Favorable Early Outcome of Carotid Artery Stenting Without Protection Devices

László K. Sztriha, MD; Erika Vörös, MD, PhD; Katalin Sas, MD; Réka Szentgyörgyi, MD, PhD; Anna Pócsik, MD; Pál Barzó, MD, PhD; Péter Szikra, BSc; Attila Makai, MD; Alex Szólics; Péter Elek, MD; László Rudas, MD, PhD, DSc; László Vécsei MD, PhD, DSc

Background and Purpose—Protection devices are increasingly used in carotid artery stenting. However, no randomized trial has been conducted to evaluate the efficacy of such devices, and arguments have also been formulated against their routine use. We set out to investigate the complication rates associated with carotid artery stenting performed without protection devices. Applicability of covered stents in the carotid system was also evaluated.

Methods—Between January 2001 and July 2003, 245 consecutive patients (260 hemispheres) underwent carotid artery stenting. No protection devices were applied. Covered stents were implanted in 31 (12.1%) cases. The incidence of complications during the intervention and the subsequent 30-day follow-up period was recorded.

Results—The technical success rate was 98.8%. One postprocedural nonneurological death (0.4%) occurred. Neurological complications (inclusive of transient ischemic attacks) were observed in 14 cases (5.4%). The rate of major complications (death, major stroke, and myocardial infarction) was 1.6% among the symptomatic and 1.5% among the asymptomatic cases. The rate of minor strokes was 3.2% in the symptomatic and 1.5% in the asymptomatic group. Of the neurological complications, 64.3% occurred postprocedurally. No ipsilateral neurological complications were detected in the subgroup treated with covered stents.

Conclusions—Carotid artery stenting without protection devices appears to be safe. Most of the neurological complications could not have been prevented with protection devices, because they occurred after the intervention. The application of covered stents may reduce the rate of embolization-related complications in the periprocedural period. **(*Stroke*. 2004; 35:2862-2866.)**

Key Words: carotid arteries ■ embolism ■ protective devices ■ stents

Angioplasty with stent placement is increasingly used instead of endarterectomy for the treatment of carotid artery stenosis.[1] Although most of the randomized studies comparing endovascular treatment with endarterectomy are still in progress, there are encouraging results in favor of the endovascular approach.[2,3] Improvements in the expertise of the physicians performing the procedures, and developments in endovascular technology, have led to decreases in the rates of complications associated with carotid stenting.[1] The most important acute complications of carotid stenting are related to the distal embolization of particles generated during the procedure.[4,5] Although endovascular protection devices may reduce the extent of intraprocedural cerebral embolization,[6,7] their application may also result in additional complications.[8,9] Recent studies[8,10] have focused mainly on the low rates of complications associated with procedures in which protection devices are used, and less attention has been paid to other means of reducing embolization. Some studies did not report the timing of the complications relative to the intervention[1] or failed to indicate complication rates separately for symptomatic and asymptomatic or for high-risk and low-risk patients.[7] We studied the 30-day outcome of carotid artery stenting performed without the use of protection devices. To our knowledge, this is the first evaluation of the use of covered stents for carotid stenosis.

Subjects and Methods

Between January 2001 and July 2003, 245 consecutive patients were included in a single-center carotid artery stenting study. Patients were included if they had symptomatic or asymptomatic, 60% to 99% (according to the North American Symptomatic Carotid Endarterectomy Trial [NASCET][11] measurement method) stenosis of a carotid artery. Exclusion criteria were the occurrence of a stroke within 6 weeks, a previous major stroke within the territory of the stenotic artery with no useful recovery of function, the presence of visible thrombus at the carotid lesion site, carotid artery dissection, vessel narrowing caused by external compression by a tumor, a life

Received June 14, 2004; final revision received August 29, 2004; accepted September 6, 2004.

