

Syndromes of Vertebrobasilar Insufficiency and Their Possible Surgical Treatment

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Occlusive disease of the vertebrobasilar system may cause a variety of syndromes depending on the artery or arteries involved and the available collateral circulation. Based on the clinical picture and the angiographic findings, some of the patients may benefit from a gross reconstructive surgical procedure in the vertebral or carotid arteries. When these arteries are not correctable by gross surgical techniques, an occipital artery-posterior inferior cerebellar artery microanastomosis may also prove beneficial. An early clinical diagnosis of the brain-stem and occipital lobes ischemia and angiographic studies of the cerebral arteries are of prime importance and the key to the surgical management of these patients.

Vertebrobasilar insufficiency may cause a variety of clinical pictures depending on the site or sites and the degree of atherosclerotic occlusive disease of the vertebrobasilar system, and also the available collateral circulation.

The most common sites of occlusion in order of frequency are the origin of the vertebral artery from the subclavian artery; the distal vertebral artery at the level of the first cervical vertebra; the basilar artery; the origin of the posterior inferior cerebellar artery (PICA); and the origin of the posterior cerebral artery from the basilar artery (Figure 1). Occasionally, the subclavian artery proximal to the origin of the vertebral artery may also become occluded and cause a reversal of the

flow in the vertebral artery, thus sealing blood from the vertebrobasilar system and the syndrome of "subclavian steal" (Figure 2).

Although often the clinical picture of the vertebrobasilar insufficiency is that of the brain stem and occipital lobe's dysfunction, at times a specific syndrome may develop due to the occlusion of a specific artery.

Syndromes of Arterial Occlusion

Subclavian Artery

Occlusion of the subclavian artery proximal to the origin of the vertebral artery may produce vertebrobasilar insufficiency by diverting the blood from the posterior circulation into the ipsilateral arm. This condition, which was originally described by Toole in 1964, is known as the subclavian steal syndrome.¹ In this syndrome, the patient may experience lightheadedness, dizziness, blurred vision, double vision, numbness of the face, and loss of consciousness with exertion or when he is strenuously using the ipsilateral arm.

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Vertebral Artery

Complete occlusion of one vertebral artery usually causes no neurological abnormality unless the other vertebral artery is either significantly occluded or is congenitally hypoplastic (Figures 3-5).

Complete occlusion of both vertebral arteries, or complete occlusion of one vertebral artery and significant narrowing of the other vertebral artery with hypoplasia of either both posterior communicating or both proximal posterior cerebral arteries and no significant collateral circulation (Figures 3 and 4), may produce transient ischemic attacks (TIA), or a progressive stroke, or a completed stroke. The transient ischemic attacks are characterized by episodes of lightheadedness, dizziness, blurred vision, double vision, complete blindness, dysphagia, dysarthria, numbness of the face specifically around the mouth and lips, and numbness and weakness of the extremities. The progressive and completed strokes may cause multiple lower cranial nerve abnormality and a unilateral or bilateral numbness and weakness of the extremities.

Occlusion of the vertebral artery near the origin of the posterior inferior cerebellar artery may produce a clinical picture similar to that of the occlusion of the PICA except for a crossed hemiplegia which may also occur.

Occlusion of the small paramedian arteries (Figure 6) may cause a contralateral hemiplegia with loss of the postural sensibility and involvement of the ipsilateral cranial nerves at the level of lesion. An example is paralysis of one half of the tongue with a contralateral hemiplegia.

Occlusion of the PICA may present itself with vertigo, hiccough, and vomiting and then produce, on the same side of the lesion, facial paresthesia, loss of pain and temperature sensations, cerebellar ataxia, nystagmus, and Horner syndrome. On the opposite side of the lesion, it may cause loss of pain and temperature sensations over the limbs and trunk, and hemiplegia. Dysphagia and dysarthria may also occur.

