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Transluminal Angioplasty of the Vertebral and Basilar Artery

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Transluminal angioplasty of brachiocephalic vessels for atherosclerotic lesions is now being performed in selected cases. We have thus far treated 17 cases of vertebral artery stenosis and one case of basilar artery stenosis by intravascular balloon dilatation techniques. Clinical presenting symptoms included vertebral basilar insufficiency, repeated transient ischemic attacks (TIAs), and multiple strokes. We performed successful transluminal angioplasty in 16 patients with marked narrowing (>70%) of the dominant vertebral artery from atherosclerosis. One patient with basilar artery stenosis with tandem atherosclerotic lesions was also treated by angioplasty techniques. Repeat angiography at 3- to 12-month intervals has revealed continued patency at the angioplasty site. Complications occurred in our one patient with basilar artery angioplasty, who suffered a brainstem infarction after treatment, and in one patient who had a TIA after bilateral vertebral artery angioplasty. Two other patients had residual vertebral stenosis but remained asymptomatic after the procedure. All other patients who had successful dilatation were asymptomatic at 6 months to 2 years (mean, 15 months) of follow-up.

These initial studies indicate that vertebral artery angioplasty may be effective for treating high-grade atherosclerotic lesions and for improving blood flow to the posterior circulation. Angioplasty of the basilar artery is technically more difficult and has a higher degree of risk because of the many perforating branches supplying the brainstem.

Percutaneous transluminal angioplasty (PTA) is being performed with increasing frequency in the innominate, subclavian, external, and internal carotid vessels [1–11]. Thus far, this technique appears to be effective for the treatment of hemodynamically significant atherosclerotic lesions. This is particularly important for vessels located in areas that are more difficult to treat surgically, such as the proximal vertebral and basilar arteries. We report our experience with transluminal angioplasty in 17 cases of hemodynamically significant atherosclerotic lesions of the vertebral artery and in one case of stenosis involving the basilar artery.

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Subjects and Methods

Patients included 10 men and seven women ranging in age from 46–77 years old (mean, 62.5). Percutaneous transluminal angioplasty (PTA) of the vertebral artery was performed in 13 patients who presented clinically with symptoms of vertebral basilar insufficiency, including dizziness, diplopia, bifacial numbness, vascular headaches, weakness, and ataxia. Two patients presented with symptoms of multiple transient posterior circulation ischemic attacks, which included transient cortical blindness, memory disturbance, and nystagmus. Two patients presented with multiple embolic strokes and had symptoms of homonymous hemianopsia, poor eye-hand coordination, visual agnosia, and vertical gaze nystagmus. All patients were managed in the acute stage of presentation by systemic heparinization or were placed on strict therapeutic doses of oral anticoagulants, including Coumadin, aspirin, and/or dipyridamole (Persantine). Only patients who failed to respond to conventional medical therapy were accepted for PTA.

In 15 patients, PTA was performed on the dominant proximal vertebral artery for athero-

sclerotic lesions judged to be hemodynamically significant with greater than 70% stenosis. In one patient with multiple TIAs and failed medical therapy, bilateral angioplasty was performed for lesions greater than 60%. One patient presented with high-grade tandem atherosclerotic lesions of the proximal and mid-basilar artery. This patient had angioplasty performed on both segments of the basilar artery.

Patients undergoing PTA of the vertebral artery were maintained on oral or parenteral anticoagulants for at least 5 days before the procedure. Using the Seldinger technique, the femoral or axillary artery was entered and 5,000 units of heparin was then given intravenously. A 5.0-French catheter was guided to the site of stenosis and a 260 cm 0.035 in. straight guidewire was advanced through the stenotic segment of the vertebral artery. The stenotic segment was then traversed by a tapered 5.0- to 7.0-French straight polyethylene catheter over the guidewire. This was immediately removed and then exchanged for an Olbert (Meadox Surgimed, Oakland, NJ) or Meditech (MediTech, Inc., Watertown, MA) dilatation balloon catheter. The balloon size used for vertebral artery angioplasty was 4.0–6.0 mm in diameter, depending on the parent vessel size, and 2.5 cm in length. The balloon was inflated once or twice to maximal pressure until the stenotic zone was successfully dilated. It was then immediately deflated and the catheter and guidewire removed. A postangioplasty angiogram was then performed. All patients were clinically monitored throughout the procedure for any changes in neurologic status.