From the Departments of Neurology (L.K.S., K.S., L.V.), Radiology (E.V., R.S., A.P., P.S., A.S.), Neurosurgery (P.B., P.E.), and the Medical Intensive Care Unit (A.M., L.R.), Cardiology Center, Albert Szent-Györgyi Medical and Pharmaceutical Center, University of Szeged; and the Neurology Research Group of the Hungarian Academy of Sciences and University of Szeged (L.V.), Szeged, Hungary.

Correspondence to Dr Laszlo K. Sztriha, Department of Neurology, Albert Szent-Györgyi Medical and Pharmaceutical Center, University of Szeged, Semmelweis u. 6, H-6725 Szeged, Hungary. E-mail sztriha@nepsy.szote.u-szeged.hu

Stroke **is available at http://www.strokeaha.org** **DOI: 10.1161/01.STR.0000147714.19871.45**

TABLE 1. Characteristics of Patients and Carotid Lesions

Patients	
Population	245 (100%)
Male	140 (57.1%)
Age	
Mean±SD, y	65.0±9.4
Range, y	33–89
70–79 y	80 (32.7%)
80–89 y*	9 (3.7%)
Severe cardiac dysfunction*	8 (3.3%)
Severe pulmonary dysfunction*	3 (1.2%)
Lesions	
Count	260 (100%)
Symptomatic	124 (47.7%)
Pathomechanism of stenosis	
Primary atherosclerotic	249 (95.8%)
Postendarterectomy restenosis*	11 (4.2%)
Fibromuscular disease	0 (0%)
Cervical radiotherapy*	0 (0%)
Degree of stenosis†	
Mean±SD, %	83.2±8.9
≥90%	111 (42.7%)
Contralateral carotid occlusion*	33 (12.7%)
Plaque morphology by angiography	
Smooth	58 (22.3%)
Irregular/ulcerated	202 (77.7%)

*Criteria for high-risk cases.
†As measured with the NASCET method.

expectancy of <2 years due to a known preexisting condition, and the inability or unwillingness of the patient to provide informed consent. Patient evaluation, intervention, and follow-up were carried out by a team of radiologists, neurologists, and neurosurgeons in accordance with a standardized protocol. The patients gave their written informed consent to the procedures, which followed institutional guidelines.

Patient Evaluation

The relevant medical history was taken and a thorough neurological examination was performed on all patients. Patients demonstrating repetitive transient ischemic attacks referable to an ipsilateral carotid stenosis were treated at the earliest opportunity. Duplex ultrasonography of the carotid arteries in all cases was used to reveal hemodynamically significant stenosis. Stenoses were considered significant if visible plaque and luminal narrowing were seen and the peak systolic velocity in the internal carotid artery exceeded 175 cm/s. Magnetic resonance angiography of the carotid arteries was performed in 21 patients (8.6%), and computed tomographic (CT) angiography in 5 cases (2.0%) because of uncertainty as to the presence of significant stenosis on ultrasonographic evaluation, due to plaque calcification or vessel tortuosity. CT or MRI of the brain was carried out in all cases. Criteria for patients at high risk included age≥80 years old, contralateral carotid occlusion, postendarterectomy restenosis, cervical radiation treatment, severe cardiac dysfunction (New York Heart Association class III/IV chronic heart failure, acute myocardial infarction within 4 weeks, unstable angina, or a coronary procedure within 4 weeks), and pulmonary disease causing considerable functional limitation. Patient and lesion characteristics are shown in Table 1.

