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Treatment of Posterior Circulation Ischemia With Extracranial Percutaneous Balloon Angioplasty and Stent Placement

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Background and Purpose—Vertebrobasilar territory ischemia (VBI) leads to disabling neurological symptoms and poses a risk for stroke by an embolic or flow-related mechanism. We present our clinical experience in the endovascular treatment of patients with symptomatic VBI from severe atherosclerosis or dissection of the vertebral and subclavian arteries that was unresponsive to medical therapy.

Methods—Twenty-one patients (9 female, 12 male) with a mean age of 65.7 years (range 47 to 81 years) underwent treatment with percutaneous endovascular balloon angioplasty and stent placement. Sixteen patients (76.2%) had evidence of contralateral involvement, and 9 (42.8%) demonstrated severe anterior-circulation atherosclerosis. Nine patients had a previous infarct in the occipital lobe, cerebellum, or pons before treatment. Follow-up was available for all patients.

Results—Balloon angioplasty with intravascular stent placement was performed in 13 vertebral artery lesions (10 at the origin, 3 in the cervical segment) and in 8 subclavian lesions. The prestenting stenosis was 75% (50% to 100%) and was reduced to 4.5% (0% to 20%) after stenting. Six of the patients with proximal subclavian stenosis demonstrated angiographic evidence of subclavian steal, which resolved in all cases after treatment. All patients showed improvement in symptoms after the procedure except for 1 who developed a hemispheric stroke after thrombotic occlusion of an untreated cavernous carotid artery stenosis (rate of major stroke and mortality=4.8%). One patient (4.8%) had a periprocedural transient ischemic attack (TIA), and none had minor stroke. At long-term follow-up (mean=20.7±3.6 months) of the surviving 20 patients, 12 (57.1%) remained symptom-free, 4 (19%) had at most 1 TIA over a 3-month period, 2 (9.5%) had at most 1 TIA per month, and 2 (9.5%) had persistent symptoms. There were no clinically evident infarcts during the follow-up period.

Conclusions—Endovascular treatment using balloon angioplasty with intravascular stent placement for symptomatic stenotic lesions resulting in VBI that is unresponsive to medical therapy appears to be of benefit in this high-risk subset of patients with poor collateral flow. **(*Stroke*. 1999;30:2073-2085.)**

Key Words: angioplasty, balloon ■ atherosclerosis ■ stenosis ■ stents ■ subclavian steal syndrome ■ vertebrobasilar insufficiency

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Hemodynamic occlusive disease of the extracranial posterior circulation is the cause of vertebrobasilar territory ischemia in a significant number of patients.[1,2] Occlusive disease of the proximal and cervical portions of the vertebral artery represents a risk for posterior-circulation ischemia.[1] More proximal stenosis of the subclavian artery can also result in decreased ipsilateral posterior-circulation flow and in some cases leads to the syndrome of subclavian steal.[3] In symptomatic patients with poor angiographically demonstrable collateral flow who have failed medical therapy and in whom surgical revascularization and bypass techniques are associated with very high risk, balloon angioplasty has been reported to be of benefit.[4,5] More recently, isolated case reports have described the use of stent-supported angioplasty as an adjunct to decrease the incidence of elastic recoil and restenosis[6,7] and as a method to treat vertebral artery dissection and pseudoaneurysms.[8,9] Data in the literature, however, are insufficient to support the primary treatment of extracranial vertebral artery stenosis and dissection by stent-supported angioplasty.[10] We report our results in the treatment of 21 consecutive patients with symptomatic vertebrobasilar territory ischemia and poor collateral flow

Received May 25, 1999; final revision received July 19, 1999; accepted July 22, 1999.

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who were treated with angioplasty and stent placement after having failed medical therapy.

Subjects and Methods

Patient Population

We reviewed all the records and angiographic studies of patients referred to the University of California San Francisco Neurointerventional Radiology Section for treatment of vascular lesions involving the extracranial subclavian and vertebral arteries over a 5-year period between April 14, 1994, and April 14, 1999. The patients in the present study had presented with symptoms of posterior-circulation ischemia that had been refractory to medical management, including oral antiplatelet and/or anticoagulant agents. The angiographic studies were fully evaluated, and quantitative analysis of the lesions was performed either with the digital subtraction angiography computer directly or by computational analysis of the preprocedure and postprocedure films. Neurological follow-up was obtained by a neurologist and was supplemented by telephone interview. Review of the pretreatment cross-sectional imaging studies (computed tomography and MRI) was performed to determine the presence of established infarcts before the angioplasty and stent procedure.

Procedure

A baseline angiographic study was performed of the bilateral subclavian, vertebral, and carotid arteries to determine the extent of vascular disease and presence of collateral circulation. An activated clotting time (ACT) was obtained before onset of the angiographic procedure. The patient was given a weight-adjusted bolus of intravenous heparin 70 U/kg body wt followed by either an hourly bolus of heparin 35 U/kg body wt or a continuous infusion of 15 U · kg body $wt^{-1} \cdot h^{-1}$. The ACT was obtained after initial heparin bolus to ensure a value greater than twice baseline ACT, or >250 seconds. The patients then underwent the procedure either under monitored anesthesia care with short-acting intravenous sedation or under general endotracheal anesthesia. Vascular access was obtained via a single-wall puncture of either the common femoral artery (n=19) or the ipsilateral brachial artery (n=2, patients 5 and 8). A 7F to 9F access sheath was used (Avanti, Cordis Endovascular Systems). A guide catheter (Brite Tip, Cordis) was then carefully placed into the aortic arch or innominate artery for treatment of the proximal left or right subclavian lesions or in the subclavian artery proximal to the vertebral artery origin. The lesion was then traversed either with a 0.035-in exchange wire (Storq, Cordis) or a combination of a 3.2F microcatheter (Rapid Transit, Cordis) and an exchange microguidewire (Stabilizer 300 cm, Cordis). In the case of severe stenosis (80%), an angioplasty balloon catheter was used to predilate the lesion to allow subsequent passage of a balloon-mounted or self-expanding stent. In cases of milder stenosis (50% to 60%), such as in the case of intimal dissection, primary stenting was performed with either a self-expanding stent (WallStent, Schneider), a balloon-mounted stent (Palmaz, Johnson & Johnson), or a balloon-premounted stent (GFX, Arterial Vascular Engineering). After insertion of the stent and before deployment, an angiographic acquisition was performed to ascertain accurate location of the stent across the stenotic lesion. Postdeployment high-pressure balloon angioplasty (12 to 21 atm) was subsequently performed if needed. The intravenous heparin administration was either halted at the end of the procedure without reversal or maintained overnight for 12 hours. The patients were placed on an oral regimen of ticlopidine (250 mg PO BID) or clopidogrel (75 mg PO QD) for 6 weeks and on indefinite aspirin (325 mg PO QD). Certain patients who had either known severe stenosis in other vessels or concomitant severe intracranial atherosclerosis were maintained on oral warfarin therapy.

Outcome

Neurological outcome was determined at the longest duration of follow-up according to a 5-point scale as follows: excellent (score=1, asymptomatic, no neurological deficits and no symptoms of vertebrobasilar ischemia), good (score=2, no neurological deficits and at most 1 transient episode of vertebrobasilar ischemia over a period of 3 months after treatment), fair (score=3, minimal neurological deficit and at most 1 transient episode per month of vertebrobasilar ischemia), poor (score=4, no improvement compared with neurological status before treatment and/or persistent symptoms of vertebrobasilar ischemia), and death (score=5, regardless of cause).

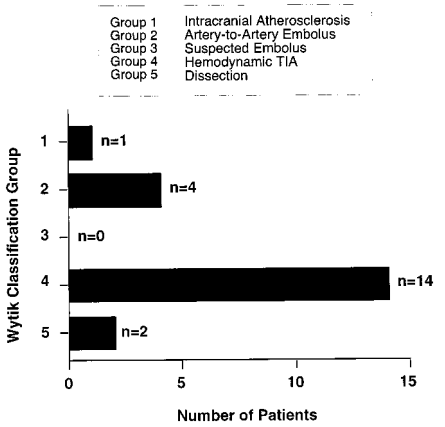


Figure 1. Distribution of patients at presentation based on the Wityk classification[1] for extracranial proximal vertebral stenosis.