Patients were closely observed overnight and, if stable, were discharged home within 24 hr on aspirin (325–650 mg/day) and/or Persantine (75–150 mg/day). Patients were followed clinically at 1-week, 1-month, 3-month, and 12-month intervals. Repeat angiography was performed at 3- to 12-month intervals to assess the angioplasty site.

PTA of the basilar artery for atherosclerosis was performed in a similar fashion except that surgical exposure of the C1 segment of the vertebral artery was performed and a 2.5-mm and 3.0-mm balloon dilatation catheter was used.

Representative Case Reports

Case 1: Vertebral Artery Angioplasty

A 76-year-old woman presented with left-sided amarosis fugax and a carotid bruit. An aortic arch angiogram revealed a high-grade stenosis of the left distal common carotid artery and the left proximal vertebral artery. A carotid endarterectomy was performed with good results.

Two years later, the patient was readmitted with episodes of bilateral leg and right arm weakness, vomiting, ataxia, and left-sided sensory deficits. Clinically, she was diagnosed as having had a stroke in the posterior circulation. She was anticoagulated but during hospitalization she suffered several more episodes of vertebral basilar insufficiency and TIAs, along with elevation of systolic blood pressure to 210 mm Hg. A repeat angiogram showed 60% stenosis of the right proximal vertebral artery secondary to atherosclerosis and greater than 80% stenosis of the left proximal vertebral artery (Fig. 1).

Transluminal angioplasty was performed once across the left-sided lesion with a 4.0-mm Olbert balloon dilatation catheter. The immediate postangioplasty angiogram showed moderate widening of the vessel at the angioplasty site along with vertical fissuring and subintimal hemorrhage of the atherosclerotic plaque (Fig. 1B).

The patient had immediate clinical and angiographic improvement with normalization of blood pressure, decreased headaches, ataxia, and nausea. She was discharged on 5 gr aspirin/day.

The patient remained asymptomatic, and a repeat angiogram was performed 10 months later. This demonstrated a widely patent proximal left vertebral artery at the angioplasty site with return to normal luminal diameter (Fig. 1C). There was no evidence of restenosis, ulceration, plaque, or intimal dissection. Clinically, at 24 months postangioplasty the patient remains asymptomatic.

Case 2: Vertebral Artery Angioplasty

A 60-year-old woman presented with TIAs and vertebral basilar insufficiency. Her symptoms included episodes of visual field loss,

