



Get Clarity On Generics

Cost-Effective CT & MRI Contrast Agents

**FRESENIUS
KABI**

[WATCH VIDEO](#)

AJNR

Angioplasty of subclavian artery stenosis proximal to the vertebral origin.

H D Damuth, Jr, A B Diamond, A S Rappoport and J W Renner

AJNR Am J Neuroradiol 1983, 4 (6) 1239-1242

<http://www.ajnr.org/content/4/6/1239>

This information is current as
of August 24, 2025.

Angioplasty of Subclavian Artery Stenosis Proximal to the Vertebral Origin

Harold D. Damuth, Jr.^{1,2}
 Arthur B. Diamond¹
 Arnold S. Rappoport¹
 John W. Renner¹

Percutaneous transluminal angioplasty was performed in nine patients with symptomatic proximal left subclavian artery stenoses. All cases were technically successful. One significant delayed complication occurred in a patient whose common carotid-to-subclavian artery bypass graft occluded several months after a successful subclavian artery angioplasty with a probable subsequent embolic occlusion of the internal carotid artery. The other patients were asymptomatic after follow-up evaluations of 4–23 months.

With the development of the Grüntzig balloon catheter, percutaneous transluminal angioplasty (PTA) has become a widely used method of treating occlusive disease throughout the vascular system [1, 2]. However, its reported use in stenotic lesions of the subclavian artery proximal to the vertebral artery origin has been very limited [3–6]. Reluctance to perform PTA in these lesions has resulted from fear of distal embolization into the cerebral circulation [7]. We report our experience with PTA in nine patients with proximal left subclavian artery stenosis.

Subjects and Methods

Nine patients with left subclavian artery stenosis located proximal to the vertebral artery origin underwent PTA. The patients were 49–69 years of age (average, 59 years). Eight were female. Seven patients complained of left arm weakness, paresthesias, and/or pain. In two patients, dizziness was the major symptom. One patient complained primarily of bitemporal headaches that resolved after PTA. One patient had an expressive aphasia as a result of a previous cerebral vascular accident and was an unreliable historian. One patient had a carotid-to-subclavian bypass graft as initial treatment for her subclavian stenosis. All patients had a differential systolic brachial pressure gradient of 20–78 mm Hg (average, 34.2 mm Hg) as measured by a sphygmomanometer, the left brachial pressure being lower than the right (table 1).

Five of the nine patients had angiographic evidence of subclavian steal; the other four had stenotic lesions without reverse vertebral flow. Four patients had additional significant extracranial carotid disease. Two patients who had symptoms confined to the left upper extremity had isolated left subclavian stenoses with no other vascular abnormalities. Four patients had symptomatic coronary artery disease.

The technical aspects of PTA have been described in the literature [1, 2, 8]. In all our cases a transfemoral approach was used. A 5 French polyethylene Hanafée catheter was placed through the stenotic lesion over a Bentson guide wire. The catheter's intraluminal position was confirmed by contrast injection and then 5,000 U of heparin was given intraarterially. An exchange for a 5 French polyethylene balloon catheter was made over a 240 cm Teflon-coated exchange guide wire. In four cases the stenosis was dilated with 6 and 7 French polyethylene straight catheters before balloon insertion. An 80 cm balloon catheter was of adequate length in all cases, and in each case the appropriate balloon size was 6 mm × 1.5 cm. The guide wire tip was not advanced beyond the proximal axillary artery to avoid intimal damage or arterial spasm. Intraarterial pressure gradient was

Received January 12, 1983; accepted after revision May 9, 1983.

¹Department of Radiology, Memorial Hospital Medical Center, 2801 Atlantic Ave., P.O. Box 1428, Long Beach, CA 90801-1428. Address reprint requests to A. B. Diamond.

²Department of Radiological Sciences, University of California, Irvine Medical Center, Orange, CA 92668.

AJNR 4:1239–1242, Nov/Dec 1983
 0195–6108/83/0406-1239 \$00.00
 © American Roentgen Ray Society

TABLE 1: PTA for Proximal Left Subclavian Artery Stenosis: Summary of Cases

Case No. (age, gender)	Systolic Blood Pressure Gradient (mm Hg)		Follow-up (months)
	Pre-PTA	Post-PTA	
1 (52,F)	20	0	23
2 (65,F)	78	16	20
3 (69,F)	40	12	20
4 (69,F)	26	10	14
5 (64,F)	40	10	13
6 (49,F)	28	10	13
7 (51,M)	36	0	7
8 (54,F)	NA	0	7
9 (57,F)	30	0	4

Note.—The only complication occurred in case 1, in whom a carotid-subclavian graft occluded several months after PTA.

measured on the initial patients, but the dampening effect of the catheter across the stenosis was believed to make this an unreliable measurement.