Quantitative Analysis and Statistics

Severity of stenosis was computed according to the NASCET criteria with dimensions obtained either from the fluorographic appearance of a reference object or via the angiography digital computer system (Toshiba).[11] Specifically, the diameter of maximal stenosis ($D_{Stenosis}$) was measured along with the most proximal diameter of the distal normal vessel (D_{Normal}), and the degree of stenosis was computed as stenosis=$[1-(D_{Stenosis}/D_{Normal})]\times 100$.[11] The paired Student's t test and ANOVA were used in the comparison of numerical variables. A logarithmic transformation of the outcome score was performed before statistical analysis to minimize variability in variance among subgroups. For categorical analysis, a Pearson's χ^2 test was used. Statistical significance was assumed for a value of $P<0.05$.

Results

Patient Demographics

Nine female and 12 male patients were treated; their ages ranged from 47 to 82 years, with a mean of 65.7 years. Eighteen of the 21 patients (85.7%) had hypertension, 7 (33.3%) had hypercholesterolemia, 6 (28.6%) had coronary artery disease, and 4 (19%) had diabetes mellitus (Table 1). Twelve patients had a significant smoking history of >30 pack-years (57.1%). According to the classification of V1 vertebral proximal stenosis of Wityk et al,[1] 1 patient was in group 1, with intracranial high-grade stenosis; 4 patients were in group 2, with evidence of artery-to-artery embolus; 14 patients were in group 4, with transient ischemic attacks (TIAs) suggestive of hemodynamic spells involving the posterior circulation; and 2 patients were in group 5, with vertebral dissection (Figure 1). Ten of the patients (47.6%) presented with brain infarcts varying in age from subacute to acute, 9 of which were in the posterior-circulation territory. The distribution of the posterior-circulation infarcts was

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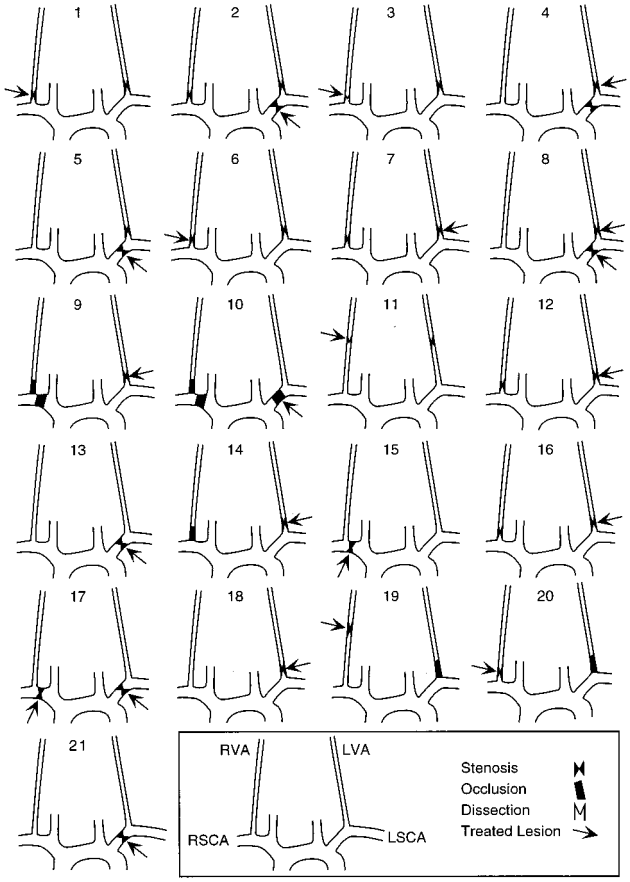


Figure 2. Schematic of the aortic arch with bilateral subclavian and vertebral arteries (carotid arteries are cut off proximally) in the 21 patients. Stenoses, occlusions, and dissections of the arteries are represented. Arrows point to the treated lesion site in each case.

as follows: occipital lobe 58.3%, pontine 25%, and cerebellar 16.7%.

Lesion Characteristics

The lesions treated were left-sided in 14 and right-sided in 7 cases (Table 2). The mean stenosis was 78.7±3.5% on left-sided and 68.6±5.1% on right-sided lesions (Figure 2). Nine patients (42.8%) had simultaneous unilateral or bilateral carotid stenosis of >60%, and 3 had unilateral or bilateral carotid occlusion (14.3%). Contralateral vertebral or subclavian occlusion or significant stenosis was encountered in 16 of 21 patients (76.2%). The location of the stenotic lesions treated in this cohort was as follows: 10 lesions (47.6%) at the origin of the vertebral artery, 3 (14.3%) at the midcervical vertebral artery, and 8 (38%) in the subclavian artery proximal to the vertebral artery origin. Of the 8 patients presenting with subclavian stenosis, 6 (75%) demonstrated subclavian steal, with retrograde flow in the vertebral artery ipsilateral to the stenosis. All 6 of these patients had resolution of the angiographic steal syndrome by the stent and angioplasty procedure (100%). The mean lesion stenosis before treatment was 75.2±14.1% and was decreased to a final value of 4.8±1.6% ($P<0.001$) after both angioplasty and stent deployment (Figure 3).

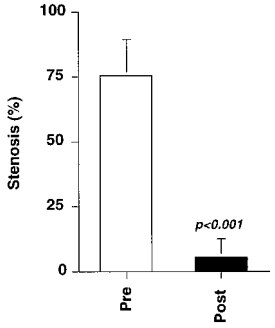


Figure 3. Graph of pretreatment and posttreatment stenosis of vertebral and subclavian stenosis determined from the digital subtraction angiograms by the NASCET method.[11]

Procedure-Related Complications

The procedure-related mortality was 4.8%, and morbidity was 9.5% (Table 3). There was 1 TIA within 24 hours of the procedure (patient 12). There were no minor or major strokes related to the territory treated during the procedure (0%), although the rate of overall major stroke was 4.8% (patient 8). Intimal dissection resulting from angioplasty was encountered in 7 procedures, 5 of which were treated by additional stent deployment; 2 cases (patients 12 and 18) in which the dissection was minimal and not flow-limiting had no further treatment.

There were 3 complications. One patient with a known severe, left cavernous internal carotid artery stenosis (>95%) (patient 8) underwent successful stent placement in the left subclavian and vertebral arteries with an uneventful immediate postprocedural course but developed acute thrombotic occlusion of the left internal carotid artery. Despite emergent angiography and superselective infusion of urokinase, the occlusion could not be recanalized. The patient developed a major hemispheric stroke and died after withdrawal of support. The second complication consisted of a stent infection at 10 days after successful deployment (patient 21); the source of infection was presumed to be phlebitis from an ipsilateral distal infected intravenous site. The ensuing arteritis required surgical removal of the stent and bypass grafting of the left vertebral artery; the patient had no neurological sequelae. The third complication (patient 5) resulted from mild thrombocytopenia (platelet count of 68 000/mm^3) after discharge from the hospital, which resolved with discontinuation of ticlopidine.

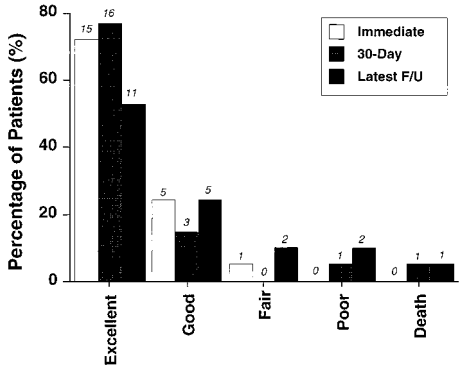


Figure 4. Neurological outcome immediately after the procedure, at 30 days, and at latest follow-up (mean, 20.7 months) as defined in Methods.