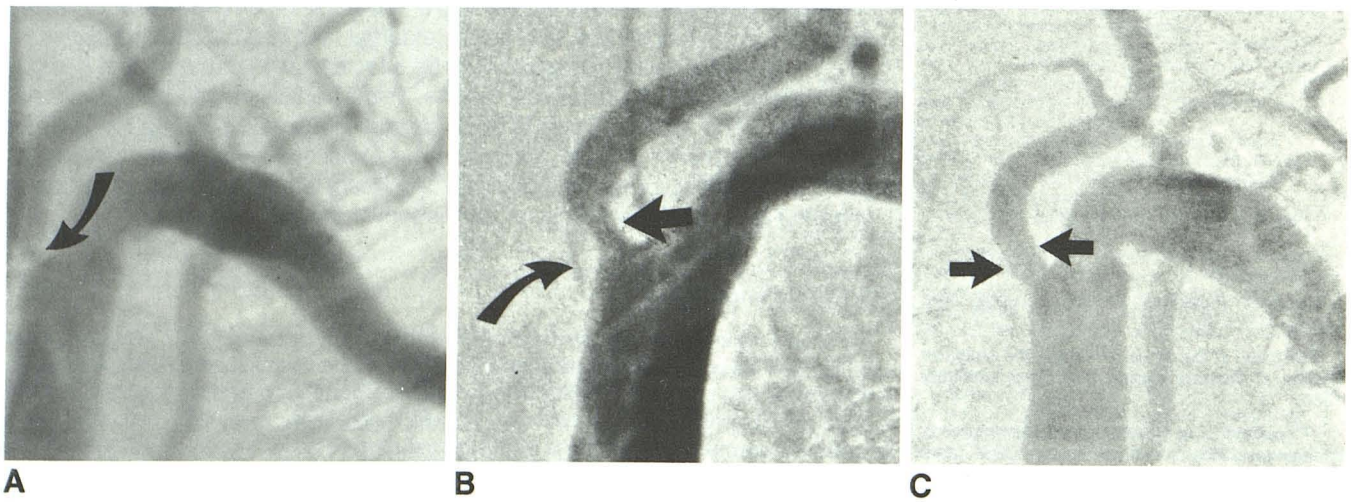

Fig. 1.—*A*, Left subclavian artery angiogram showing high-grade stenosis of left proximal vertebral artery (*arrow*).
***B*, Immediate postangioplasty angiogram showing mild increase in diameter of proximal vertebral artery (*straight arrow*) along with vertical fissuring of atherosclerotic plaque. *Curved arrow* shows contrast in subintimal region of atherosclerotic plaque.**
***C*, Left vertebral angiogram obtained 10 months after angioplasty. Proximal left vertebral artery is widely patent with return to normal luminal diameter. There is no evidence of restenosis, ulceration, plaque, or intimal dissection (*arrows*).**

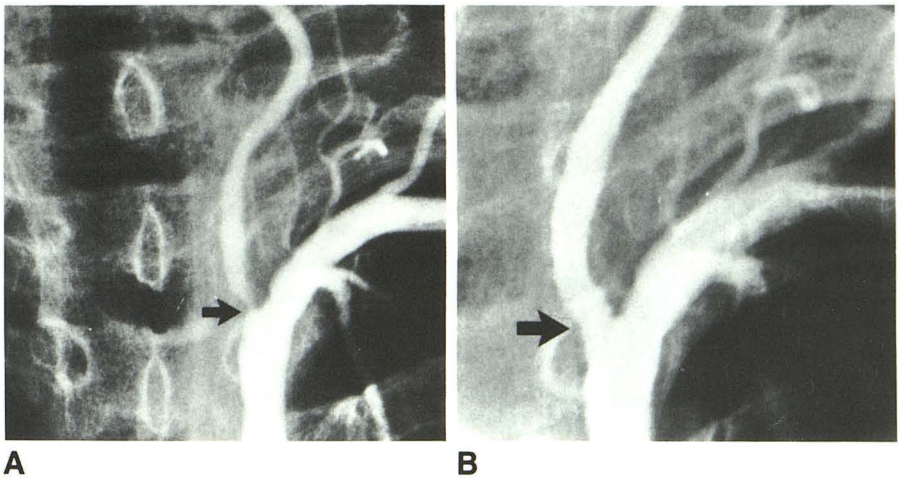

Fig. 2.—*A*, High-grade stenosis of proximal left vertebral artery due to atherosclerosis in a 60-year-old woman with symptoms of vertebral basilar insufficiency and transient ischemic attacks.

***B*, Angiogram 3 months after angioplasty shows complete return to normal luminal diameter of proximal vertebral artery (*arrow*).**

incoordination, ataxia, dizziness, and nystagmus. She had previously suffered a stroke in the left cerebral hemisphere. Angiography revealed occlusion of the left internal carotid artery and high-grade stenosis (>80%) of the proximal left vertebral artery (Fig. 2A). An extracranial to intracranial bypass graft from the superficial temporal artery to the middle cerebral artery was attempted but was unsuccessful. Soon after surgery, despite intravenous heparin therapy, she developed increasing aphasia and TIAs in the posterior circulation. PTA of the left proximal vertebral artery was performed using a 4.0-mm dilatation balloon catheter.

Immediately after the angioplasty procedure, the patient's aphasia improved along with her symptoms of vertebral basilar insufficiency. Presumably, her aphasia improved as a result of increased flow to the middle cerebral artery territory, across the posterior communicating artery. A repeat vertebral angiogram performed 3 months later showed a complete return to normal luminal diameter at the angioplasty site (Fig. 2B). At 10 months postangioplasty, the patient continues to do well.