Stenting Protocol

Treatment with aspirin 100 mg plus clopidogrel 75 mg or ticlopidine 2×250 mg daily was started at least 4 days before the procedure and continued for a minimum of 4 weeks postprocedure. If this combined antiplatelet treatment had not been given before stenting, 100 mg aspirin and 300 mg clopidogrel were administered on the day of the intervention. All stenting procedures were performed under local anesthesia by the same neuroradiologist, with experience in >4000 diagnostic cerebral angiographies and 15 proctored carotid stenting procedures before this study. Percutaneous access was gained through the femoral artery. Brachiocephalic angiography with intracranial views and assessment of the collateral cerebral circulation always preceded the stenting. The extent of the carotid artery stenosis was measured with the NASCET[11] method, and the plaque surface morphology was noted (Table 1). All lesions found noninvasively to be hemodynamically significant proved to be ≥60% with the NASCET method. The final percentage stenosis was based on the angiographic findings. Carotid artery stenting was achieved by a standard technique[12] with low-profile devices and gentle manipulation. Heparin 5000 IU was given intraarterially 1 to 2× during the intervention. No protection devices were used. The following appropriately-sized self-expandable stents were implanted during the 257 successful procedures: 207 (80.5%) Carotid Wallstents (Boston Scientific), 31 (12.1%) Symbiot covered stents (Boston Scientific), 18 (7.0%) Precise stents (Cordis), and 1 (0.4%) Smart stent (Cordis). Predilatation was applied in 14 (5.4%) and postdilatation in 245 (95.3%) cases. Stent overdilatation was avoided. The residual stenosis in all successfully treated vessels was <30%. Covered stents were used at the discretion of the interventionalist. The covered stent (Symbiot) applied features a self-expanding nitinol stent encased in a thin porous polytetrafluoroethylene membrane. No such stent was applied in 2001, 7 were implanted in 2002, and 24 were used from January to July 2003. The vital signs were recorded regularly, the cardiac rhythm was monitored continuously, and neurological assessment was frequent during the intervention. Intravenous atropine, up to 2 mg, was administered as necessary for bradycardia. Control angiograms were recorded on procedure completion to evaluate recanalization and to exclude embolization into intracranial vessels. Three patients whose stenting failed technically subsequently underwent carotid endarterectomy.

Patient Follow-Up

Control neurological examination was performed routinely 24 hours and 30 days after stenting. If a patient exhibited a neurological deterioration, brain CT and control angiography (in the event of intraprocedural complications) or carotid ultrasonography were conducted without delay. Heart rate and blood pressure were checked regularly in the postinterventional period. Carotid duplex ultrasonography was carried out routinely 4 weeks postprocedure. The incidence of complications during the intervention and the subsequent 30-day follow-up period was recorded. A transient ischemic attack was defined as a focal retinal or hemispheric event from which the patient made a complete recovery within 24 hours. Minor stroke was identified as a new neurological deficit that either resolved completely within 30 days or increased the National Institutes of Health Stroke Scale score by ≤3. Major stroke was defined as a new neurological deficit that persisted after 30 days and increased the National Institutes of Health Stroke Scale score by ≥4. Complications were considered "intraprocedural" if they occurred between the attainment of femoral arterial access and successful vascular access site hemostasis. Complications arising at any time up to 30 days after this period were regarded as "postprocedural." Eight patients (3.3%) missed the 30-day evaluation visit but presented later. For the 6 patients (2.4%) who did not attend for control, follow-up information was obtained from their general practitioners.

Statistical Analysis

Proportions were compared by using χ^2 or Fisher exact tests, as appropriate. Two-sided P values are reported. $P<0.05$ was considered significant.

TABLE 2. Complications Within 30 Days

Category	Symptomatic (n=124)		Asymptomatic (n=136)		Overall (n=260)	
	no.	%	no.	%	no.	%
Death	0	0.0	1	0.7	1	0.4
Neurological complications						
Major strokes	2	1.6	1	0.7	3	1.2
Minor strokes	4	3.2	2	1.5	6	2.3
All ipsilateral strokes	5	4.0	3	2.2	8	3.1
TIAs	4	3.2	1	0.7	5	1.9
Other complications						
Myocardial infarction					0	0.0
Angina					1	0.4
Severe hypotension					1	0.4
Procedure-related bleeding					1	0.4
Stent occlusion					2	0.8
Restenosis					0	0.0
Femoral AV fistula					1	0.4

TIAs indicates transient ischemic attacks.

Results

Of the 260 arteries in the 245 patients, 257 in 242 patients were treated successfully (technical success rate: 98.8%). The procedure failed because of extreme vessel tortuosity in 2 cases and because of the inability to guide the stent through a calcified subtotal occlusion in 1 patient. Sixty procedures (23.1%) were conducted on high-risk patients, 37 in symptomatic (29.8%) cases, and 23 in asymptomatic (16.9%) cases.