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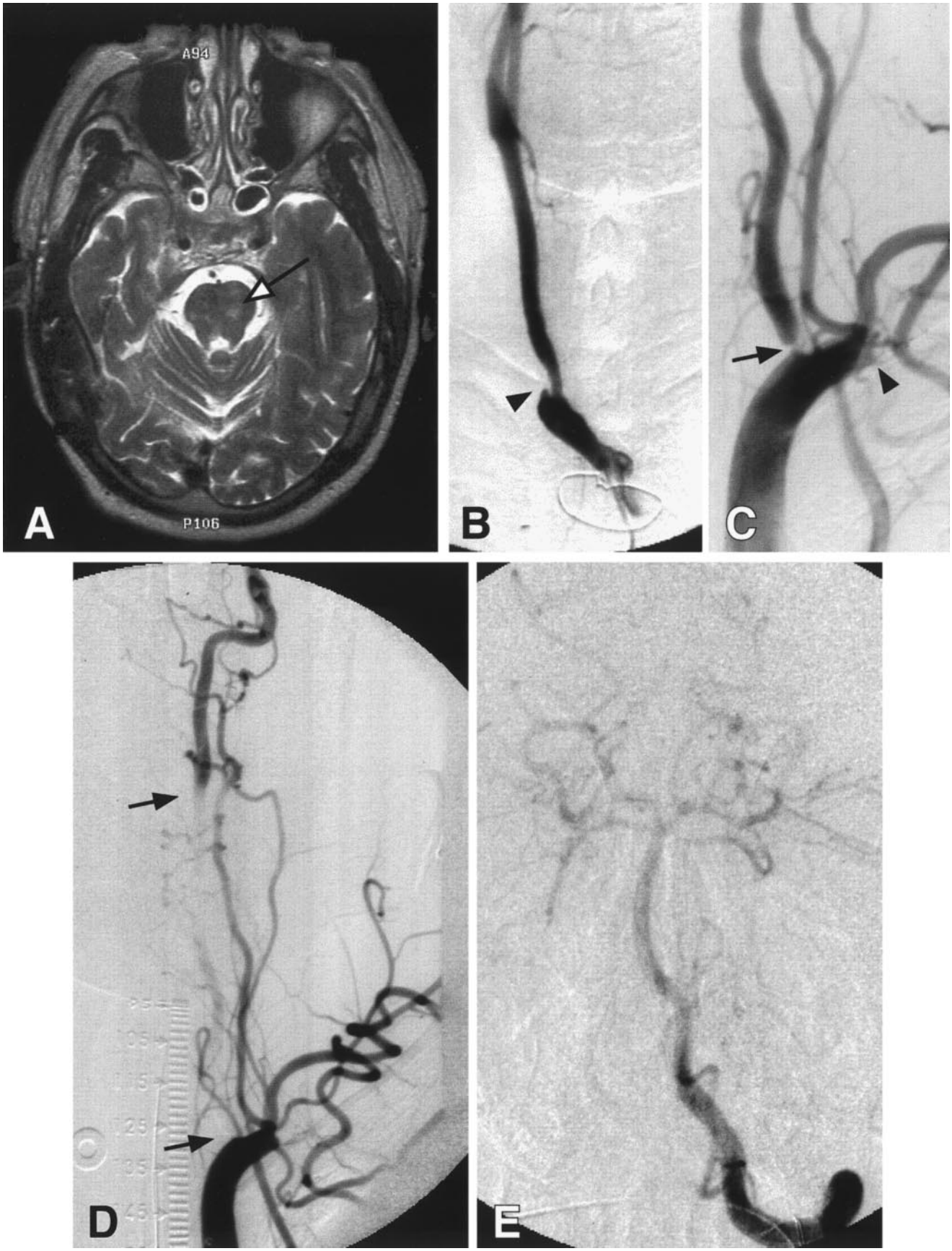


Figure 5.

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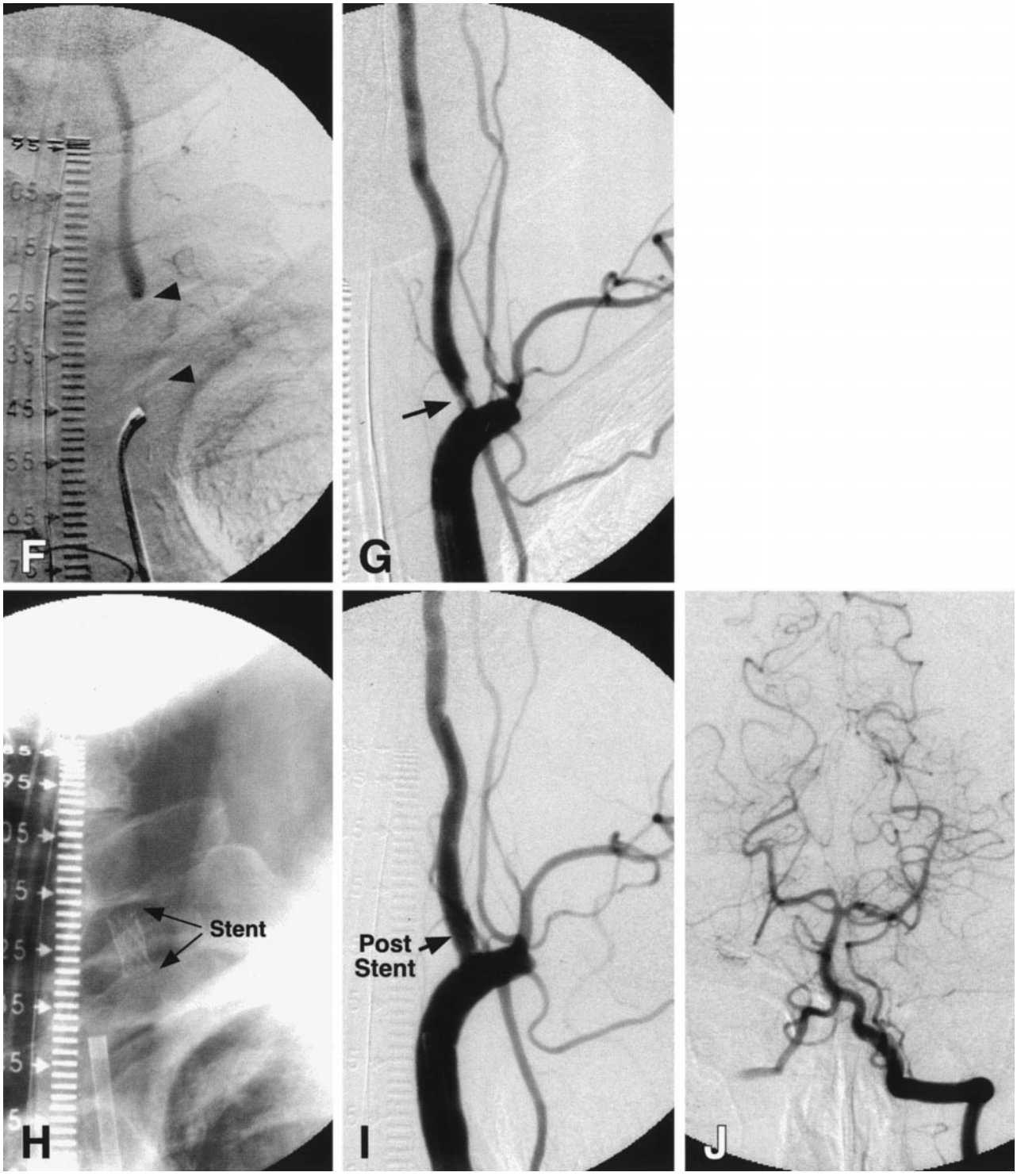


