

## Basilar Artery Occlusion after Superficial Temporal Artery-Superior Cerebellar Artery Anastomosis

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Superficial temporal artery-superior cerebellar artery anastomosis for basilar artery stenosis was performed in a patient who had experienced recurrent nonhemispheric symptoms, and whose angiogram revealed a midbasilar stenosis. Four months after the operation he was readmitted to our hospital complaining of transient motor weakness and numbness of the left extremities. No neurological deficit was found on admission, but 1 hour later the left-sided hemiparesis occurred again and progressed. On the angiograms the upper basilar system was visualized via the superficial temporal artery, and the midbasilar artery was occluded. This conversion seemed to be a natural course rather than the influence of the hemodynamic change after the operation.

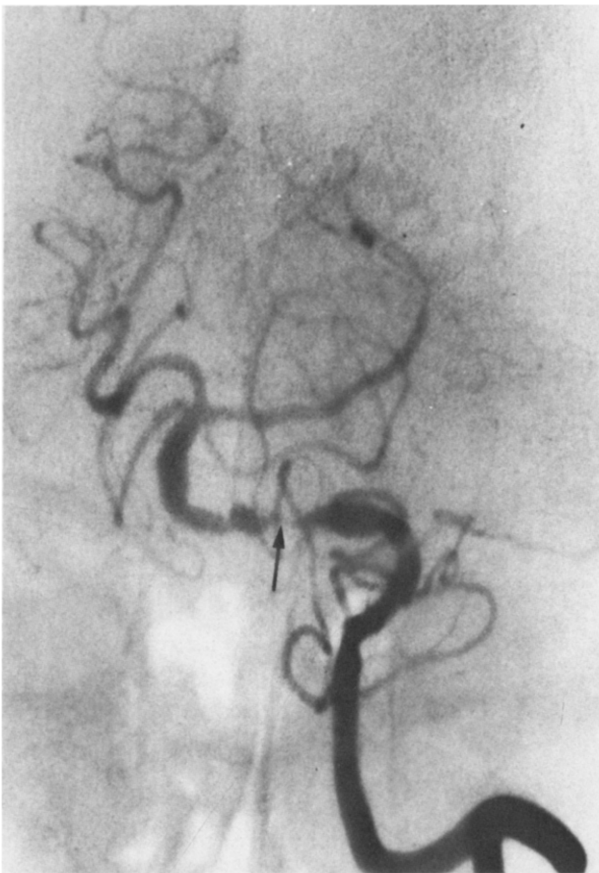
**KEY WORDS:** Anastomosis; Basilar artery; Occlusion; Stenosis; Superior cerebellar artery; Superficial temporal artery

In recent years superficial temporal artery-superior cerebellar artery (STA-SCA) anastomosis for basilar artery stenosis has been performed in patients suffering from brainstem ischemia [1-3]. However, the management of intracranial artery stenosis remains controversial, and it is not known what effects the bypass surgery has on the evolution of a bypassed arterial stenosis [9]. Recently we encountered a patient who had suffered from left-sided hemiplegia 4 months after STA-SCA anastomosis for basilar artery stenosis, and whose angiogram revealed occlusion of the basilar artery. This case is reported, and the possibility of progression of stenosis to complete occlusion of the arteries after revascularization procedure is discussed.

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### Case Report

A 53-year-old man was admitted to our hospital on November 22, 1983, complaining of motor weakness of the left extremities and double vision. For 1 year prior to admission he had experienced motor weakness and numbness of his left extremities, blurred vision, and dull occipital pain almost every day. With rest, these symptoms disappeared within 30 minutes. The past history included a 2-year history of hypertension, treated with antihypertensive agents. Neurological examination revealed a right oculomotor palsy (limitation of ocular movement and a slightly dilated pupil), left hemiparesis, broad-based gait, and numbness of the left arm. Lumbar puncture provided cerebrospinal fluid with a normal cell count and protein content. The ophthalmoscopic examination demonstrated marked arteriosclerotic changes in the retinal arteries. Computed tomography scans, with and without contrast medium, suggested cerebral infarction of the medial surface of the right frontal lobe, but no detectable lesion was found in the brainstem. As it was supposed that he suffered from ischemia of the brainstem, he was treated with the antiplatelet drug ticlopidine hydrochloride and intravenous low-molecular-weight dextran starting on the day of admission. On December 6 and 9, 1983, cerebral angiography was performed, but we could not insert the catheter via the femoral arteries into the abdominal aorta, because of tortuous iliac arteries. A left direct common carotid angiogram demonstrated the occlusion of the A<sub>2</sub> segment of the right anterior cerebral artery, whose territory was supplied by retrograde filling from the right middle cerebral artery. A left retrograde transbrachial vertebral angiogram showed greater than 90% midbasilar stenosis at the part just distal to the origin of the anterior inferior cerebellar artery. The narrowed segment was about 10 mm long on the angiogram (Figure 1). The posterior communicating arteries were not visualized on the carotid angiograms. About 3 weeks later he recovered without neurological deficit, and no more ischemic episodes occurred. Because the patient was considered to have a high risk of further brainstem infarction, a re-