The complications observed during the procedure and the 30-day follow-up are shown in Tables 2 and 3. The 1 postprocedural nonneurological death (0.4%), in a high-risk patient, occurred because of hemorrhagic shock resulting from uncontrollable bleeding of a previously unknown adenocarcinoma of the sigmoid colon. Neurological complications arose in 14 cases (5.4%; 95% CI, 2.6 to 8.1%). Myocardial infarction was not detected. The rate of major complications (death, major stroke, and myocardial infarction) was 1.6% among the symptomatic (low-risk, 1.1%; high-risk, 2.7%; $P=0.51$) and 1.5% among the asymptomatic (low-risk, 0%; high-risk, 8.7%; $P=0.03$) cases ($P=1.0$). The rate of minor strokes was 3.2% in the symptomatic (low-risk, 3.4%; high-risk, 2.7%; $P=1.0$) and 1.5% in the asymptomatic (low-risk, 1.8%; high-risk, 0%; $P=1.0$) group ($P=0.43$). The rate of major complications was 0.5% in the low-risk and 5.0% in the high-risk group ($P=0.04$). The rate of minor strokes was 2.5% and 1.7% in the low- and high-risk populations, respectively ($P=1.0$).

Of the 14 neurological complications, 5 (35.7%) occurred intraprocedurally and the remainder after completion of the intervention. Thirteen (92.9%) neurological complications developed ipsilaterally to the stented carotid artery, 8 in cases with ≥90% stenosis (7.2%) and 5 in those with <90% stenosis (3.4%, $P=0.16$). The rate of ipsilateral neurological complications among the patients with irregular or ulcerated plaques was 4.5% (9 of 202), whereas among those with smooth lesions it was 6.9% (4 of 58; $P=0.50$). All strokes were ischemic. One major stroke occurred in a patient (No. 12, Table 3) who took only aspirin after discharge. Stent occlusion was diagnosed in 2 patients within the 30-day follow-up; one of them (No. 3, Table 3) experienced a major stroke during coronary artery bypass surgery performed under anticoagulant but not combined antiplatelet treatment; the other remained symptom-free. Restenosis was not observed. One patient experienced severe hypotension requiring intensive care. Blood transfusion was necessary in 1 case because of a considerable blood loss from the femoral puncture site.

Of the 31 covered stents, 14 (45.2%) were implanted in symptomatic cases; 23 (74.2%) were used for irregular/

TABLE 3. Neurological Complications Within 30 Days

No.	Age, y	Sex	Risk	Stenosis Type	Plaque	Category	Side*	Timing
1	55	M	H	Sympt	Irregular	Minor stroke	Ipsilateral	Postprocedural
2	57	F	H	Sympt	Smooth	TIA	Ipsilateral	Intraprocedural
3	63	F	H	Asympt	Ulcerated	Major stroke	Ipsilateral	Postprocedural
4	65	M	L	Sympt	Irregular	TIA	Ipsilateral	Postprocedural
5	66	F	L	Sympt	Irregular	TIA	Ipsilateral	Intraprocedural
6	68	F	L	Asympt	Smooth	TIA	Ipsilateral	Intraprocedural
7	69	M	L	Sympt	Irregular	Minor stroke	Ipsilateral	Postprocedural
8	71	F	L	Sympt	Irregular	Minor stroke	Ipsilateral	Postprocedural
9	72	M	L	Sympt	Irregular	Minor stroke	Contralateral	Postprocedural
10	72	M	L	Sympt	Smooth	TIA	Ipsilateral	Postprocedural
11	74	M	L	Asympt	Smooth	Minor stroke	Ipsilateral	Intraprocedural
12	78	F	L	Sympt	Irregular	Major stroke	Ipsilateral	Postprocedural
13	79	M	L	Asympt	Irregular	Minor stroke	Ipsilateral	Postprocedural
14	82	M	H	Sympt	Irregular	Major stroke	Ipsilateral	Intraprocedural

M indicates male; F, female; H, high-risk; L, low-risk; asympt, asymptomatic stenosis; sympt, symptomatic stenosis; TIA, transient ischemic attack.