Figure 5. A 52-year-old right-handed man with diplopia, dysarthria, and left hemiparesis (patient 9). Axial T2-weighted MR study performed 2 months before procedure reveals increased signal focus in the left pons consistent with infarct (A). Digital subtraction angiography (DSA) of the innominate artery in the anteroposterior projection performed 2 months before the procedure reveals stenosis of the right common carotid artery origin, chronic occlusion of the right subclavian artery (arrowhead at stump), and no evidence of right vertebral artery filling (B). Angiography of the left subclavian artery shows a focal high-grade stenosis of the origin (arrow) and distal occlusion of the left subclavian artery (arrowhead at stump) (C). Repeat angiography at the time of the procedure reveals complete occlusion of the left vertebral artery (lower arrow) with distal reconstitution at the C4 vertebral level via muscular branches of the thyrocervical trunk (upper arrow) (D) and only faint filling of the posterior circulation (E). Late-venous-phase DSA of the left subclavian artery reveals retrograde contrast pooling within 1 cm of the cranial edge of the left subclavian artery (arrowheads) (F), confirming persistent patency of the proximal left vertebral artery beyond the occluded origin. DSA of the left subclavian artery after recanalization, thrombolysis (urokinase 250 000 U), and balloon dilatation of the occluded origin (G) shows persistent irregular stenosis (arrow). Fluorographic image of the Palmaz stent at the vertebral artery origin after deployment (H). Angiography of the left subclavian artery shows excellent filling of the left vertebral artery with reconstitution of the native vessel caliber (I). Posttreatment injection of the left subclavian artery (J) shows significantly improved perfusion of the posterior circulation with retrograde filling of the right vertebral artery (compare with E).

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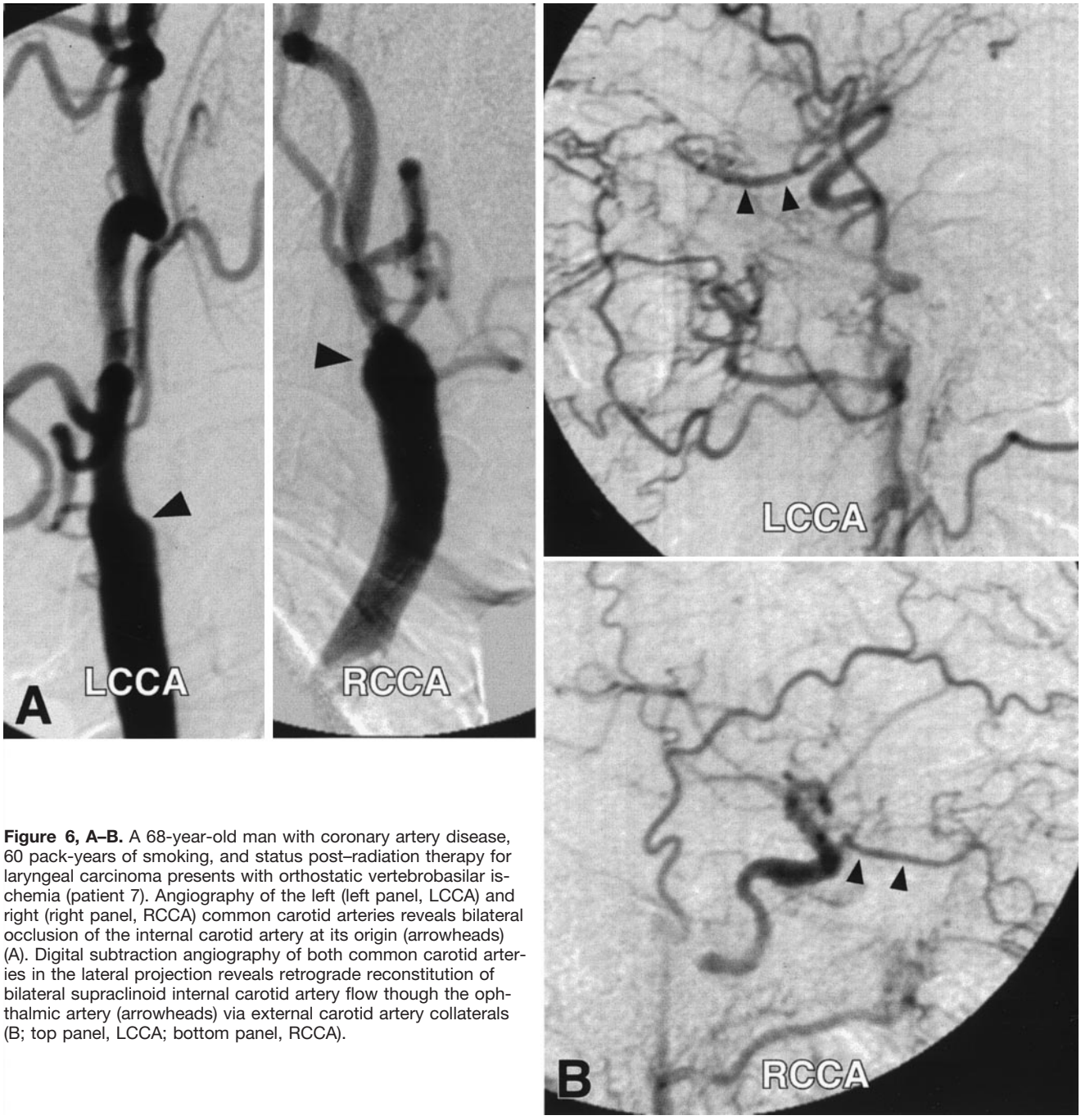


Figure 6, A–B. A 68-year-old man with coronary artery disease, 60 pack-years of smoking, and status post–radiation therapy for laryngeal carcinoma presents with orthostatic vertebrobasilar ischemia (patient 7). Angiography of the left (left panel, LCCA) and right (right panel, RCCA) common carotid arteries reveals bilateral occlusion of the internal carotid artery at its origin (arrowheads) (A). Digital subtraction angiography of both common carotid arteries in the lateral projection reveals retrograde reconstitution of bilateral supraclinoid internal carotid artery flow though the ophthalmic artery (arrowheads) via external carotid artery collaterals (B; top panel, LCCA; bottom panel, RCCA).

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Clinical Outcome

Neurological outcome was determined immediately after the procedure, at 30 days, and at latest follow-up (mean of 20.7±3.6 months). The majority of patients (20 of 21, 95.2%) were symptomatic and experienced either improvement in their posterior-circulation symptoms or no further episodes of ischemia after the procedure, except for 1 patient who developed a transient arm weakness that resolved within 24 hours (1 of 21, 4.8%) (Figure 4). At 30 days, neurological outcome was excellent in 13 patients (61.9%) and good in 7 patients (33.3%), and 1 patient (4.8%) died. At latest follow-up, the neurological outcome was excellent in 12 patients (57.1%), good in 4 patients (19%), fair in 2 patients (9.5%), and poor in 2 patients (9.5%), and 1 patient (4.8%) died. There were no clinically evident strokes in the posterior-circulation territory during the entire follow-up period. Assessment of symptom-free status (neurological score=1) revealed it to be inversely correlated with contralateral disease ($\chi^2=6.3$, $P<0.012$), carotid occlusion ($\chi^2=4.7$, $P<0.03$), and anterior-circulation atherosclerosis ($\chi^2=7.8$, $P<0.005$). Moderate carotid stenosis (<60%) and sex were not correlated with outcome.