Occlusion of the anterior spinal artery may result in a flaccid quadriplegia due to ischemia of the pyramidal tracts and their decussations, and loss of pain and temperature sensations below the lesion because of ischemia of the spinothalamic tracts.

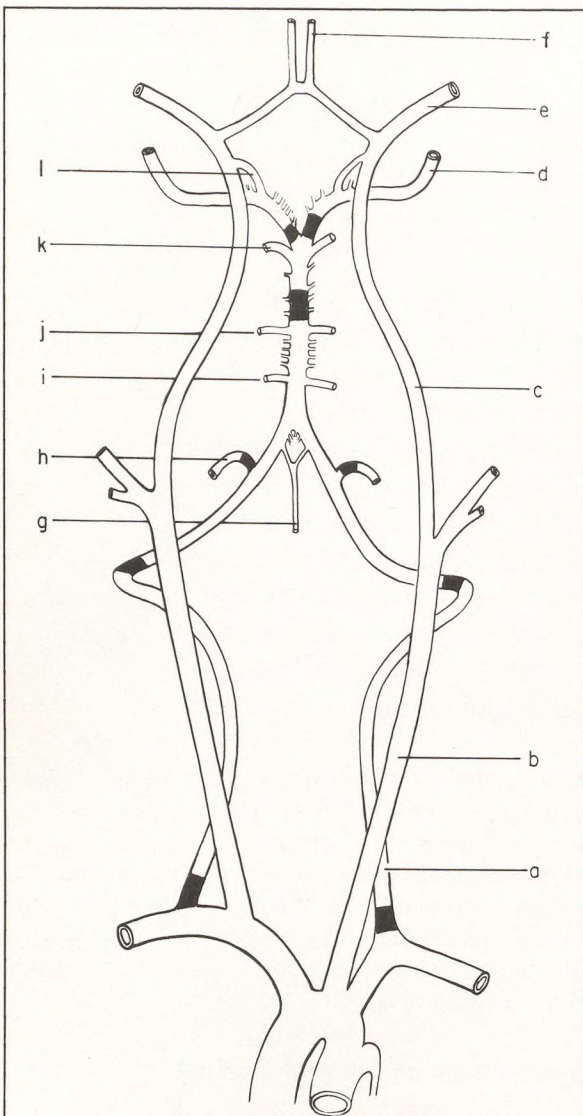


Figure 1. Arteries supplying the brain and the common sites of occlusive atherosclerotic disease in the vertebral-basilar system (solid black areas): (a) left vertebral artery, (b) common carotid artery, (c) internal carotid artery, (d) posterior cerebral artery, (e) middle cerebral artery, (f) anterior cerebral artery, (g) anterior spinal artery, (h) posterior inferior cerebellar artery, (i) anterior inferior cerebellar artery, (j) internal auditory artery, (k) superior cerebellar artery, (l) posterior communicating artery.

Basilar Artery

Complete occlusion of the basilar artery is not as common as the partial occlusion of this artery, but when it occurs it may produce a sudden onset of coma, pinpoint pupils, quadriplegia, and areflexia. The body temperature rises and the patient soon dies.

Partial occlusion of the basilar artery is more common than complete occlusion and is often seen in the distal portion of the basilar artery. It almost invariably causes vertigo. Visual field defects and drop attacks are also frequent manifestations. Double vision, paresthesia over the face especially around the mouth and lips, dysarthria, dysphagia, alternating or nonalternating hemiplegia, and unilateral or bilateral hypoesthesia are other symptoms and signs of the basilar artery insufficiency.

Small infarctions may produce rather discrete neurological abnormalities, for example, an ipsilateral sixth or seventh nerve palsy and a contralateral hemiplegia, or an ipsilateral paralysis of the conjugate gaze and a contralateral hemiplegia.

Akinetic mutism is another neurological manifestation which is thought to be due to a lesion in the central core of the upper brain stem in the region of the reticular formation. In this condition, the patient is completely conscious, but lies in bed speechless and motionless. The muscles are usually hypotonic and there is no reaction even to strong painful stimuli.

