Vertebrobasilar Disease

Time for a New Strategy

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Vertebrobasilar ischemia is a common disorder which is now frequently diagnosed by medical students, house officers, practitioners, and consultants alike. Although brainstem softening was recognized in the 19th century, the underlying pathology was not understood until the classic report of Kubik and Adams in 1946. These authors called attention to the clinical features in patients discovered at postmortem to have had an occlusion of the basilar artery and emphasized the abrupt onset and very poor prognosis of this condition. After Fisher reported that transient ischemic attacks frequently occurred in patients with occlusion of the internal carotid artery and, in fact, that they warned of an impending stroke, Denny-Brown, Sickert, Millikan, Fang, Williams, and others called attention to similar transient ischemic episodes related to disease of the posterior circulation. The term "vertebrobasilar insufficiency" was born.

Treatment for this newly described condition began in the 1950's with enthusiastic use of anticoagulant therapy as a result of a number of clinical trials, all of which were uncontrolled. Also, in all of these trials vertebrobasilar disease was considered as a single entity. Most studies revealed a small measure of benefit from anticoagulant treatment when large groups of patients were studied. Clinicians are still debating the indications for anticoagulant therapy in patients with vertebrobasilar ischemia.

Since the 1950's, a number of therapies have been used for posterior circulation ischemia. Surgically created extracranial-intracranial shunts (usually occipital to posterior inferior cerebellar artery) have been tried in a small number of patients; the indications for this procedure remain uncertain. Some investigators have advocated carotid endarterectomy for patients with vertebrobasilar ischemia and coexistent carotid stenosis; when formally studied, carotid surgery offered little or no benefit in a group of patients with this combination of findings. Antiplatelet aggregating agents, e.g., aspirin, sulfinpyrazone, and dipyridamole, are used in patients with occlusive vascular disease, but their effectiveness in posterior circulation disease has not been rigorously studied. Although there have been major improvements in the recognition and diagnosis of vertebrobasilar disease, there has been little progress in the past two decades in the treatment of this disorder.

At the same time major gains have been made in the treatment of carotid system disease during the last 25 years. These gains were possible because it was recognized that this entity is not a homogeneous syndrome. The vascular pathology occurs at different loci (for example, the internal carotid artery bifurcation, the middle cerebral artery, and the lenticulostriate arteries) and has varying morphology and severity (ulcerating plaques, severe stenosis, thrombotic occlusion). Widespread use of angiography and newer, noninvasive tests have aided in the recognition of the underlying vascular pathology so that treatment can be directed at the disordered pathophysiology in the individual patient. Examples include endarterectomy for tight stenosis at the carotid bifurcation, surgery or antiplatelet agents for plaque disease in an attempt to prevent embolization from these plaques, anticoagulation for nonoperable stenosis of the internal carotid artery at the carotid siphon or middle cerebral artery, or extracranial-intracranial (superficial temporal artery to middle cerebral artery) shunts for patients with diminished flow due to severe nonremediable occlusive disease. A small measure of the relative activity in this field is the fact that at the Fourth Joint Conference on Stroke and Cerebral Circulation in February, 1979, nine papers dealt directly with the diagnosis and treatment of carotid artery disease, while none specifically considered vertebrobasilar disease.

Can we apply what we have learned from our experience with carotid disease to the posterior circulation? Clinical experience indicates that vertebrobasilar disease is also a far from homogeneous syndrome (table). Some patients die after occlusion of the basilar artery, but others with recurrent vertebrobasilar insufficiency seem to do quite well. In fact, when large numbers of patients with vascular disease are considered, the group with posterior circulation disease generally has a better prognosis. Several subgroups of patients with vertebrobasilar disease also have a good prognosis. Would it not be wise to follow the trail of success provided by the carotid artery story?
and begin to separate the large entity of posterior circulation ischemia into clinically recognizable subdivisions which share a similar pathophysiology and prognosis? Appropriate treatment could then be considered for each subdivision.