Ultrasonographic or angiographic follow-up was obtained in 12 of the surviving 20 patients. Three of the 10 patients had evidence of mild-to-moderate restenosis, 1 had severe intimal hyperplasia, and 1 had complete occlusion at 1 year (patient 10). This occurred

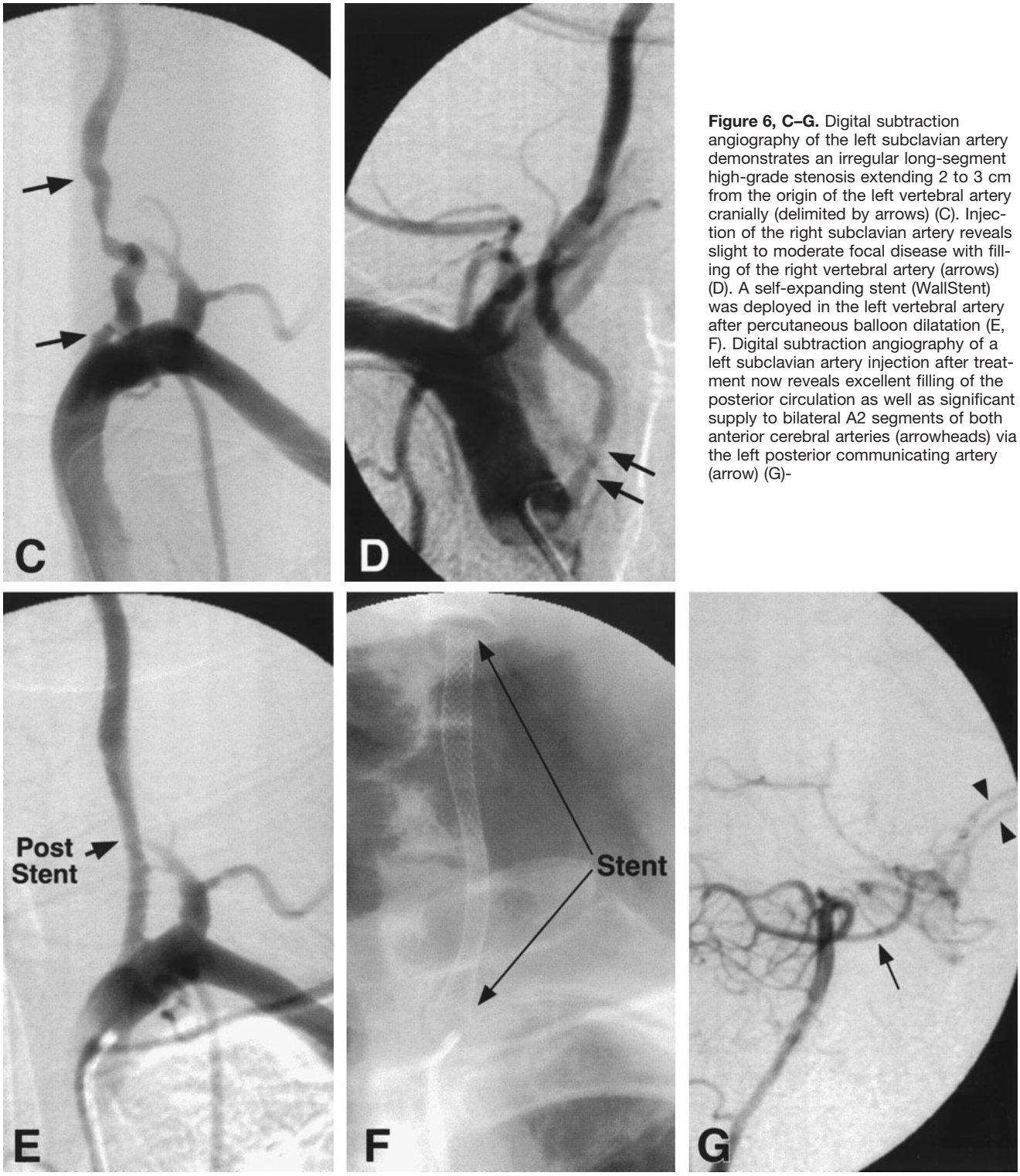


Figure 6, C–G. Digital subtraction angiography of the left subclavian artery demonstrates an irregular long-segment high-grade stenosis extending 2 to 3 cm from the origin of the left vertebral artery cranially (delimited by arrows) (C). Injection of the right subclavian artery reveals slight to moderate focal disease with filling of the right vertebral artery (arrows) (D). A self-expanding stent (WallStent) was deployed in the left vertebral artery after percutaneous balloon dilatation (E, F). Digital subtraction angiography of a left subclavian artery injection after treatment now reveals excellent filling of the posterior circulation as well as significant supply to bilateral A2 segments of both anterior cerebral arteries (arrowheads) via the left posterior communicating artery (arrow) (G)-

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in a patient with obliterative vasculopathy and concomitant occlusion of the left internal carotid artery, contralateral subclavian artery, and bilateral common femoral artery stenoses.

Illustrative Cases

Patient 9

A 52-year-old right-handed man with coronary artery disease and previous radiation therapy for a mediastinal tumor developed a stroke after noting sudden diplopia, left leg weakness, and dysarthric speech. An angiogram showed bilateral subclavian artery occlusions, with >90% stenosis of the left vertebral artery origin (Figure 5). An MR scan revealed a left pontine infarct. The patient was given intravenous anticoagulation with heparin, converted to warfarin, and discharged. Three days later, he had another episode of diplopia with worsening speech and left hemiparesis despite

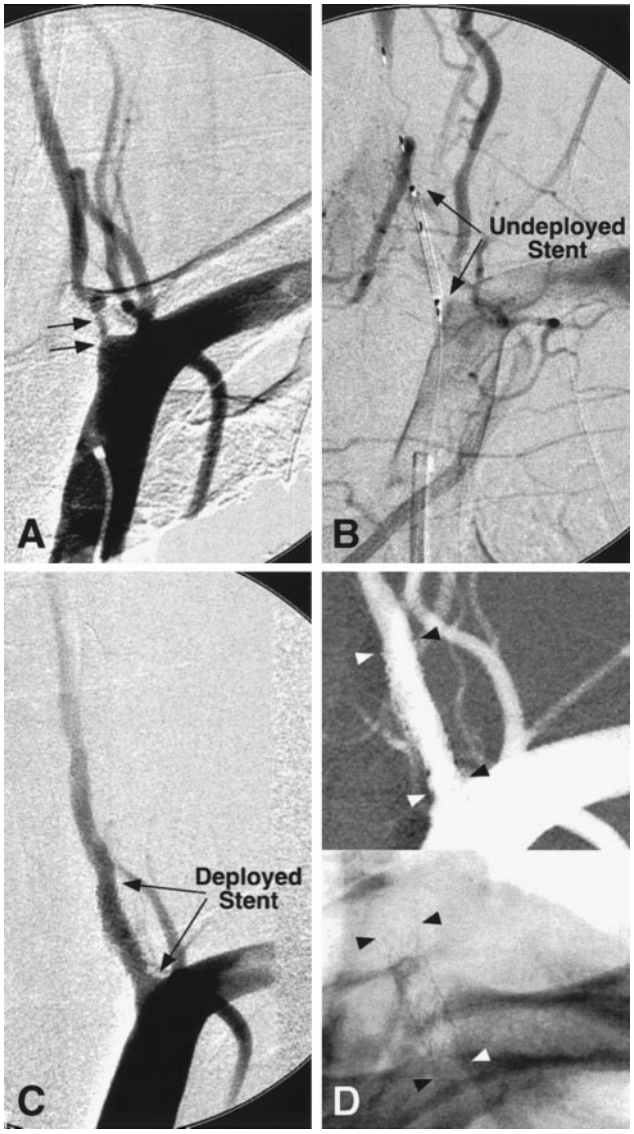


Figure 7. A 53-year-old right-handed man with hypertension and hyperlipidemia presented with transient attacks of visual loss and an occipital lobe infarct despite antiplatelet therapy 2 months before presentation (patient 18). Digital subtraction angiography of the left subclavian artery reveals a corkscrew-like irregularity and high-grade stenosis of the origin of the left vertebral artery (arrows) (A). A microguidewire was used to traverse the stenotic lesion and allow the passage of a stent-microcatheter, which is then positioned across the luminal irregularity (B). Postdeployment angiography of the left subclavian artery shows reestablishment of the native vessel caliber (C). Detail of the left vertebral artery origin shows stent interstices (arrowheads delineate cranial and caudal extent of the stent) in situ by plain fluorography (D, bottom panel) and corresponding angiogram (D, top panel)