Careful monitoring of the neurologic status after dilatation was performed. Our patients are routinely sent to the postoperative recovery room after all interventional procedures. Patients without other significant medical problems were discharged within 24–48 hr. In all cases, premedication included 10 grains of aspirin for one or more days before the procedure. Persantine, 25 to 50 mg, was given to several patients as well. Daily aspirin was prescribed after dilatation on a long-term basis for all patients.

Results

All nine procedures were technically successful and performed with relative ease. There were no procedural complications. In six (66%) of nine cases, there was restoration of a normal luminal diameter. In the other three, some residual stenosis remained but was not considered hemodynamically significant.

Table 1 shows the reduced systolic blood pressure gradient after PTA. In all cases there was return of a normal radial pulse after PTA, often quite dramatically. The length of clinical follow-up is also shown in table 1.

Many patients who were most severely impaired by their subclavian stenosis (i.e., arm weakness, paresthesias, and/or pain) noted immediate and dramatic relief after PTA. Clinical follow-up was obtained on a regular basis, the longest being 23 months. No residual significant brachial pressure or radial pulse gradient was noted. With the exception of one patient who had a previous carotid-to-subclavian graft that thrombosed after PTA, there were no patients with recurrent symptoms. Follow-up angiograms were not obtained, except in our representative case.

Representative Case Report

A 52-year-old woman had repeated episodes of vertigo and left arm numbness for 1½ years. An arch angiogram obtained at another hospital at the time of initial evaluation revealed a markedly stenotic proximal left subclavian artery. A common carotid–subclavian artery bypass was performed. Some symptomatic relief occurred for about 3

months, after which vertigo, light-headedness and headaches recurred.

Ten months after the bypass, the patient was referred to Memorial Hospital Medical Center of Long Beach. An angiogram showed the graft to be patent, but there was narrowing of both proximal and distal anastomotic sites (fig. 1A). The proximal subclavian stenosis was unchanged.

An arch angiogram 4 months later because of persistent symptoms was essentially unchanged. A systolic blood pressure gradient of 30 mm Hg and a diminished radial pulse were noted on the left. Transluminal angioplasty of the proximal subclavian artery stenosis was performed successfully (fig. 1B). The patient noted immediate relief from dizziness and headache. Her hospital course was unremarkable and she was discharged in satisfactory condition.

A transient episode of expressive aphasia and heaviness on the right side occurred several weeks after angioplasty. There was complete resolution of the symptoms and the patient was placed on Coumadin. Despite adequate anticoagulation, she experienced recurrent episodes of abrupt onset of heaviness and numbness involving the right side of her face, right arm, and right leg. She also noted transient visual disturbances in the left eye.

An arch angiogram 4 months after angioplasty revealed that the common carotid–subclavian bypass graft had become occluded (fig. 1C). Total occlusion of the left internal carotid artery was also noted. The angioplasty site in the proximal subclavian artery remained widely patent. Subsequently, the patient noticed spontaneous resolution of her symptoms and was symptom-free 22 months later.

Discussion

Subclavian artery stenosis constitutes a small but significant part of extracranial arterial disease. Subclavian or innominate artery occlusion or stenosis occurs in 17% of all patients with cerebrovascular insufficiency [8].

Clinical manifestations of subclavian artery stenosis are variable and generally are divided into those referable to vertebrobasilar insufficiency and arm ischemia [9, 10]. In our series, two (22%) of nine patients primarily had symptoms of vertebrobasilar insufficiency, while the other seven (78%) experienced ischemic symptoms in their arms as their predominant manifestation.

Unusual clinical presentations of subclavian artery stenosis undoubtedly occur in situations in which other vascular disease exists and the stenotic subclavian artery is unable to serve as a significant collateral pathway. In one of our patients with concomitant carotid occlusive disease, symptoms of bitemporal headaches were relieved after subclavian angioplasty. Chest wall ischemia, with symptoms simulating angina pectoris, may be the result of a "steal" phenomenon from the intercostal arteries through the internal mammary, supreme intercostal, and/or lateral thoracic arteries [11]. Resolution of atypical chest pain after PTA may be explained by this phenomenon.

