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## Case Reports

# Regional Cerebral Blood Flow During an Attack of Vertebrobasilar Insufficiency

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Using technetium-99m-labeled hexamethylpropyleneamineoxime ( $^{99m}\text{Tc}$ ]HM-PAO) and single-photon emission computed tomography, we measured changes in regional cerebral blood flow in a 58-year-old man during an attack of vertebrobasilar insufficiency. Angiography demonstrated compression of the left vertebral artery by the osteophytes of cervical spondylosis when the patient turned his head to the left. Measured in the orthostatic position while turning his head to the left during a typical attack of vertebrobasilar insufficiency, regional cerebral blood flow was significantly reduced in the left cerebellum and the right occipital region. Our study illustrates the capability of  $^{99m}\text{Tc}$ ]HM-PAO single-photon emission computed tomography to measure transient reductions in regional cerebral blood flow and to relate these changes to the pathophysiology of vertebrobasilar insufficiency. (*Stroke* 1988;19:1426-1430)

Whereas transient ischemic attacks (TIAs) in the carotid artery system are believed to be caused predominantly by fibrinoplatelet emboli from ulcerated plaques at the origin of the internal carotid artery, the pathophysiologic mechanisms producing TIAs in the vertebrobasilar artery system appear to be more complex.<sup>1-6</sup> In 1960, Sheehan et al<sup>7</sup> postulated the syndrome of spondylotic vertebral artery compression and documented arteriographically that TIAs due to vertebrobasilar insufficiency (VBI) could be caused by compression of the vertebral artery in the neck by the osteophytes of cervical spondylosis. Subsequent reports by neurosurgeons described the benefits of surgery (decompression of the vertebral arteries) for treating the symptoms of VBI and for preventing permanent occlusive diseases.<sup>8-10</sup>

The posterior cerebral circulation, however, has anatomic characteristics that enable multiple collateral channels to preserve cerebral blood flow. Reduced perfusion pressure due to occlusion of one vertebral artery is usually compensated for by collateral circulation via the opposite vertebral artery and/or the circle of Willis. Stenosis of one vertebral artery alone (demonstrated angiographically) does not necessarily reduce blood flow sufficiently to cause clinical signs and symptoms. For technical

reasons, it has been difficult to directly measure blood flow in the posterior cerebral circulation during attacks of VBI to document regional cerebral blood flow (rCBF) dynamics.

We report a case of VBI due to spondylotic compression of the vertebral artery (confirmed angiographically) during turning of the head. The resulting reductions of blood flow in the posterior cerebral circulation during the symptoms of VBI that followed were documented using single-photon emission computed tomography (SPECT) and intravenous technetium-99m-labeled hexamethylpropyleneamineoxime ( $^{99m}\text{Tc}$ ]HM-PAO).

### Case Report

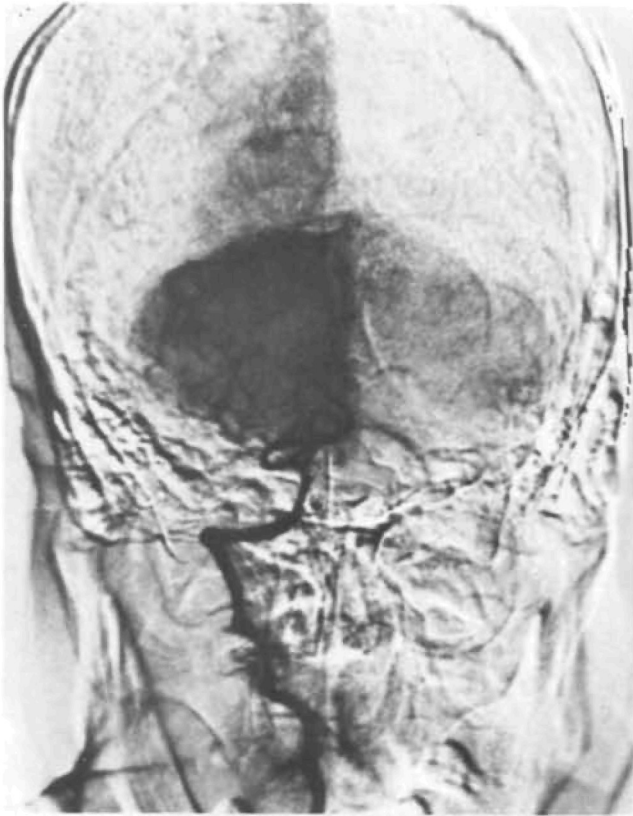
Over 2 months, a 58-year-old man experienced sudden and transient attacks of blurred vision when he turned his head abruptly to the left. Each attack was often accompanied by dizziness, a fainting