Anterior Inferior Cerebellar Artery

Occlusion of the anterior inferior cerebellar artery produces cerebellar ataxia, peripheral facial paresis, Horner syndrome, deafness and loss of pain and temperature sensations over the face, all on the same side of the lesion. On the opposite side there is loss of pain and temperature sensation over the extremities and the trunk.

Superior Cerebellar Artery

Occlusion of the superior cerebellar artery causes an ipsilateral cerebellar ataxia, involuntary choreiform movements and a Horner syndrome. On the opposite side of the lesion, it produces loss of pain and temperature sensations which include the face as well, thus differentiating this syndrome from that caused by occlusion of the PICA. A partial deafness and a central facial paresis may also be seen on the contralateral side.

Posterior Cerebral Artery

The perforating branches of the posterior cerebral artery near the junction of the posterior communicating artery supply the thalamus, subthalamic nucleus, red nucleus, the superior cerebellar peduncle, the lateral half of the cerebral peduncle, and the retrolenticular part of the internal capsule. The distal posterior cerebral artery supplies the occipital lobe (Figure 6).

Complete occlusion of the trunk of the posterior cerebral artery may cause hemiplegia, hemianes-

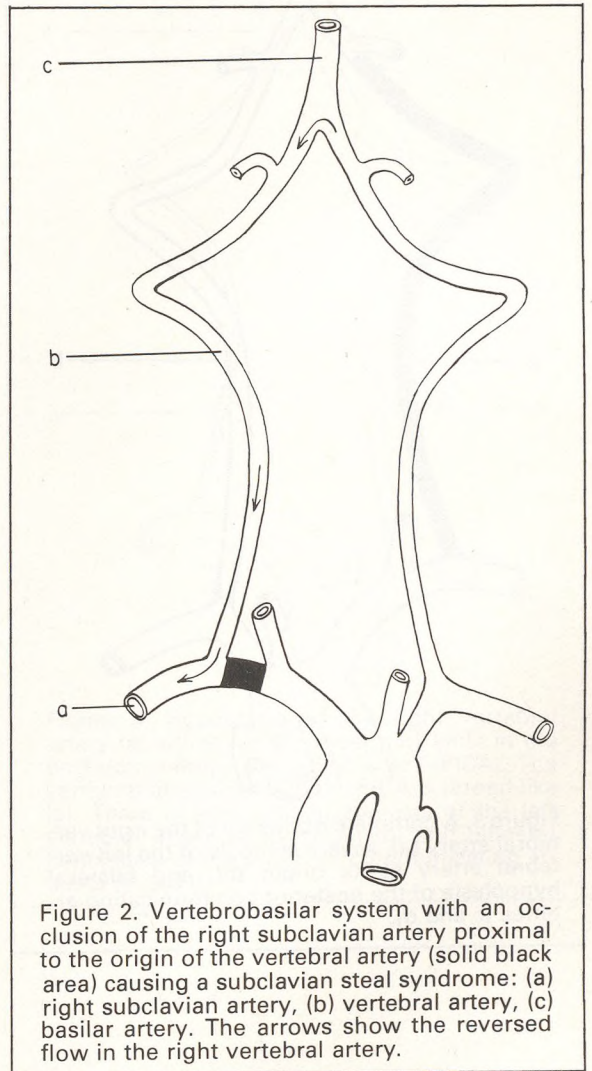
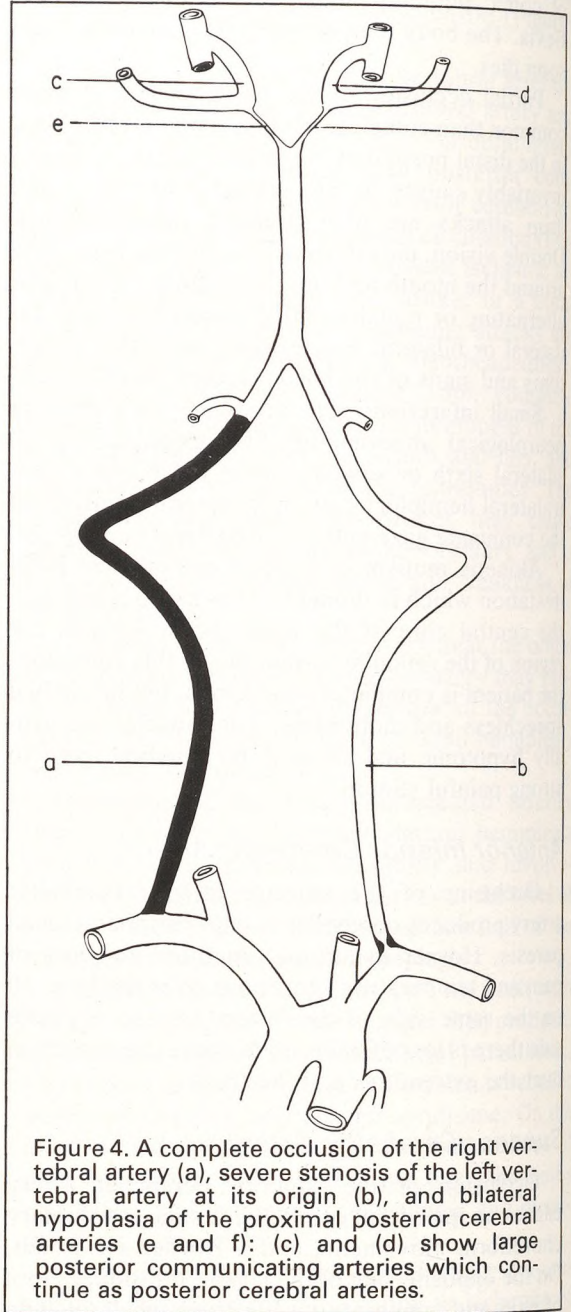
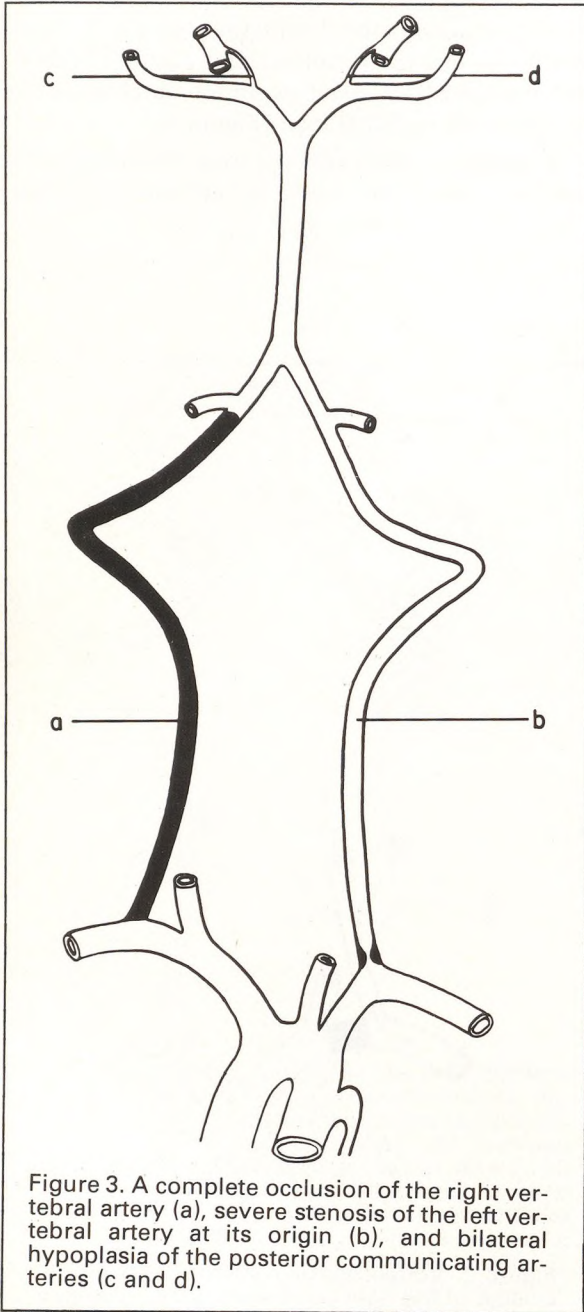