### Subdivisions

**Basilar Branch Occlusion.** The penetrating and circumferential branches of the basilar artery may be occluded by atheromatous narrowing of the proximal portion of a basilar branch or by atheroma within the basilar artery occluding the lumen of the branch. This lesion is more common in patients with a diathesis for small vessel disease (hypertensive or diabetic patients). The resultant clinical syndrome is usually limited to a single penetrating vessel, e.g., a median-pontine branch, or to a single circumferential vessel, especially the anterior inferior cerebellar artery. More extensive basilar territory infarction generally does not occur in this group of patients, although they may be vulnerable to other small vessel lesions throughout the central nervous system. Treatment logically should consist of reduction of blood pressure after the acute ischemic period has passed and control of diabetes. The long-term use of anticoagulation or agents which affect platelet aggregation theoretically have little application in this group of patients.

**Lacunar Strokes.** Lacunes are small deep infarcts in the territory of penetrating vessels. The arteriopathy underlying this disorder has been studied by Fisher, who was able to document lipohyalinotic change with destruction of the vessel wall. This pathology is related to hypertension and is a lesion separate from atherosclerosis of larger vessels. Several lacunar syndromes of penetrating branch disease in the basilar system have been defined: dysarthria-clumsy hand syndrome (slurring of speech, facial weakness, and slight clumsiness of one hand) and ataxic hemiparesis (incoordination of an arm and/or leg with increased reflexes and weakness of the same limb). These lesions are also self-limited. Treatment is similar to that of basilar branch disease, i.e., control of hypertension.

**Basilar Occlusion.** Occlusion of the basilar artery is generally accompanied by bilateral long tract signs and segmental ischemia. The prognosis is usually poor, but some patients with complete occlusion of the basilar artery survive with little permanent loss of function. The clinical deficit often accumulates within the first few weeks after occlusion, and late increases in deficit are less likely (a pattern similar to experience with carotid artery occlusion). During the first few weeks after occlusion, extension of clot, embolization from the clot, and establishment of collateral circulation may occur. As with internal carotid artery occlusion, treatment should be vigorous in the initial period after occlusion (e.g., head-down position, maintenance or slight elevation of blood pressure, and short-term anticoagulation with heparin). Long-term anticoagulation would theoretically seem to have less rationale but has not been systematically studied in this group of patients.

**Basilar Artery Stenosis.** Clinical signs are identical to those of basilar occlusion. However, as with carotid stenosis, serious brainstem and cerebellar infarction may occur when occlusion becomes complete. Also, as with carotid stenosis, antiplaquelet agents, long-term anticoagulation, and shunting procedures (perhaps to branches at or rostral to the anterior inferior cerebellar artery level) are all hypothetically useful. The therapeutic strategies for this condition need to be studied in a rigorously controlled fashion.

**"Top of the Basilar" Ischemia.** A subgroup of patients with posterior circulation disease have deficits referable to the high brainstem and posterior cerebral artery territories. In our experience and as stated in the literature, the deficit usually develops abruptly. Embolic occlusion is found within the distal basilar artery or its tributaries at postmortem. Since the basilar artery is widest at its origin, at the junction of the vertebral arteries, and tapers distally, emboli small enough to traverse the vertebral artery will not usually be trapped until they reach smaller distal basilar branches. These findings support the idea that rostral basilar disease is frequently embolic. More angiographic confirmation is required however.

### Table: Representative Clinical Patterns

<table>
<thead>
<tr>
<th>Anatomical site:</th>
<th>Left medial pontine infarction</th>
<th>Left lateral pontine infarction</th>
<th>Left cerebellar infarction</th>
<th>Occipital lobe and thalamic infarction</th>
<th>Left pontine lacunar infarction</th>
<th>Right and left pontine infarction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vessel affected:</td>
<td>Left basilar branch occlusion</td>
<td>Left vertebral occlusion</td>
<td>Left vertebral occlusion</td>
<td>Top of basilar embolus</td>
<td>Left penetrating vessel occlusion</td>
<td>Basilar occlusion</td>
</tr>
<tr>
<td>Deficit:</td>
<td>1. Diplopia</td>
<td>1. Decreased temperature</td>
<td>1. Inability to walk</td>
<td>1. Cortical blindness</td>
<td>1. Right arm, leg ataxia</td>
<td>1. Quadriparesis</td>
</tr>
<tr>
<td></td>
<td>2. Left Vth nerve palsy</td>
<td>2. Decreased pain and temperature</td>
<td>2. Vomiting</td>
<td>2. Small poorly reactive pupils</td>
<td>2. Increased extensor plantar reflexes</td>
<td>2. Extensor plantar reflexes</td>
</tr>
<tr>
<td></td>
<td>3. Decreased position sense</td>
<td>3. Decreased right face</td>
<td>3. Ataxia of left limbs</td>
<td>3. One or both eyes deviated down and in</td>
<td>3. Right extensor plantar reflex</td>
<td>3. Dysarthria</td>
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<td></td>
<td>5. Left Horner's syndrome</td>
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## References