FIGURE 1. Vertebral angiography in 58-year-old man with symptoms of vertebrobasilar insufficiency with head in anteroposterior position (top left, top right, bottom left) and turned to left (bottom right). Right vertebral artery is congenitally small and perfuses only right cerebellum (top left). Left vertebral artery is large and dilated and appears elongated and tortuous with angulation and is displaced by osteophytic spur at C3-C4 level (bottom left). Left vertebral artery perfuses both cerebellar hemispheres and right occipital lobe (top right). When head is turned to left, severe stenosis of left vertebral artery due to osteophytic compression is seen at C3-C4 level (bottom right). No stenosis was seen in right vertebral artery.

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sensation, and weakness of both legs. These symptoms appeared only when the patient was standing and could be prevented if he turned his head slowly. Attacks varied in severity but always cleared rapidly once the patient returned his head to the neutral position. In the previous 2 years, he had complained of occasional dull and pulsatile pain in the occipital and nuchal areas associated with a feeling of stiffness in his shoulder. He gave no history of hypertension or diabetes.

On examination, his blood pressure was 134/82 mm Hg and his pulse rate was 64/min and regular. There were neither significant orthostatic hypotension nor bruit in his neck or subclavian regions. Neurologic examination was negative except for a questionable left eyelid ptosis, but forced turning of his head to the left caused dizziness and blurring of his vision. Cervical spine x-ray disclosed lateral osteoarthritic spurring most markedly between C3 and C4. Results from x-ray computed tomography and magnetic resonance imaging were negative.

An aortogram showed normal branching of the carotid and vertebral arteries from the aortic arch. Bilateral vertebral angiography was performed with his head placed in an anteroposterior position and with his head turned to the left (Figure 1). The right vertebral artery was narrow and elongated and predominantly perfused the right cerebellum (Figure 1, top left). The left vertebral artery was dilated, elongated, and tortuous, with some angulation due to the cervical spurs (Figure 1, bottom left); it perfused both cerebellar hemispheres and the right occipital region (Figure 1, top right). Although carotid angiography was not performed, we assumed from these findings that the left occipital lobe was perfused by collateral vessels from the circle of Willis. When the patient's head was turned to the left, severe stenosis of the left vertebral artery was seen at the C3–C4 level (Figure 1, bottom right). No stenosis was seen in the right vertebral artery.

We measured rCBF twice using rotating gamma cameras (GE Starcam AC/T, Milwaukee, Wisconsin) after the intravenous injection of 20 mCi of [ $^{99m}\text{Tc}$ ]HM-PAO (Amersham Pharmaceuticals Co., Ltd., Japan). In the first study [ $^{99m}\text{Tc}$ ]HM-PAO was injected into the antecubital vein with the patient standing with his head turned as far as possible to the left; he was asked to maintain this posture for 2 minutes. The patient was then placed in a supine position with his neck straight and relaxed, and the scanning was started 10 minutes after the injection. The patient reported that he experienced his usual TIA symptoms of blurred vision and dizziness when he turned his head and while [ $^{99m}\text{Tc}$ ]HM-PAO was injected. In the second study 6 days later, [ $^{99m}\text{Tc}$ ]HM-PAO was injected intravenously with the patient standing but without turning his head. Blood pressure and pulse rate were not significantly different with his head in the neutral position or turned to the left.

