have performed a total of 115 reconstructions of the vertebral artery at the skull base with minimal morbidity and mortality rates and have attained good relief of symptoms and the disappearance of disability. Long-term patency appears to be adequate, but the technical considerations of providing a suitable conduit and supply of blood and the biomechanics of head motion must continue to receive careful clinical and experimental trials.

Finally, the carotid-vertebral communication constitutes an extracranial limb of the circle of Willis, a revolutionary concept. The bypass to the vertebral artery at skull base is not simple, and its construction requires meticulous technique and understanding of the biomechanics of head motion. The reality of this procedure has necessitated a diagnostic approach which has integrated diverse specialties, extended the focus from the cerebral hemispheres to the entire brain and extended the realm of technical possibility. The system approach to brain blood flow integrates brain perfusion with neurological function.

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EXTRACRANIAL CEREBROVASCULAR DISEASE

Diagnosis and Management

Edited by

Francis Robicsek, M.D.
Chairman, Department of Thoracic and Cardiovascular Surgery,
Charlotte Memorial Hospital and Medical Center,
Charlotte, North Carolina

and

Clinical Professor of Surgery
University of North Carolina at Chapel Hill,
Chapel Hill, North Carolina

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