Figure 2. Vertebrobasilar system with an occlusion of the right subclavian artery proximal to the origin of the vertebral artery (solid black area) causing a subclavian steal syndrome: (a) right subclavian artery, (b) vertebral artery, (c) basilar artery. The arrows show the reversed flow in the right vertebral artery.



thesia, homonymous hemianopia, ataxia, abnormal movements, and thalamic syndrome.

Occlusion of the perforating branches may cause hemiballismas, thalamic syndrome, or syndrome of retrolenticular capsule, which is characterized by hemiplegia, hemianesthesia, and homonymous hemianopia.

Occlusion of the posterior cerebral artery beyond the origin of the perforators causes homonymous hemianopia which is usually without loss of macular vision.

Surgical Treatment

Although specific syndromes of the vertebrobasilar insufficiency may be seen, the surgical treatments at the present time are limited to the procedures that would increase the total blood flow to the brain stem and the occipital lobes. This augmentation of the flow can be established by two means: (1) gross vascular repairs of the vertebral or carotid arteries; and (2) a microvascular anastomosis between the occipital artery and a branch of the vertebrobasilar system.

The procedure of choice in the subclavian steal syndrome seems to be a bypass vein graft from the ipsilateral common carotid to the subclavian artery distal to the origin of the vertebral artery. The subclavian endarterectomies have also been tried, but the results have not been as good as the vein graft.

When the occlusive process involves the origin of the vertebral artery and is partial, a subclavian-vertebral artery bypass vein graft, an angioplasty vein patch, or a reconnection of the vertebral artery to the subclavian artery may be carried out. Some surgeons, in specific patients, have also suggested an external carotid-vertebral artery anastomosis or a foraminotomy of the foramen transversarium.^{2,3}

Occasionally, when the vertebral arteries are not repairable due to an extensive atherosclerotic disease yet there are good communications between the anterior and the posterior circulations and the carotid arteries are severely narrowed, a carotid endarterectomy may improve the vertebrobasilar circulation and relieve the ischemic syndrome. There are, however, some stroke patients whose atherosclerotic disease in the vertebrobasilar system is very severe and there is no significant