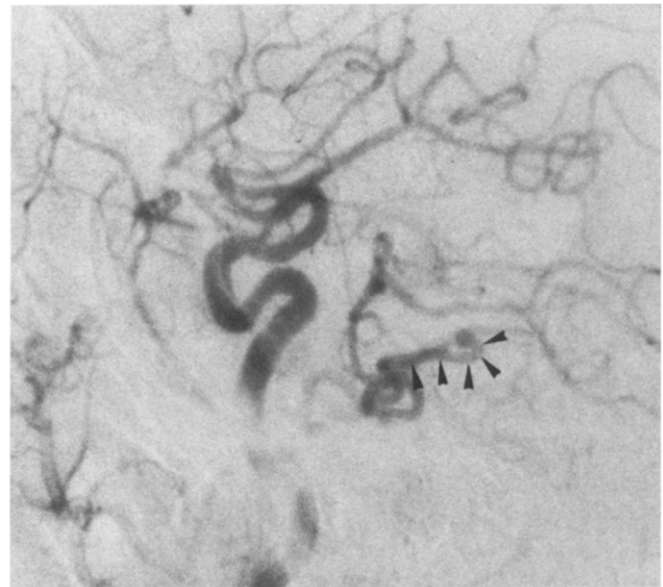
**Figure 1.** Left transbrachial retrograde angiogram disclosing a severe long-segmented stenosis, about 10 mm long, just distal to the origin of the anterior inferior cerebellar artery (arrow).

vascularization procedure—anastomosis between the superficial temporal artery and the superior cerebellar artery—was planned.

On February 23, 1984, the patient underwent the revascularization procedure. An end-to-side anastomosis was performed between the distal end of the frontal branch of the right superficial temporal artery and the ambient portion of the right superior cerebellar artery. Postoperatively he was neurologically normal except for a right trochlear nerve palsy that disappeared 2 months later. The pulsation of the right superficial temporal artery was distinctly palpable. He was maintained on the antiplatelet drug, and the inhibition of the platelet aggregation was well controlled. He was discharged on March 16, 1984.

On July 9, 1984, he experienced a sudden motor weakness of the left extremities while working and fell to the ground. He could not move his left extremities, and complained of numbness of the left arm, nausea, and dull occipital pain. Immediately after the onset he was transferred to our hospital.

On readmission there were no neurological deficits,

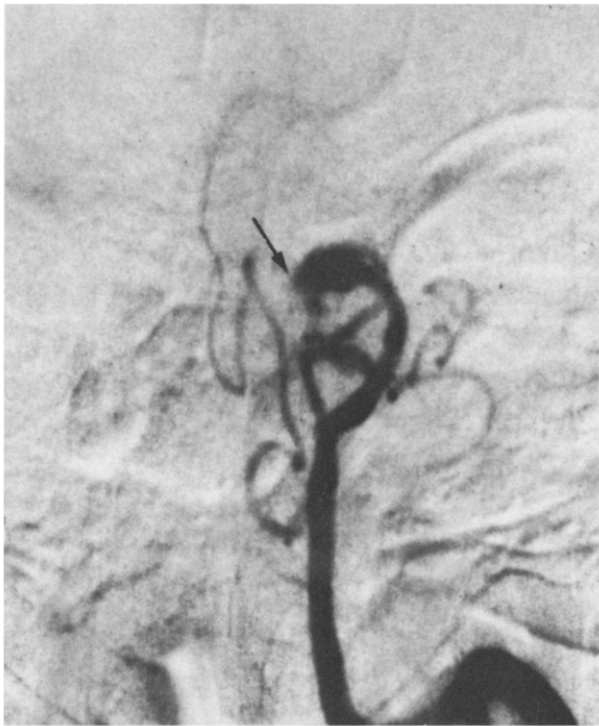


**Figure 2.** Right common carotid angiogram showing good filling of the upper basilar system via the dilated superficial temporal artery (arrowheads).