Case 3: Tandem Stenotic Lesions of the Basilar Artery

A 76-year-old man presented with multiple episodes of brainstem ischemia. His symptoms included left-sided hemiparesis, numbness, and paresthesias; bilateral blurring of vision; hemianopsia; and rotational vertigo. The patient was placed on intravenous heparin, oral Coumadin, aspirin, and Persantine. In spite of this, he continued to have recurrent TIAs 2–4 times a day.

Cerebral angiography showed tandem high-grade stenotic lesions of the proximal and mid-basilar artery (>80%) (Fig. 3A). After 6 weeks in the intensive care unit without improvement in symptoms, the patient was referred for transluminal angioplasty. Three attempted dilatations of the basilar artery from a femoral cerebral approach with silicone and latex balloons were unsuccessful. During inflation, the balloons were deformed by the atherosclerotic plaque, and there was no significant change in caliber of the lesions.

The patient continued to be symptomatic, and a repeat treatment under general anesthesia and barbiturate coma, using a surgical approach to the C1 portion of the vertebral artery, was performed. A balloon dilatation catheter with a 2.5-mm diameter balloon was guided to the site of basilar stenosis under intraoperative fluoroscopic control. Dilatation was performed for 10 sec and then repeated once with a 3.0-mm balloon. The catheter was then advanced to the site of stenosis in the mid-basilar artery and dilated for 10 sec.

The postprocedure angiogram showed a widely patent basilar artery at both angioplasty sites (Fig. 3B). Unfortunately, after awakening from general anesthesia, the patient had a left-sided hemiparesis with impaired horizontal eye movement indicative of brainstem infarction. Over the course of several weeks, there was very little improvement in his condition.

Results

PTA of the vertebral artery was successfully performed in 16 of 17 cases without complication. The immediate postangioplasty angiogram usually revealed longitudinal fissuring of the atherosclerotic plaque due to fracturing with subintimal hemorrhage. In nine (53%) of 17 cases there was complete restoration of normal luminal diameter on follow-up angiography. Progressive dilatation is caused by healing of the intima and subintimal layers after dehiscense and disruption during the balloon dilatation procedure. There is delayed scarring and retraction of tissue, with subsequent increase in intraluminal diameter of the angioplasty site. In the other eight cases (47%), the residual stenosis was no longer symptomatic. All but two of these patients had substantially increased vessel diameters on follow-up angiography performed between 3 and 12 months. Patients were assessed by serial neurologic examinations 6 to 24 months (mean, 15 months) after the procedure.

Clinically, in all but one of the vertebral angioplasty cases there was marked improvement in symptoms without further episodes of vertebral basilar insufficiency, TIAs, or strokes. In one patient with repetitive TIAs, bilateral vertebral angioplasty was performed. This patient subsequently had three more episodes of TIAs over a 3-month period. Upon further workup, these events were thought to be caused by transient episodes of hypotension from cardiac arrhythmias.

One patient (case 3), who had tandem lesions of the basilar artery and presented with repetitive TIAs (on intravenous

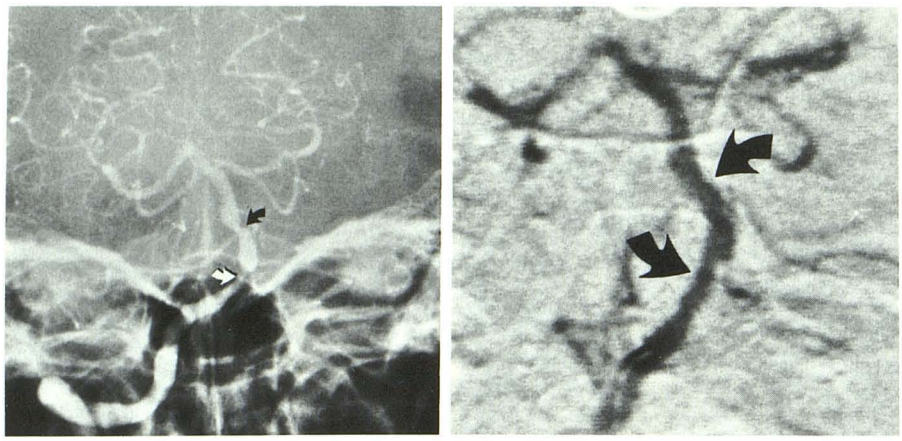

Fig. 3.—*A*, Right vertebral angiogram in Towne's projection. There are two high-grade atherosclerotic lesions involving proximal and mid-basilar arteries (*arrows*). Anterior inferior cerebellar artery arises between the two stenotic lesions.