therapeutic anticoagulation. The patient was transferred to our institution 2 months after his infarct. On examination, he was dysarthric and had a right-sided internuclear ophthalmoplegia and decreased sensation in the left V2 distribution with flattening of the left nasolabial fold. His motor examination revealed weakness in both upper (⅘) and lower (⅘, proximal greater than distal) extremities. Repeat angiography revealed, in addition to the previously noted occlusion of right subclavian origin, a new complete occlusion of the left vertebral artery origin. This vessel had been patent though stenotic in the angiogram performed 2 months before admission. Closer inspection revealed the distal left vertebral artery to be reconstituted at the C4 vertebral level via muscular branch collaterals of the thyrocervical trunk. Late venous-phase angiography showed retrograde stasis of the contrast column down to within 1 cm of the left subclavian artery. A microguidewire and catheter were navigated into the very proximal left vertebral artery, where a small test injection confirmed intraluminal placement without intimal dissection. With the microcatheter in this position, 250 000 U urokinase was administered over a period of 15 minutes. A balloon angioplasty catheter (4×20 mm Powerflex, Cordis) was used to dilate the origin, with an inflation of 13 atm of pressure. Postangioplasty angiography revealed persistent stenosis of the origin, suggesting lesion recoil. A Palmaz stent, mounted on an angioplasty balloon (5×20 mm Powerflex, Cordis) was therefore deployed across the residual stenosis. Posttreatment angiography showed excellent restoration of antegrade flow in the left vertebral artery with complete patency of the vertebral artery origin to its normal native diameter. Injection of the left subclavian artery showed excellent antegrade flow to the distal basilar, posterior cerebral, and superior cerebellar arteries, with retrograde flow into the right vertebral artery. The patient was maintained on heparin, was converted to warfarin, and was able to ambulate on his own on postprocedure day 3. He was discharged home the next day. He has shown good recovery from his left hemiparesis and ophthalmoplegia and has had no further neurological episodes at 26 months of follow-up. Ultrasound follow-up at 24 months confirms anterograde flow in the left cervical vertebral artery.

Patient 7

A 68-year-old right-handed man with coronary artery disease, a history of 60-pack-year smoking, and laryngeal cancer status postradiation therapy noticed an increase in the frequency of episodes of orthostatic dizziness, visual changes, and subjective heaviness in the arms and legs lasting ≈30 seconds. The patient underwent cerebral angiography, which revealed bilateral occlusions of the internal carotid arteries (Figure 6) at the origin. The right vertebral artery was irregular in contour, and the left showed a 1-cm segment of irregular caliber at its origin and a more distal stenosis. The patient was placed on oral anticoagulant therapy but showed no improvement in symptoms after a period of 3 months. Given the lack of response to medical therapy and the extensive carotid occlusive disease, a decision was made to treat the vertebral artery stenosis. A microguidewire and catheter were used to cross the left vertebral artery stenosis, which underwent balloon dilatation of the proximal and distal stenoses. This was followed by deployment of a self-expanding WallStent (5×40 mm) into the left vertebral artery origin, thus covering both stenoses. Postdeployment angiography revealed complete reconstitution of the vessel lumen, with evidence of normal distal perfusion. Injection of the left subclavian artery now showed filling of the bilateral anterior cerebral arteries from the left vertebral artery via a patent left posterior communicating artery (Figure 6G). The patient tolerated the procedure with no neurological deficits and was

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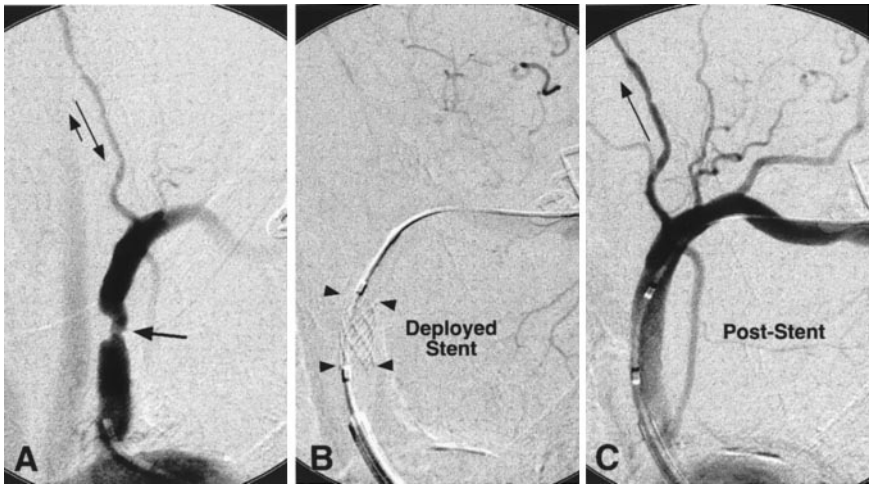


Figure 8. An 81-year-old woman with hypertension, diabetes mellitus, and peripheral vascular disease presents with complaints of orthostatic vertebrobasilar ischemia and subclavian steal syndrome (patient 17). Digital subtraction angiography (anteroposterior projection) of the left subclavian artery shows a hemodynamically significant stenosis (65%) (arrow) proximal to the origin of the left vertebral artery (A). Late-phase angiography shows flow reversal in the left vertebral artery consistent with a left subclavian steal, as suggested by negative velocities by cervical ultrasonography (short arrow shows direction of angiographic flow in early phase and long arrow in late phase) (A). A percutaneous transfemoral approach was used to place a guide catheter at the origin of the left subclavian artery and traverse the lesion with a guidewire; a balloon-mounted Palmaz stent was deployed across the lesion to a diameter of 7 mm (arrowheads outline interstices) (B). Digital subtraction angiography after stent deployment reveals excellent distal flow in the left vertebral and subclavian arteries with resolution of late-phase retrograde flow in the left vertebral artery (C).

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discharged on postprocedure day 4 on oral warfarin therapy. Despite angiographic improvement in the left vertebral flow and in posterior-circulation perfusion, the patient later complained of recurrent TIAs, stereotyped by loss of vision, blank stare with open eyes, followed by an unresponsive state lasting seconds to minutes. These attacks were different from the ones noted before the procedure because of their orthostatic independence. An ambulatory EEG was negative, but a Holter monitor showed a 9-second pause, for which the patient underwent placement of a VDD pacemaker.

Patient 18

A 53-year-old right-handed man with hypertension, hyperlipidemia, and status post C4-5 and C5-6 cervical fusion presented 14 months before admission with transient ischemic episodes consisting of visual field deficits. He was treated with aspirin but developed a stroke in the right occipital lobe 2 months later, which left a residual left lower quadrantanopia. The patient was placed on warfarin after MR angiography, and a conventional angiogram disclosed the presence of a highly stenotic and irregular lesion in the proximal left vertebral artery origin (Figure 7). Repeat angiography 7 months later revealed worsening in the stenosis and irregularity of the left vertebral artery origin. Consequently, an endovascular approach was undertaken to revascularize the left vertebral origin stenosis. A 7F guide catheter was placed in the left subclavian artery, and an AVE stent (GFX 4×18 mm) was positioned primarily across the lesion and deployed by balloon inflation to 9 atm (Figure 6). Poststent angiography revealed improved flow to the distal vertebral artery and posterior circulation. The patient was discharged on antiplatelet therapy and has remained asymptomatic at 4 months of neurological follow-up.

Patient 17

An 81-year-old 60-pack-year female smoker with hypertension, diabetes mellitus, and peripheral vascular disease who had had 2 previous aortofemoral bypass graft procedures began to have increasing episodes of orthostatic lightheadedness and dizziness. A vascular ultrasound of the cervical vessels disclosed the finding of bidirectional flow in the left vertebral artery consistent with left subclavian steal syndrome. The patient underwent diagnostic angiography showing 65% stenosis of the left subclavian artery proximal to the origin of the left vertebral artery (Figure 8). The left vertebral artery was nondominant and exhibited weak anterograde opacification, followed by retrograde flow in the late phase of the subclavian contrast injection, a phenomenon consistent with a left subclavian steal. A Palmaz stent was mounted on an angioplasty balloon catheter (Powerflex 7×20 mm, Cordis) and advanced across the lesion. Poststent angiography revealed resolution of the stenosis in the left subclavian artery origin with excellent anterograde flow in the hypoplastic left vertebral artery and resolution of the steal phenomenon. The patient noted immediate improvement in her symptoms after the procedure and has remained asymptomatic at 8 months of follow-up.