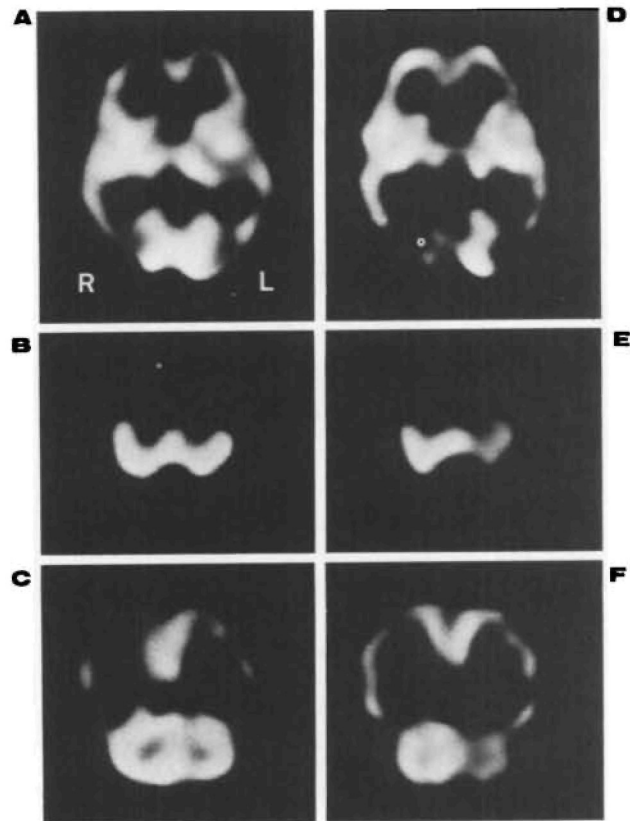


FIGURE 2. Regional cerebral blood flow (rCBF) in 58-year-old man with symptoms of vertebrobasilar insufficiency measured after intravenous injection of 20 mCi of technetium-99m-labeled hexamethylpropyleneamine-oxime. With head in neutral position, rCBF was uniform without hemispheric asymmetry in all brain regions examined (A, B, C). When head was turned to left, rCBF was reduced in right occipital region (D), opposite the vertebral artery compression, and rCBF was reduced in left cerebellar regions (E, F). Asymmetry of cerebellar hemispheric blood flow was most marked in coronal slice perpendicular to orbito-meatal line. At this level, mainly posterior portions of both cerebellar hemispheres are imaged (F).

With the patient's head in the neutral position, rCBF showed no hemispheric asymmetries throughout the brain (Figure 2, A–C). When he turned his head to the left, however, rCBF was markedly reduced in the right occipital region (Figure 2D), opposite the side of the vertebral artery compression, and in the left cerebellar hemisphere (Figure 2, E and F). Asymmetry of cerebellar blood flow was most marked in the coronal slice perpendicular to the orbito-meatal line, which predominantly includes the posterior lobes of the cerebellar hemisphere (Figure 2F). Isotope uptake in the right occipital lobe and the left cerebellum were reduced by 27.5% and 16.7%, respectively, compared with homologous regions of the opposite hemisphere. The range of asymmetries for rCBF in the occipital lobe and the cerebellar hemispheres in eight neurologically normal subjects was  $3.2 \pm 2.6\%$  in our laboratory;

the rCBF asymmetries in this patient were considered to be abnormal.

### Discussion

In clinical practice the diagnosis of VBI is sometimes difficult. It has been argued that TIAs within the vertebrobasilar artery system are caused by various hemodynamic factors such as postural and other causes of systemic hypotension, transient inadequacy of the cerebral collateral circulation, compression of the vertebral arteries by cervical spondylotic osteophytes, and hemorheologic factors such as polycythemia.<sup>2,5,8-10</sup> However, many clinical symptoms that are characteristic for VBI, such as dizziness, ataxia, diplopia, blurred vision, syncope, etc., are not unusual complaints among the elderly and are often difficult to differentiate from those due to nonvascular causes. Additionally, the symptoms of VBI are usually more transient than those of carotid TIAs and seldom produce neurologic deficits lasting long enough to be evaluated by a physician. Furthermore, the symptoms of VBI are often precipitated only by certain conditions such as assuming the upright posture, turning the head and neck, or micturition.