Before the advent of PTA, treatment of symptomatic subclavian artery stenosis was surgical. A transthoracic approach was used that carried an 8% mortality and a 25%

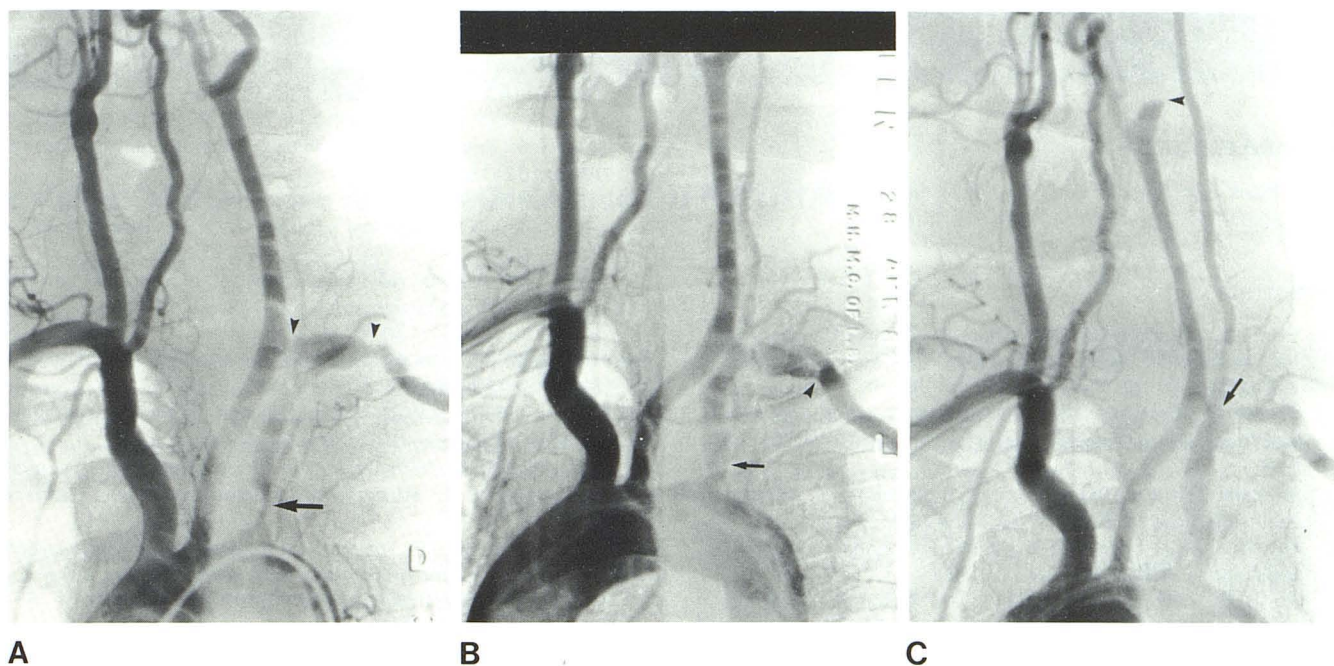


Fig. 1.—Arch angiograms, right posterior oblique projections. **A**, Proximal subclavian artery stenosis (arrow). Carotid-to-subclavian bypass graft with proximal and distal stenoses (arrowheads). Retrograde filling of left vertebral artery demonstrated on delayed filming (not shown). **B**, After PTA. Minimal residual stenosis (arrow). Antegrade filling of left vertebral artery. Distal

anastomosis of graft is seen better than in **A** (arrowhead). **C**, 4 months after PTA. Contrast is in proximal carotid end of occluded graft (arrow), and left internal carotid artery is occluded (arrowhead). Proximal subclavian artery is unchanged. Good antegrade left vertebral artery flow remains.

serious postoperative complication rate [9]. This technique was largely abandoned in favor of an extrathoracic, supraclavicular approach using a carotid-subclavian anastomosis either directly or via a venous or prosthetic graft. With this procedure, morbidity and mortality were reduced significantly [12]. Investigation of the carotid circulation before surgery is essential to prevent a carotid steal phenomenon, which would occur if significant carotid stenosis were present. If present, the carotid lesion should be corrected before anastomosis. In one study with long-term follow-up of 28 patients, 92% were either asymptomatic or clinically improved 3–10 years after extrathoracic surgery [13]. Although recent surgical advances have significantly reduced the risk of surgical correction of subclavian artery stenosis, the inherent risks of anesthesia remain. In addition, the cost of surgery and extended hospitalization are certainly more than would be anticipated with angioplasty.