*Indicates whether the complication developed ipsi- or contralaterally to the stented carotid artery.

ulcerated stenoses, and 8 (25.8%) were used in high-risk cases. No ipsilateral neurological complications developed in the patients receiving covered stents, as opposed to the 5.8% complication rate (13 ipsilateral neurological symptoms with 226 treated vessels) with regular stents ($P=0.38$). No technical difficulties were experienced with the use of covered stents.

The rate of neurological complications was 2.0% (1 during 49 procedures) in 2001, 5.9% (6 of 102) in 2002, and 6.4% (7 of 109) from January to July 2003.

Discussion

The rate of all strokes and death, 4.8% among the symptomatic and 2.9% among the asymptomatic cases in our study, involving low- and high-risk patients compares favorably with the results obtained in the low-risk populations undergoing endarterectomy in the NASCET[11] and Asymptomatic Carotid Atherosclerosis Study (ACAS)[13] investigations in which the rate of all strokes and death was 5.8% for symptomatic and 2.3% for asymptomatic patients, respectively.

The release of microemboli composed of thrombotic and plaque substances into the cerebral circulation is considered to be the main cause of the early complications associated with stenting and also with endarterectomy. The phases with increased microembolic signals at stenting as detected by transcranial Doppler include predilatation, stent deployment, and postdilatation.[6] In an attempt to reduce periprocedural complication rates during stenting, cerebral protection devices were developed, those most commonly used being distal filters and occlusive distal balloons. A recent study[10] of 815 stenting procedures with cerebral protection devices revealed a 30-day stroke and death rate of 3.8% for symptomatic and 3.2% for asymptomatic lesions. A review[1] of the global carotid artery stent registry by Wholey et al demonstrated a 2.23% rate of strokes and procedure-related deaths in 4221 cases undergoing protected carotid stenting, whereas the corresponding rate among the 6753 cases treated without protection was 5.29%. A systematic review[7] of the early outcome of carotid angioplasty and stenting in both symptomatic and asymptomatic patients found that the combined stroke and death rate with cerebral protection was 1.8%, that is, significantly lower than the 5.5% in those treated without protection devices. However, in addition to causing increases in the intervention time, the technical complexity and the cost, the application of protection devices may also lead to complications, including hemodynamic intolerance due to balloon occlusion or congested nets, spasm or dissection of the carotid artery, and difficulties with retrieval of the device.[5,8,9] Furthermore, predilatation, which is often necessary in protected stenting, and the removal of the protection devices may result in embolization.[9] Not all particles may be removed from behind the balloon occlusion–type device, and small particles (even though probably clinically insignificant) may pass through the filter systems. Protection devices do not prevent the late embolization of particles trapped in the stent meshes. The Stenting and Angioplasty With Protection in Patients at High Risk for Endarterectomy (SAPPHIRE) study[3] found a 4.4% overall perioperative stroke and death rate of stenting with protection devices in high-risk cases, whereas in the asymptomatic subgroup the rate was as high as 5.8%.[14]

Our results indicate that postprocedural embolization may be responsible for a significant proportion of the complications. Neurological complications could not have been prevented with a protection system in 9 (64.3%) cases in our series, because these were not intraprocedural complications. Similarly, in a study[15] of 111 patients stented without protection devices, 10 (71.4%) of the 14 neurological complications occurred postprocedure. The low intraprocedural complication rate in our study may be related to several factors. Low-profile devices were used, and predilatation of the lesions was avoided whenever possible. The stents were not overexpanded during postdilatation. As far as we are aware, this is the first published report on the application of (among others) covered Symbiot stents for carotid stenosis. Covered stents prevent the passage of atherosclerotic material through the stent mesh. In order not to obstruct the flow into the external carotid artery and also because of their maximum diameter of 6 mm, these stents were used for lesions involving only the internal carotid artery and not extending into the common carotid artery. As a learning curve effect was not seen in our series, the low complication rate observed with covered stents is unlikely to be because of their more frequent application toward the end of the study period. Our results indicate that covered stents may safely and efficiently reduce neurological complications due to embolizations during stent deployment and postdilatation and also postprocedurally. Their utilization may additionally decrease the restenosis rate. Further studies are expected to provide more data on the use of covered stents for carotid stenosis, although large case numbers would be necessary to demonstrate statistically significant differences in complication rates.