***B*, Immediate postangioplasty digital subtraction film showing a widely patent basilar artery at both sites where percutaneous transluminal angioplasty was performed (*arrows*).**

heparin, oral Coumadin, aspirin, and Persantine), had PTA performed from a C1 vertebral approach. This patient had two to three daily episodes of hemiparesis, aphasia, and ataxia over a 6-week period in the intensive care unit prior to the procedure. After the procedure, the patient developed a brainstem infarct with hemiplegia. This was the one permanent complication in this series.

Discussion

The majority of patients presenting with symptoms of vertebral basilar insufficiency, repeated TIAs, and strokes in the posterior circulation territory can be managed conservatively with anticoagulation and antiplatelet medication [12–15]. Most patients with stenosis or occlusion of one proximal vertebral artery are asymptomatic. In patients with high-grade stenosis of one or both vertebral arteries that are refractory to medical management, PTA may be of benefit. In patients presenting with TIAs, their chances of having a stroke are 7% per year [16]; in 5 years, the chance is between 20–62% [17–20].

Thus far, initial reports on transluminal angioplasty of the vertebral artery appear favorable [21, 22]. Our experience with 17 successful procedures without significant complication also appears encouraging. However, we recommend that all patients be initially managed by medical therapy. Only if patients remain symptomatic should PTA be considered. Potential complications of angioplasty include stroke, vessel rupture, vessel occlusion, brainstem ischemia leading to respiratory arrest, and distal embolization. Particularly in the posterior circulation, these potential hazards may have disastrous consequences. Thus far, we have not had to perform a second angioplasty procedure for recurrent symptoms due to restenosis in any patient, although our longest clinical follow-up is only 24 months. Longer clinical follow-up with serial neurologic evaluations is needed.

Angioplasty of the basilar artery is still risky. One previous report by Sundt et al. [23] describes the successful treatment of two patients with high-grade basilar artery atherosclerotic lesions who had good short-term angiographic and clinical results after angioplasty. In one of our cases, the patient had tandem stenotic lesions of the basilar artery, and although surgical bypass was considered, it was felt to be too difficult. Both superior cerebellar and anterior inferior cerebellar arteries required a bypass procedure to possibly alleviate symptoms. In spite of aggressive medical therapy, the patient continued to have daily brainstem ischemia over 6 weeks, and PTA was thus undertaken as a final option. The patient's brainstem infarction after the angioplasty procedure was probably caused by occlusion of small perforating branches to the upper medulla and pons. This was in the same distribution as his prior TIAs.

Owing to the many perforating branches arising directly from the basilar artery to the brainstem and thalamus, angioplasty of this vessel is very different from angioplasty of other vascular territories. It has been shown experimentally that there are extensive intimal changes that occur in the acute and subacute stages after PTA. These are characterized by abrasion of the endothelium with disruption of the internal elastic lamina and media, edema, platelet aggregation, and accumulation of cellular debris [24]. These changes along with the mechanical action of compressing fibrous plaque that has already accumulated along the endothelial surface makes this a much more dangerous procedure as there is a relatively high likelihood of occluding the numerous perforating branches.

In conclusion, PTA of the vertebral artery can be effective for the treatment of hemodynamically significant atherosclerotic lesions with relatively low morbidity. If restenosis does occur, a second procedure can be performed. If this technique fails, then surgical bypass or endarterectomy should be considered. Long-term follow-up is still required to compare and assess these two treatment methods.