Discussion

Extracranial disease of the vertebral and subclavian arteries is difficult to diagnose and monitor with noninvasive techniques and has been less well defined than carotid vascular disease.[1,2,12] Moufarrij et al,[13] in their study of 96 patients with >50% stenosis, found the 5-year survival rate to be 27% lower than in a control population. In that study, the majority of deaths were cardiac-related (52.5%) compared with stroke (20%). This finding is consistent with our data, because patients with coexisting carotid disease had a higher stroke rate than those with isolated vertebral artery disease. Altogether, patients with vertebral artery stenosis had an 8.5-fold higher stroke rate than a control population.[13] Recently, Wityk et al[1] analyzed the subset of patients in the New England Medical Center (NEMC) Posterior Circulation Registry with extracranial vertebral artery disease (V1 segment). The majority of patients treated in our study (66.7%) were in Wityk group 4, that is, patients with significant blood flow restriction to the posterior circulation from vertebral artery or subclavian artery stenosis with a high frequency of contralateral stenosis or occlusion, and thus presented with hemody-

TABLE 1. Demographic Characteristics of Patients, Clinical Presentation, Lesion Site, History of Previous Infarct, Coexisting Medical Problems, Status of Contralateral Vertebral and Subclavian Arteries, and Extent of Anterior-Circulation Disease

Patient	Age, y/Sex	Presenting Symptoms	Lesion Site	Previous Infarcts	Contralateral Posterior-Circulation Disease	Significant Anterior-Circulation Disease (>70%)	Coexisting Medical Problems
1	79/F	Episodic VBI, blindness, loss of consciousness	Bilateral VA origin stenosis	None	Left VA origin stenosis (70%)	Y	HTN
2	70/M	Orthostatic VBI, lightheadedness, dizziness	LSCA origin stenosis	None	Left VA origin stenosis (60%) Right VA origin stenosis (50%)	Y	HTN, smoking, renal insufficiency, AAA, anemia
3	67/M	Right PCA infarct with residual quadriparesis 1 mo before admission	RVA origin dissection	Bilateral internal capsule, left occipital lobe	Occluded left VA origin, distal RVA stenosis (80%)	N	HTN, DM, Hchol
4	78/M	Episodic VBI, nausea, vomiting, intermittent quadriparesis	LVA origin stenosis	Right occipital and midpons infarcts	Midbasilar stenosis, occluded distal tandem right VA	Y	HTN, CAD, s/p MI
5	77/M	Episodic VBI, visual changes, dizziness	LSCA origin stenosis	Bilateral cerebellar and occipital infarcts	Left VA diffuse stenosis	Y	HTN, CAD, AAA, smoking, HChol, renal insufficiency
6	78/F	Episodic VBI, vertigo, dizziness, dysarthria	RVA origin stenosis	Pontine infarct	Left VA diffuse stenosis	N	HTN
7	68/M	Orthostatic VBI, dizziness, visual changes, transient hemiparesis	LVA origin and proximal stenosis	None	Right VA stenosis	Y Bilateral ICA occlusions	CAD, smoking, laryngeal CA, radiation therapy
8	74/M	Orthostatic VBI, dizziness, visual changes, staggering gait	LSCA and LVA stenosis with subclavian steal	Left occipital	No	Y	HTN, Hchol, PVD
9	52/M	Residual diplopia, dysarthria, left leg weakness	LVA thrombotic occlusion/stenosis/ thrombolysis of occluded LVA	Pontine infarct	RSCA occlusion RVA occlusion	N	CAD, HTN, smoking, lung CA, radiation therapy
10	47/F	Subclavian steal, dizziness, abnormal gait	LSCA origin occlusion	None	RSCA occlusion RVA occlusion	Y Left ICA occlusion	HTN, HChol, obliterative arteriopathy, basilar aneurysm, s/p GDC coiling
11	50/M	Episodic VBI, dizziness, acute visual field deficit	RVA dissection at C5 vertebra	Right cerebellar bilateral occipital infarcts	Subacute right PICA occlusion, chronic LVA dissection with flap and pseudoaneurysm on coumarin	N	None
12	69/M	Episodic VBI, sudden loss of consciousness and fall	LVA origin stenosis	None	Right VA stenosis (50%)	Y Left ICA occlusion	HTN, DM, PVD
13	51/F	Episodic VBI, vertigo, dizziness, diplopia, left arm claudication	LSCA stenosis, subclavian steal	None	Diffuse extracranial atherosclerosis	N	CAD, HTN, smoking, PVD, HChol, s/p aortobifemoral BG, s/p L CCA-SCA BG
14	62/F	Episodic VBI, dizziness, acute visual field deficit	LVA origin stenosis	Left occipital infarct	Occluded right VA	Y	HTN, smoking, hypothyroidism, PVD, prior right CEA
15	82/F	Episodic VBI, dizziness	RSCA stenosis, subclavian steal	None	Aberrant subclavian stenosis	N	CAD, HTN, smoking, hypothyroidism, s/p MI
16	65/F	Episodic VBI, dizziness, vertigo, bilateral hand numbness	Bilateral VA origin stenosis	None	Right VA stenosis	N	HTN, smoking

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TABLE 1. Continued

Patient	Age, y/Sex	Presenting Symptoms	Lesion Site	Previous Infarcts	Contralateral Posterior-Circulation Disease	Significant Anterior-Circulation Disease (>70%)	Coexisting Medical Problems
17	81/F	Episodic VBI, dizziness, vertigo, bilateral hand numbness	Bilateral SCA stenosis, left subclavian steal	Left corona radiata infarct	Right SCA stenosis	N	HTN, smoking, DM, PVD, s/p femoral BG
18	53/M	Episodic VBI, dizziness, lightheadedness	LVA origin stenosis	Left occipital lobe infarct	None	N	Hchol, smoking
19	59/F	Episodic VBI, staggering gait, imbalance, presyncope with head turn	Mid RVA stenosis	None	Occluded left VA origin	N	HTN, smoking, HChol
20	60/M	Vertebrobasilar territory ischemia	RVA origin stenosis	None	Occluded left VA origin	N	HTN, vehicular whiplash
21	56/M	Vertebrobasilar territory ischemia, dizziness, vertigo, transient hemiplegia	LSCA stenosis	None	Occluded right VA (V3 segment)	N	HTN, smoking, DM

VBI indicates vertebrobasilar territory ischemia; PCA, posterior cerebral artery; VA, vertebral artery; LSCA, left subclavian artery; RVA, right vertebral artery; LVA, left vertebral artery; RSCA, right subclavian artery; PICA, posterior inferior cerebellar artery; ICA, internal carotid artery; HTN, hypertension; AAA, abdominal aortic aneurysm; DM, diabetes mellitus; Hchol, hypercholesterolemia; CAD, coronary artery disease; s/p, superior/posterior; MI, myocardial infarction; CA, cancer; PVD, peripheral vascular disease; GDC, Guglielmi detachable coil; BG, bypass graft; and CEA, carotid endarterectomy.