Concerning postprocedural complications, adherence to the combined antiplatelet regimen appears important. In 2 cases of postprocedual major strokes among our patients, the combined antiplatelet medication had been interrupted. A previous stenting study,[16] in which solely aspirin was administered, reported high complication rates. The combination of aspirin with clopidogrel, as compared with aspirin alone, appears to reduce the thromboembolic potential (as measured by transcranial Doppler) after carotid endarterectomy.[17] This may apply to carotid stenting, too. The results of our study can be generalized to patients with atherosclerotic stenosis of the carotid artery. To provide the best possible treatment for the patients, collaboration within a multidisciplinary consultation team is necessary during the patient evaluation, intervention, and follow-up.

There are some limitations to our study. Transcranial Doppler studies or brain MRI examinations with diffusion-weighted sequences were not performed to detect cerebral embolization. It has been documented[4] that a majority of the new lesions seen on postintervention MRI images are not detected at neurological examination. Patients with a history of stroke were treated at least 6 weeks after the event, which may have resulted in a lower rate of complications as compared with an earlier intervention. Although patients who had failed to attend the 30-day check-up were reported to be

symptom-free, the occurrence of minor neurological complications cannot be fully excluded.

No randomized study has as yet been conducted to investigate the efficacy of protection devices. Our study indicates that carotid artery stenting without protection devices can be performed within the complication rate limits (perioperative stroke and death <6% for symptomatic and <3% for asymptomatic patients) recommended by the American Heart Association for carotid endarterectomy[18] and that complication rates can be achieved that are similar to those observed with protection devices. We propose that the application of covered stents may reduce embolization. Subgroup analysis of the endarterectomy versus carotid stenting trials currently under way is expected to provide data on the value of protection devices. Developments in endovascular technology, pharmacological management and expertise should lead to further reductions in the complication rates associated with carotid artery stenting.