REFERENCES

1. Damuth HD, Diamond AB, Rappoport AS, Renner JW. Angioplasty of subclavian artery stenosis proximal to the vertebral origin. *AJNR* **1983**;4:1239–1242

2. Numaguchi Y, Puyau FA, Provenza LJ, Richardson DE. Percutaneous transluminal angioplasty of the carotid artery. *Neuroradiology* **1984**;26:527–530
3. Tsai FY, Matovich V, Hieshima G, et al. Percutaneous transluminal angioplasty of the carotid artery. *AJNR* **1986**;7:349–358
4. Motarjeme A, Keifer JW, Zuaka AJ. Percutaneous transluminal angioplasty of the brachiocephalic arteries. *AJNR* **1982**;3:169–174
5. Vitek JJ, Morawetz RB. Percutaneous transluminal angioplasty of the external carotid artery: preliminary report. *AJNR* **1982**;3:541–546
6. Vitek JJ. Percutaneous transluminal angioplasty of the external carotid artery. *AJNR* **1983**;4:796–799
7. Kobina GS, Bergman H. Angioplasty in stenosis of the innominate artery. *Cardiovasc Intervent Radiol* **1983**;6:82–85
8. Bockenheimer S, Mathias K. Percutaneous transluminal angioplasty in arteriosclerotic internal carotid artery stenosis. *AJNR* **1983**;4:791–792
9. Wiggli U, Gratzl O. Transluminal angioplasty of stenotic carotid arteries: case reports and protocol. *AJNR* **1983**;4:793–795
10. Theron J, Melancon D, Ethier R. "Pre" subclavian steal syndromes and their treatment by angioplasty. *Neuroradiology* **1985**;27:265–270
11. Galachia JP, Bajaj AK, Vine DL, Roberts RW. Subclavian artery stenosis treated by transluminal angioplasty: six cases. *Cardiovasc Intervent Radiol* **1983**;6:78–81
12. Dyken ML. Anticoagulant and platelet-antiaggregating therapy in stroke and threatened stroke. Symposium on cerebrovascular disease. *Neurol Clin* **1983**;1:223–242
13. Kistler JP, Roppor AH, Heros RC. Therapy of ischemic cerebral vascular disease due to atherothrombosis. *N Engl J Med* **1984**;311(2):100–105
14. Weksler BB, Lewin ML. Anticoagulation in cerebral ischemia. *Stroke* **1983**;14(5):658–663
15. Garde A, Samuelsson K, Fahlgren H, Hedberg E, Hjerne LG, Ostman J. Treatment after transient ischemic attacks: a comparison between anticoagulant drug and inhibition of platelet aggregation. *Stroke* **1983**;14(5):677–681
16. Millikan CH. Treatment of occlusive cerebrovascular disease. In: Siekert RG, ed. *Cerebrovascular survey report for joint council subcommittee on cerebrovascular disease.* Rochester, MN: Whiting Press, **1976**:141–171
17. Acheson J, Hutchinson EC. The natural history of focal cerebral vascular disease. *Q J Med* **1971**;157:15
18. Brown OW, Kerstein MD. The surgical management of transient ischemic attacks. *Angiology* **1984**;35(1):12–21
19. Cartlidge NE, Whisnant JP, Elueback LR. Carotid and vertebral basilar transient cerebral ischemic attacks. *Mayo Clin Proc* **1977**;52:117–120
20. Sandok BA, Furlan AJ, Whisnant JP, Sundt TM. Guidelines for the management of transient ischemic attacks. *Mayo Clin Proc* **1978**;53:665–674
21. Courtheoux P, Tournade A, Theron J, et al. Transcutaneous angioplasty of vertebral artery atheromatous ostial stricture. *Neuroradiology* **1985**;27:259–264
22. Theron J, Courtheoux P, Henriet JP, Pelouze G, Derlon JM, Maiza D. Angioplasty of supra-aortic arteries. *J Neuroradiol* **1984**;11:187–200
23. Sundt TM, Smith HC, Campbell JK, Vlietstra RE, Cucchiara RF, Stanson AW. Transluminal angioplasty for basilar artery stenosis. *Mayo Clinic Proc* **1980**;55:673–680
24. Zollikofer CL, Salomonowitz E, Sibley R, et al. Transluminal angioplasty evaluated by electron microscopy. *Radiology* **1984**;153:369–374