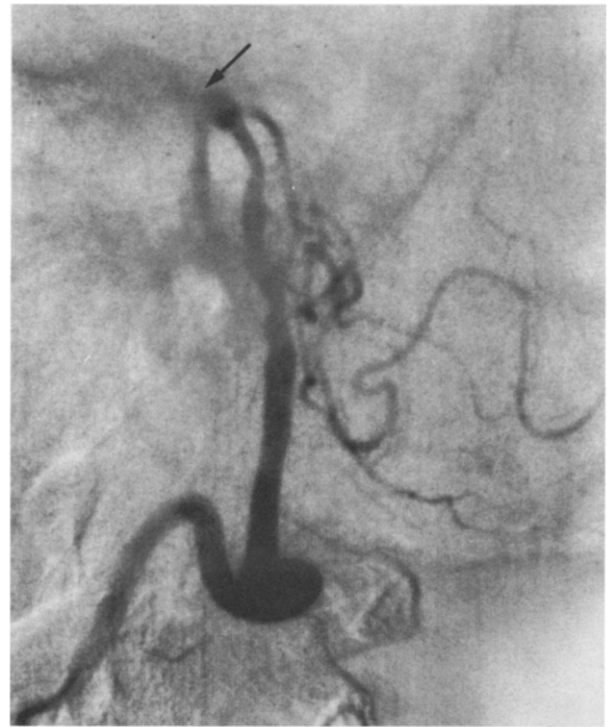
but the episode suggested a transient ischemic attack. One hour later the left hemiparesis occurred again and progressed. The neurological examination revealed conjugate deviation to the left, left facial palsy, dysarthric speech, left hemiplegia, and bilateral Babinski reflexes. On the next day, the neurological deterioration progressed further: horizontal nystagmus and palsies of the right abducens and right facial nerve were added to the previous symptoms. Palatal and lingual pareses were also noted bilaterally, but no evidence of involvement of the third cranial nerve was found. A computed tomography scan demonstrated no abnormal lesion in the brainstem. On July 13 and 20, 1984, cerebral angiography was performed. A right common carotid angiogram showed excellent filling of the upper basilar system via the superficial temporal artery (Figure 2). A left retrograde transbrachial vertebral angiogram disclosed complete occlusion of the previously stenotic basilar artery segment just distal to the origin of the anterior inferior cerebellar artery (Figure 3). He was treated with urokinase and low-molecular-weight dextran, and his neurological deficits gradually improved. Although he remained paretic 2 months after the onset, he could walk without a stick, and continued to improve slowly.

## Discussion

Occlusion of the bypassed artery after STA-MCA anastomosis for middle-cerebral-artery or internal-carotid-artery stenosis has been reported recently. The occlusion occurred asymptotically in some cases [4],



A



B

**Figure 3.** Left transbrachial retrograde angiograms showing complete occlusion of the previous stenotic lesion (arrow). (A) Anteroposterior view; (B) lateral view.

but in almost all cases was associated with neurological symptoms, and the angiograms generally showed good filling of the bypasses [4,8,9]. Most of the patients suffered from neurological symptoms within a few days after their bypass operations. It was postulated that the vascular stasis and the decrease of blood flow through the stenotic artery after the bypass surgery would predispose the stenosis to occlusion in the immediate postoperative period.

Ausman et al [2] first reported a successful surgical procedure, superficial temporal artery-proximal superior cerebellar artery anastomosis, for a patient with upper-brainstem ischemia. In their report the patient showed recurrent nonhemispheric episodes, and repeated angiograms showed the progression of midbasilar stenosis to occlusion. The bypass surgery was performed for ischemia of the upper brainstem because of the midbasilar occlusion. In the literature these surgical procedures were done in patients with upper-brainstem ischemia because of basilar-artery occlusion or stenosis [3], and in the latter case postoperative occlusion of the bypassed artery, as occurred in the intracranial internal carotid artery system, could be expected. Little information is available regarding the natural history of patients with basilar-artery stenosis, but it seems more

severe than other intracranial-artery stenoses [5-7,10,11]. The prognosis of basilar artery stenosis depends on collateral circulations involving the long circumferential cerebellar vessels with late filling of the distal segment of the basilar artery. Our case had no angiographic visualization of the posterior communicating artery or other collaterals, and severe long-segmented stenosis was present at the midbasilar artery. He experienced transient nonhemispheric symptoms almost every day for 1 year prior to admission. In our opinion he had a high risk of major brainstem infarction; consequently, we adopted a revascularization procedure as appropriate treatment. As a result, he developed brainstem infarction 4 months after the bypass surgery. However, the residual symptoms were not severe, and oculomotor nerve palsy, which meant ischemia of the midbrain, was not present during the second hospitalization. The progression of the stenosis to occlusion seemed to be a natural course because the preoperative angiogram revealed a long segment of severe stenosis of the midbasilar artery, and the occlusion occurred 4 months after the surgery. We conclude that STA-SCA anastomosis plays an important role in the prevention of fatal brainstem infarction.

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