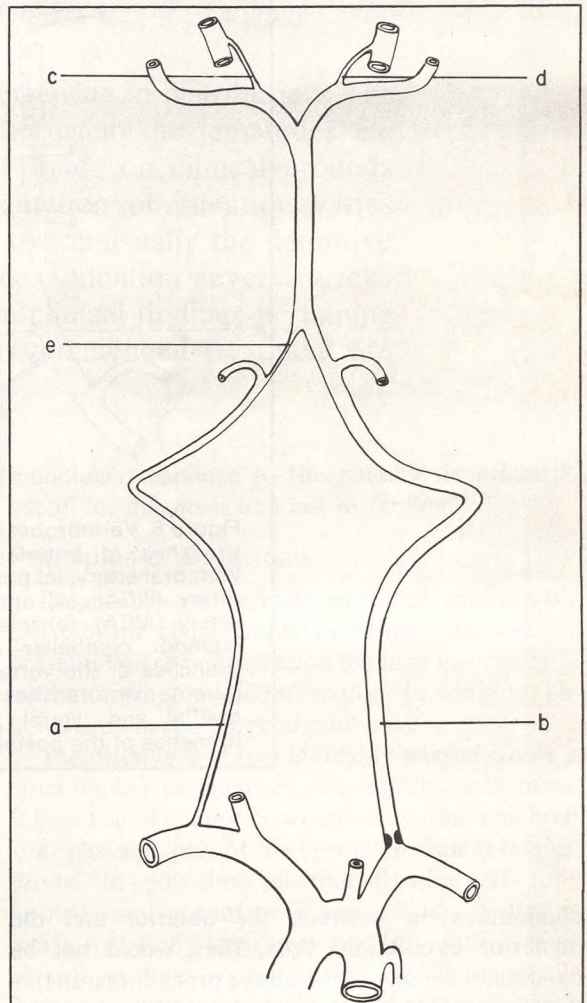


Figure 5. Hypoplasia of the right vertebral artery (a) which for the most part ends in the posterior inferior cerebellar artery (PICA). The vertebral artery distal to the PICA is thread-like (e). There is also marked stenosis of the left vertebral artery at its origin (b) and bilateral hypoplasia of the posterior communicating arteries (c and d).

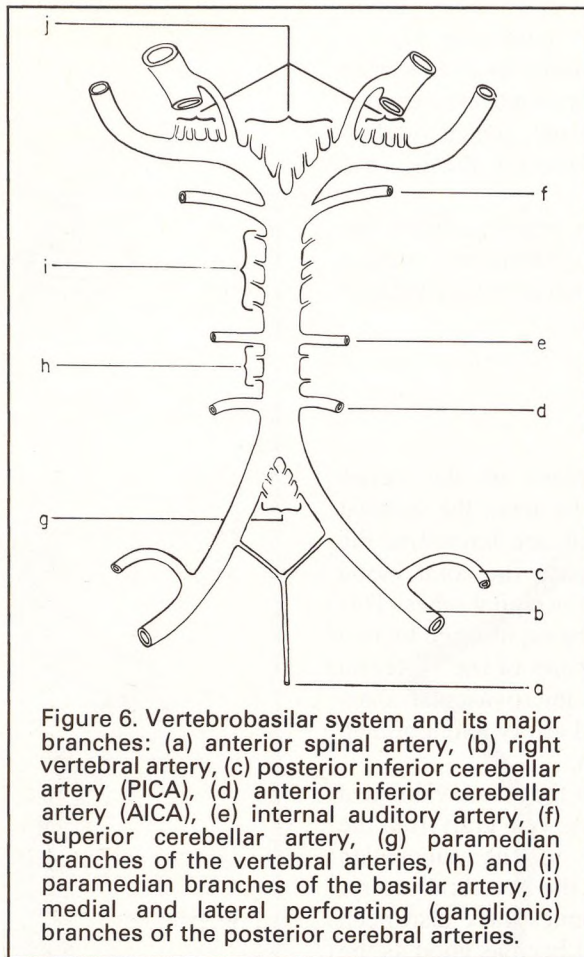


Figure 6. Vertebrobasilar system and its major branches: (a) anterior spinal artery, (b) right vertebral artery, (c) posterior inferior cerebellar artery (PICA), (d) anterior inferior cerebellar artery (AICA), (e) internal auditory artery, (f) superior cerebellar artery, (g) paramedian branches of the vertebral arteries, (h) and (i) paramedian branches of the basilar artery, (j) medial and lateral perforating (ganglionic) branches of the posterior cerebral arteries.

communication between the anterior and the posterior circulation; thus, they would not be candidates for any of the above procedures. In this group of patients, an occipital artery-posterior inferior cerebellar artery anastomosis may prove beneficial. This operation was originally applied in a patient in 1975 and subsequently in other candidates with encouraging results.^{4,5}

It seems that some of the patients who suffer from the brain stem and occipital lobe ischemia and have had TIAs or strokes may benefit from a gross or microvascular procedure in the vertebrobasilar system. The operations are designed to improve the posterior circulation and prevent the patients from having further strokes. In certain circumstances, the operations may also help the

acute neurological problems. The key to success, however, lies in an early clinical diagnosis of the brain stem and occipital lobe ischemia and the angiographic studies of the cerebral arteries.

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