There are only a few reports of rCBF measurements in patients with VBI.<sup>11</sup> To be clinically relevant, measurements of rCBF in VBI should 1) be three-dimensional, 2) possess adequate spatial and temporal resolution, 3) be possible during physiologic conditions that precipitate attacks of VBI, 4) be noninvasive, 5) use clinically accessible instrumentation, and 6) allow serial measurement.

Our study indicates that measurement of rCBF using [<sup>99m</sup>Tc]HM-PAO and SPECT provides a useful tool for evaluating the cerebral hemodynamics during attacks of VBI precipitated by head turning. [<sup>99m</sup>Tc]HM-PAO is a newly developed tracer isotope designed to be used for the routine three-dimensional imaging of rCBF using SPECT instrumentation.<sup>12-15</sup> [<sup>99m</sup>Tc]HM-PAO has a high extraction fraction on first pass and is deposited in the brain in amounts proportional to rCBF. Isotope activity extracted by the brain plateaus within 2 minutes after intravenous injection; thereafter, regional distribution of [<sup>99m</sup>Tc]HM-PAO is constant without redistribution over 2 hours. Recirculation effects are minimal because the majority of activity in the blood is tightly bound to erythrocytes and plasma proteins after first-pass delivery to the brain. Although a method for calculating absolute blood flow has yet to be developed, a unique feature of [<sup>99m</sup>Tc]HM-PAO is that it may be used to monitor rapid changes in rCBF. The tracer can be injected intravenously through an indwelling catheter in any posture and during any physical activity. Thereafter, data can be acquired using SPECT after the patient is supine and in a comfortable position.

Our results demonstrate that the [<sup>99m</sup>Tc]HM-PAO SPECT method detects transient cerebral ischemia of short duration. In our patient, rCBF was reduced

in the hindbrain during a transient attack of VBI provoked by turning his head while standing. The resulting reductions in rCBF were striking compared with the normal rCBF pattern during a control study with the patient's head in the neutral position when he was without symptoms of VBI. We correlated these rCBF reductions during the provoked symptoms of VBI with angiographic findings obtained before and after the patient turned his head. Angiography showed a large, dilated left vertebral artery that predominantly supplied blood to the posterior circulation (both cerebellar hemispheres as well as the right occipital cortex), while the congenitally small right vertebral artery supplied blood to only part of the right cerebellar hemisphere (Figure 1, top left and top right). The left occipital cortex was assumed to be supplied by the left internal carotid artery, which gave rise to the left posterior cerebral artery since the basilar artery did not supply this vessel. These arteriographic findings correlated well with our observations that rCBF was reduced in the left cerebellar hemisphere and the right occipital cortex during compression of the left vertebral artery by turning of the patient's head. The fact that rCBF in the left occipital lobe was not reduced when he turned his head confirms our angiographic observation that this area was most likely perfused by the left posterior cerebral artery, which originated from the left internal carotid artery.

Regional ischemia demonstrated by rCBF measurements explains our patient's clinical symptoms of VBI, which consisted of transient blurred vision after he turned his head, with dizziness, ataxia, and unsteadiness of posture. Review of the literature suggests that our study is the first to demonstrate the regional hemodynamics of the posterior cerebral circulation, which account for the symptoms of VBI. Although the cause of VBI in this patient was unusually specific and demonstrable (compression of the vertebral artery in his neck), our approach is applicable to future investigations of the pathophysiology of other types of transient attacks of VBI.

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KEY WORDS • cerebral blood flow • tomography, emission computed • vertebrobasilar insufficiency