In embolic disease a careful search for the origin of the embolus (intra-arterial from a vertebral or proximal basilar plaque or from the heart) is warranted. Depending on the source of embolus, treatment strategies would be similar to those used in embolic disease of the anterior circulation and would include antiplatelet aggregating agents, or Warfarin, or a surgical attack on the source of the embolus.

**Vertebral Artery Disease in the Neck.** Hutchinson and Yates called attention to the frequency with which the nuchal portion of the vertebral artery is the site of atheroma. Atheroma might serve as the nidus for platelet and thrombotic material which embolizes distally. Postmortem studies of patients with vertebrobasilar system occlusion have documented the frequent presence of small distal emboli. In a number of patients with major vertebrobasilar attacks, angiography has demonstrated widely patent vessels with only irregular narrowing of the proximal vertebral artery. Do ulcerated plaques in the vertebral artery have the same angiographic appearance as in the carotid artery, or does the smallness and tortuosity of the vertebral artery alter their appearance? Do antiplatelet antagonists have something to offer in this group of patients? Fisher described a number of patients with unilateral or bilateral occlusion of the vertebral artery in the neck. These patients frequently have remarkable collateral circulation, and although they may have TIA's, they seldom have major brainstem infarction. Patients with subclavian steal may have strokes, but these are usually infarctions that would warrant therapeutic trials of Warfarin over a longer period of time.

**Intracranial Vertebral Occlusion.** In this subdivision several different syndromes may occur:

1. **Lateral medullary infarction:** Postmortem study of patients with posterior inferior cerebellar artery (PICA) territory ischemia has usually demonstrated occlusion of the intracranial vertebral artery, causing a decrease in flow in the PICA, the major vertebral branch. The lateral medullary syndrome is a relatively benign condition. Most patients survive and usually do not develop subsequent posterior circulation strokes. A small number of patients succumb because of associated massive cerebellar infarction with herniation or because of ischemia at more rostral levels of the basilar artery due to emboli from the vertebral artery at the time of occlusion, or associated stenosis or smallness of the contralateral vertebral artery, or extension of clot into the basilar artery. The latter group of patients have signs of dysfunction outside the lateral medullary area early in their course and are thus potentially separable from those with a "pure" lateral medullary syndrome.

2. **Cerebellar infarction:** Large cerebellar infarction may not be associated with clinical evidence of brainstem infarction and can cause death by producing pressure on adjacent fourth ventricle and posterior fossa structures. This syndrome is treated by agents which reduce cerebral swelling (steroids, mannitol, glycerol) or surgical decompression. Small infarcts are difficult to recognize but produce a clinical picture quite similar to "labyrinthitis" with dizziness and veering to the side as the principal features. Occlusion of the vertebral artery is a common cause of cerebellar infarction.

3. **Hemi-infarction of the medulla:** After a prodromal period of headache and episodic dizziness, the patient may develop hemiparesis accompanied by vertigo, vomiting, tinnitus, nystagmus, unilateral hearing loss, ataxia, or dysphagia. The findings can be explained by ischemia of the medial medulla (XII nucleus, medial lemniscus, and pyramid) and lateral medulla (nucleus ambiguous, spinal tract and nucleus of V, inferior cerebellar peduncle, vestibular nuclei, and spinothalamic tract).

In most patients with occlusion of the vertebral artery, as in patients with internal carotid artery occlusion, the major clinical syndrome evolves over a short period of days to a few weeks. During this time collateral circulation is developing, and clot extension and embolization may occur. Late occurrence of deficit is less common. Perhaps a trial of bed rest to allow development of collateral flow, surveillance for large cerebellar infarction, and a short-term trial of heparin-like drugs would be worth consideration in this group of patients. Unilateral intracranial vertebral artery stenosis can be a more unstable situation that would warrant therapeutic trials of Warfarin over a longer period of time.