An interesting situation occurred in our case 1, in which PTA was performed in a patient who had a malfunctioning, surgically placed, common carotid-subclavian bypass graft. Although there was immediate symptomatic relief after PTA, the patient began experiencing transient ischemic attacks about 3 weeks after PTA. The angiogram 4 months later showed occlusion of both the graft and left internal carotid artery (fig. 1C). Presumably, after successful dilatation of the subclavian stenosis, the equalization of pressure between the carotid and subclavian artery resulted in stasis of flow in the graft with subsequent thrombosis. We theorize

that propagation of the thrombus into the internal carotid artery from the graft resulted in embolization to the internal carotid artery and the multiple transient ischemic attacks. This situation illustrates a potential hazard in performing subclavian artery PTA in patients with common carotid-subclavian artery grafts. Because of this experience, we now consider PTA of the subclavian artery to be contraindicated in the presence of a patent bypass graft of this type.

Concern has been expressed about distal embolization into the vertebral artery during the dilatation procedure [3, 7]. While this is certainly a theoretical possibility, it has not occurred in any of our nine patients. Naturally, this concern is of no consequence when the vertebral artery is occluded or takes separate origin from the aorta. Unlike Bachman and Kim [3], we believe that the instant angioplasty is performed, antegrade flow is established. Consequently, PTA of the subclavian artery is believed to be safe regardless of the direction of flow in the vertebral artery.

In our experience, PTA in subclavian artery stenosis has proven to be a technically easy and highly successful procedure with no technical failures, no procedural complications, and no recurrences of symptoms in nine patients with follow-ups of up to 23 months. A stenotic lesion in the subclavian artery in a patient with cerebral, arm, or chest wall symptoms should be considered for PTA. Long-term patency should be anticipated as the subclavian is a high-flow, large-caliber vessel similar to the iliac system. However, we caution against the procedure in a patient with

carotid-subclavian bypass graft because of the risk of graft and carotid occlusion as the flow in the graft is reduced after elimination of the subclavian stenosis.

ACKNOWLEDGMENTS

We thank Diana Scura and Mary Ahlin for assistance in manuscript preparation.

REFERENCES

1. Grüntzig A, Kumpe DA. Technique of percutaneous transluminal angioplasty with the Grüntzig balloon catheter. *AJR* **1979**;132:547-552
2. Katzen BT, Chang J. Percutaneous transluminal angioplasty with the Grüntzig balloon catheter. *Radiology* **1979**;130:623-626
3. Bachman DM, Kim RM. Transluminal dilatation for subclavian steal syndrome. *AJR* **1980**;135:995-996
4. Novelline RA. Percutaneous transluminal angioplasty: newer applications. *AJR* **1980**;135:983-988
5. Martin EC, Diamond NG, Casarella WJ. Percutaneous transluminal angioplasty in non-atherosclerotic disease. *Radiology* **1980**;135:27-33
6. Motarjeme A, Keifer JW, Zuska AJ. Percutaneous transluminal angioplasty of the brachiocephalic arteries. *AJNR* **1982**;3:169-174, *AJR* **1982**;138:457-462
7. Kadir S, Kaufman SL, Barth KH, White RI Jr. *Selected techniques in interventional radiology*. Philadelphia: Saunders, **1982**:182-188
8. Ring EJ, McLean GK, Freiman DB. Selected techniques in percutaneous transluminal angioplasty. *AJR* **1982**;139:767-773
9. Fields WS, Lemak NA. Joint study of extracranial arterial occlusion VII subclavian steal—a review of 168 cases. *JAMA* **1972**;222:1139-1143
10. Herring M. The subclavian steal syndrome: a review. *Am Surg* **1977**;43:220-228
11. Buonanno FS, Toole JF. Can subclavian steal simulate angina pectoris? *Angiology* **1981**;32:329-333
12. Edwards WH, Mulherin JL Jr. The surgical approach to significant stenosis of vertebral and subclavian arteries. *Surgery* **1980**;87:20-28
13. Derin GP, Ballotta E. The surgical treatment of atherosclerotic occlusion of the innominate and subclavian arteries. *J Cardio-vasc Surg (Torino)* **1981**;22:532-538