References

1. Wholey MH, Al-Mubarek N, Wholey MH. Updated review of the global carotid artery stent registry. *Catheter Cardiovasc Interv*. 2003;60:259–266.
2. CARESS Steering Committee. Carotid revascularization using endarterectomy or stenting systems (CARESS): phase I clinical trial. *J Endovasc Ther.* 2003;10:1021–1030.
3. Yadav J; for the SAPPHIRE investigators. Stenting and angioplasty with protection in patients at high risk for endarterectomy: the SAPPHIRE study. Presented at: American Heart Association Scientific Session; November 2002; Chicago, IL.
4. van Heesewijk HP, Vos JA, Louwerse ES, Van Den Berg JC, Overtoom TT, Ernst SM, Mauser HW, Moll FL, Ackerstaff RG; Carotid PTA and Stenting Collaborative Research Group. New brain lesions at MR imaging after carotid angioplasty and stent placement. *Radiology.* 2002;224:361–365.
5. Reimers B, Corvaja N, Moshiri S, Sacca S, Albiero R, Di Mario C, Pascotto P, Colombo A. Cerebral protection with filter devices during carotid artery stenting. *Circulation*. 2001;104:12–15.
6. Al-Mubarak N, Roubin GS, Vitek JJ, Iyer SS, New G, Leon MB. Effect of the distal-balloon protection system on microembolization during carotid stenting. *Circulation*. 2001;104:1999–2002.
7. Kastrup A, Groschel K, Krapf H, Brehm BR, Dichgans J, Schulz JB. Early outcome of carotid angioplasty and stenting with and without cerebral protection devices: a systematic review of the literature. *Stroke*. 2003;34:813–819.
8. Cremonesi A, Manetti R, Setacci F, Setacci C, Castriota F. Protected carotid stenting: clinical advantages and complications of embolic protection devices in 442 consecutive patients. *Stroke*. 2003;34:1936–1941.
9. Eckert B, Zeumer H. Editorial comment–Carotid artery stenting with or without protection devices? Strong opinions, poor evidence! Stroke. 2003;34:1941–1943.
10. Reimers B, Schluter M, Castriota F, Tubler T, Corvaja N, Cernetti C, Manetti R, Picciolo A, Liistro F, Di Mario C, Cremonesi A, Schofer J, Colombo A. Routine use of cerebral protection during carotid artery stenting: results of a multicenter registry of 753 patients. *Am J Med.* 2004;116:217–222.
11. North American Symptomatic Carotid Endarterectomy Trial Collaborators. Beneficial effect of carotid endarterectomy in symptomatic patients with high-grade carotid stenosis. *N Engl J Med.* 2001;325:445–453.
12. Vitek JJ, Roubin GS, Al-Mubarek N, New G, Iyer SS. Carotid artery stenting: technical considerations. *Am J Neuroradiol*. 2000;21:1736–1743.
13. Executive Committee for the Asymptomatic Carotid Atherosclerosis Study. Endarterectomy for asymptomatic carotid artery stenosis. *JAMA*. 1995;273:1421–1428.
14. Barr JD, Connors JJ 3rd, Sacks D, Wojak JC, Becker GJ, Cardella JF, Chopko B, Dion JE, Fox AJ, Higashida RT, Hurst RW, Lewis CA, Matalon TA, Nesbit GM, Pollock JA, Russell EJ, Seidenwurm DJ, Wallace RC; American Society of Interventional and Therapeutic Neuroradiology; American Society of Neuroradiology; Society of Interventional Radiology. Quality improvement guidelines for the performance of cervical carotid angioplasty and stent placement. *J Vasc Interv Radiol.* 2003;14:1079–1093.
15. Qureshi AI, Luft AR, Janardhan V, Suri MF, Sharma M, Lanzino G, Wakhloo AK, Guterman LR, Hopkins LN. Identification of patients at risk for periprocedural neurological deficits associated with carotid angioplasty and stenting. *Stroke*. 2000;31:376–382.
16. Diethrich EB, Ndiaye M, Reid DB. Stenting in the carotid artery: initial experience in 110 patients. *J Endovasc Surg*. 1996;3:42–62.
17. Payne DA, Jones CI, Hayes PD, Thompson MM, London NJ, Bell PR, Goodall AH, Naylor AR. Beneficial effects of clopidogrel combined with aspirin in reducing cerebral emboli in patients undergoing carotid endarterectomy. *Circulation*. 2004;109:1476–1481.
18. Biller J, Feinberg WM, Castaldo JE, Whittemore AD, Harbaugh RE, Dempsey RJ, Caplan LR, Kresowik TF, Matchar DB, Toole J, Easton JD, Adams HP Jr., Brass LM, Hobson RW 2nd, Brott TG, Sternau L. Guidelines for carotid endarterectomy: a statement for healthcare professionals from a Special Writing Group of the Stroke Council, American Heart Association. *Stroke*. 1998;29:554–562.

Favorable Early Outcome of Carotid Artery Stenting Without Protection Devices

László K. Sztriha, Erika Vörös, Katalin Sas, Réka Szentgyörgyi, Anna Pócsik, Pál Barzó, Péter Szikra, Attila Makai, Alex Szólics, Péter Elek, László Rudas and László Vécsei

Stroke. 2004;35:2862-2866; originally published online October 28, 2004;
doi: 10.1161/01.STR.0000147714.19871.45
Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231

Print ISSN: 0039-2499. Online ISSN: 1524-4628