**Bilateral Vertebral Occlusive Disease.** Severe stenosis, hypoplasia, or occlusion of both intracranial vertebral arteries are included in this subdivision. In our experience bilateral decrease in flow at the level of the intracranial vertebral arteries carries a prolonged hazard of serious, often fatal brainstem or cerebellar infarction. In this condition there is much less possibility for collateral circulation than in patients with basilar occlusion (in which the long circumferential cerebellar arteries are occasionally adequate). Creation of a surgical shunt (e.g., occipital-posterior inferior cerebellar artery) seems worthy of study in these patients who may be at high risk.

**Patients with “Normal” Angiograms Despite Symptoms Not Explainable by Branch Disease.** In this group of patients, symptoms are clearly of vascular origin and referable to the posterior circulation, and signs which are bilateral or referable to the territories of both medial and lateral penetrating vessels argue against single branch disease. Angiography is normal or reveals only minor irregularity of vessels. The parallel situation in the anterior circulation of TIA's or strokes without angiographic explanation is well known. In cases considered in this group, the epidemiology (lack of high blood pressure or diabetes) and the clinical anatomy frequently do not suggest a lacune or basilar branch occlusion, either of which could produce a normal angiogram. The explanation for this phenomenon is not clear. Functional vascular
spasm (e.g., migraine), artery to artery emboli which pass rapidly, or hemodynamic disturbances related to neck and head posture have all been suggested but remain unproven. Bilateral or extensive branch disease is a possibility which has been documented in some cases. This group is probably not homogeneous but could be considered as a separate subdivision for treatment trials.

Discussion

The classification of patients with posterior circulation disease into subdivisions frequently requires a combination of clinical observation and vertebral angiography. Since neither angiography nor anticoagulant therapy should be undertaken lightly, it seems wise to limit their use to patients with clear-cut or highly probable ischemic disease of the posterior circulation. Recurrent dizziness, especially if unaccompanied by other symptoms, and lasting more than six weeks is rarely caused by serious vertebrobasilar disease. Similarly, attacks of sudden postural loss (drop spells), especially when unaccompanied by other symptoms of posterior circulation ischemia, are in my experience not commonly due to vertebrobasilar disease. It may be possible in some cases of typical stroke syndromes, such as with the clinical picture of a lacune or basilar branch occlusion, to be sufficiently certain of the diagnosis to omit angiography. In these subgroups surgical or anticoagulant therapy would not be used. In other patients the severity of the neurological deficit or of associated medical disease might contraindicate aggressive therapy like surgery or anticoagulants and so obviate the need for angiography. It seems reasonable to manage this group of patients with a period of bed rest (one to two weeks) and ASA (5 grains t.i.d.) and dipyridamole (75 mg q.i.d.). In the remainder of patients in whom the pathophysiology is unclear and treatment is not contraindicated, angiography would certainly be needed to substantiate the abnormal vascular anatomy. Contrary to expectations, there is a smaller percentage of complications with angiography of the posterior circulation than in studies of the anterior circulation. If angiography confirms a vertebral or basilar occlusion, bed rest and short-term anticoagulation might be tried. In patients with severe stenosis of an intracranial vertebral or basilar artery, especially if the clinical deficit is not severe, Warfarin might be used. In patients with severe bilateral vertebral stenosis or with basilar stenosis with persistent symptoms despite anticoagulation, surgical shunts may help. In patients with minor irregularity of the extracranial or intracranial vessels or normal angiography, a trial of aspirin and dipyridamole might suffice.

I am hopeful that the next few years will witness new vigor in the treatment of vertebrobasilar disease. In my opinion this change will occur when clinicians begin to define the pathophysiology of the TIA or stroke in the individual patient with vertebrobasilar disease. Controlled clinical trials of treatment can then be undertaken in relatively homogeneous subgroups which share a common prognosis and pathophysiology. The lumping of all patients with vertebrobasilar disease into one category is impractical and no longer makes sense.

References

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