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TABLE 2. Technical Summary of the Procedure Describing the Location of the Lesion, Pretreatment and Posttreatment Stenosis, Stent and Balloon Dimensions, Presence of Subclavian Steal Syndrome and Its Resolution by Treatment, Technical Complications if Any, and Treatment Steps Taken to Deal With the Complication

Patient	Treated Site	Pretreatment Stenosis, %	Poststent Deployment Stenosis, %	Stent Type/Dimensions (diameter×length, mm)	Subclavian Steal/ Resolution?	Technical Complications	Treatment of Complication
1	RVAO	90	0	WallStent (8×20)	...	Dissection from PTA	Stent placement
2	LSCA	80	20	WallStent (8×40)	N	None	...
3	RVAO	50	15	WallStent (5×20)	...	Dissection from PTA	Stent placement
4	LVAO	60	20	Palmaz (4×15)	...	None	None
5	LSCA	70	0	Palmaz (4×15, 8×40)	N	Iatrogenic LSOA Dissection distal to LVA orig	Tandem distal stent placement
6	RVAO	80	0	Palmaz (4×15)	...	None	...
7	LVAO	80	0	WallStent (5×40)	...	None	...
8	LSCA	80	15	Palmaz LSCA (8×40)	Y/Y	Cervical LVA dissection from PTA	Stent placement in cervical LVA
	LVAO			Palmaz LVAO (4×15)			
9	LVAO	100	0	Palmaz (5×20)	...	None	...
10	LSCA	100	0	WallStent (7×20)	Y/Y	None	...
11	RVAO	50	10	WallStent (5×20)	...	None	...
12	LVAO	80	10	WallStent (5×20)	...	Non–flow-limiting dissection	None
13	LSC	70	0	WallStent (10×20)	Y/Y	None	...
14	LVAO	70	0	Palmaz (4×15)	...	None	...
15	RSCA	70	10	WallStent (8×20, 8×20)	Y/Y	None	...
16	LVAO	80	0	Palmaz (4×15)	...	None	...
17	LSCA	LSCA 60	LSCA 0	Palmaz LSCA (7×15)	Y/Y	Dissection from first stent in RSCA	Tandem stent placement in RSCA
	RSCA	RSCA 70	RSCA 0	Palmaz RSCA (8×15, 8×15)			
18	LVAO	95	0	GFX (4×18)	...	Non–flow-limiting dissection	None
19	RVA	60	0	GFX (4×12)	...	None	...
20	RVAO	80	0	Palmaz (6...15)	...	None	...
21	LSCA	70	0	Palmaz (10×20)	Y/Y	None	...

RVAO indicates right vertebral artery origin; LVAO, left vertebral artery origin; PTA, percutaneous transluminal angioplasty; other abbreviations as in Table 1.

TABLE 3. Summary of Clinical Outcome, Immediately Postprocedure, at 30 Months and at Latest Follow-Up

Patient	Immediate Treatment Outcome	Procedure-Related Complications	Outcome at 30 Days	Latest Neurological Follow-Up, mo	Latest Neurological Outcome
1	Resolution of symptoms excellent	None	Excellent	68	Excellent
2	Resolution of symptoms excellent	None	Excellent	36	Good
3	Resolution of symptoms excellent	None	Excellent	18	Excellent
4	Resolution of symptoms excellent	None	Good	32	Fair
5	Resolution of symptoms excellent	Transient thrombocytopenia	Excellent	33	Excellent
6	Improvement in symptoms good	None	Excellent	30	Poor
7	Improvement in symptoms good	None	Good	32	Poor
8	Good; acute LICA occlusion at 24 h	Acute LICA cavernous occlusion at 24 h postprocedure, hemispheric infarct, death	Death	...	Death
9	Resolution of symptoms excellent	None	Excellent	26	Excellent
10	Resolution of symptoms excellent; Return of left radial pulse	None	Excellent	16	Fair
11	Good; partial improvement in visual field deficit	None	Good	22	Excellent
12	Improvement in symptoms good	TIA postprocedure consisting of mild right-sided motor weakness	Good	11	Good
13	Excellent; no further TIAs, return of left radial pulse	None	Excellent	14	Excellent
14	Resolution of symptoms excellent	None	Good	22	Good
15	Excellent; no further TIAs, return of right radial pulse	None	Excellent	26	Excellent
16	Resolution of symptoms excellent	None	Excellent	7	Excellent
17	Resolution of symptoms excellent	None	Good	8	Excellent
18	Resolution of symptoms excellent	None	Excellent	6	Excellent
19	Improvement in symptoms good	None	Good	3	Good
20	Resolution of symptoms excellent	None	Excellent	1	Excellent
21	Resolution of symptoms excellent	LSCA arteritis at 10 days and pseudoaneurysm requiring surgical stent removal and bypass grafting	Excellent	2	Excellent

LICA indicates left internal carotid artery; LSCA, left subclavian artery.

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namic spells or TIAs occurring with changes in orthostatic posture. In their analysis, Wityk et al found that group 4 patients constituted 16% of registry patients and had the highest proportion (92%) of bilateral vertebral artery abnormalities. Four patients in our group (19%) belonged to Wityk group 2: we did not find a significant difference in long-term outcome between these 2 subgroups of patients. The difference in proportion of group 4 patients between our study and the NEMC is undoubtedly the result of selection bias. Patients were referred to our service for endovascular treatment and inclusion after having undergone negative studies for a cardiac embolic source and after having failed oral anticoagulant or antiplatelet therapy. Analysis of our patient population identified hypertension as the most common medical problem, followed by smoking. Hypercholesterolemia, although not as common as hypertension or smoking, was found to be associated with more severe, bilateral atherosclerotic involvement of the vertebral and subclavian arteries. These results are congruent with those of others that have identified hypertension and smoking as the most common associated comorbid conditions.[1,14] Although it is unclear whether the symptoms of vertebrobasilar territory ischemia are the result of thromboembolic events or whether they occur because of hypoperfusion,[15] analysis of microemboli in the posterior circulation has revealed that the majority were the result of coexistent cardiac disease, with no correlation with disease of the vertebrobasilar system.[16] This result suggests that the majority of non–cardiac-related posterior-circulation ischemia symptoms are related to hemodynamic impairment, in contrast to carotid disease, in which emboli are the major source of anterior-circulation strokes.

The paucity of collateral flow in the patient population treated in the present study is underlined by the extent of bilateral vertebral or subclavian stenosis or occlusion and anterior-circulation disease. Accordingly, among the 8 patients treated who presented with subclavian steal, 4 had bilateral disease and 2 of the remaining 4 had developed an artery-to-artery embolus in the posterior circulation. This is an important characteristic, because the subclavian steal phenomenon in itself has previously been reported to be relatively benign and perhaps not to warrant aggressive surgical or interventional treatment when not symptomatic or not associated with hemodynamic spells.[3,17]

Although percutaneous balloon angioplasty alone has been used in the treatment of vertebral-origin stenosis, there has been increasing impetus to use intravascular stents because of the frequent observation of vessel recoil and restenosis after unsupported angioplasty,[4] as illustrated in Figure 5 (compare Figure 5G and 5H). Results from a number of studies evaluating the use of stents in the carotid artery have

suggested that it may be useful in a subset of patients with very high medical risk or in patients with tandem lesions or bilateral carotid stenosis.[18,19] Although surgical repair of the vertebral artery origin and subclavian artery has been performed with excellent results by centers with large experience, it is technically more difficult and has less well defined outcomes in the patient cohort described in the present study, namely, patients with poor collateral flow with a high proportion of bilateral vertebral/subclavian and coexistent carotid disease.[14,20–22] Surgical treatment has consisted of either ostial vertebral endarterectomy, subclavian endarterectomy, or reimplantation of the vertebral artery in the subclavian or carotid artery. In their series of 325 lesions treated surgically in 290 patients, Thevenet and Ruotolo[20] described a low mortality of only 0.6%. Only 13.8% of lesions in that series were bilateral, compared with 76.2% in our patient population. Furthermore, surgical repair was associated with a relatively high rate of postoperative Horner's syndrome (28%), a complication we did not encounter. At long-term follow-up, 68% of patients were asymptomatic, compared with 57% in our group. In the case of subclavian stenosis, surgical bypass procedures and carotid-subclavian bypass grafting have been surgically offered. Although they have been reported to have good long-term outcome, they are associated with a relatively high rate of perioperative morbidity, ranging up to 18%.[14,21,22]

Conclusions

The results in this clinical retrospective series, the largest to date, point to endovascular balloon angioplasty and stent placement as a useful technique for the treatment of vertebrobasilar territory ischemia in patients who have failed best medical therapy. Although long-term angiographic follow-up will be needed to assess the durability of stent patency, the clinical outcome in this patient group, with a high proportion of contralateral and anterior-circulation disease, suggests a sustained benefit of the procedure.

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Acknowledgments

We would like to thank Drs W. Smith, C. Johnston, and D. Bonovitch for their assistance in clinical management of the patients in this series.

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