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Prospective Analysis of Cerebral Infarction After Carotid Endarterectomy and Carotid Artery Stent Placement by Using Diffusion-Weighted Imaging

Hong Gee Roh, Hong Sik Byun, Jae Wook Ryoo, Dong Gyu Na, Won-Jin Moon, Byung Boong Lee, and Dong-Ik Kim

BACKGROUND AND PURPOSE: Small emboli arising from a friable plaque during carotid endarterectomy (CEA) and carotid artery stent placement (CAS) constitute a potentially important cause of periprocedural ischemic complications. To evaluate the frequency and significance of cerebral ischemic lesions of embolic origin after CEA and CAS, we examined patients with moderate to severe carotid stenosis by using diffusion-weighted (DW) imaging.

METHODS: Twenty-four patients undergoing 26 CEAs and 20 patients undergoing 22 CAS were prospectively studied with pre- and post-treatment DW imaging of the brain within 7 days (mean, 2.3 days) before and within 7 days (mean, 3.2 days) after treatment. DW images were analyzed by two neuroradiologists blinded to the clinical results after CEA and CAS. Any new hyperintense lesion on DW image was interpreted as a post-treatment ischemic lesion. We compared post-treatment ischemic lesions with change in neurologic status, presence of plaque ulcerations, and severity of stenosis and compared the frequency of overall post-treatment complications between the two procedures.

RESULTS: In 25 (96%) of 26 CEAs, post-operative brain DW images were unchanged. In one patient (4%), a new single asymptomatic hyperintensity was observed in the striatocapsule on the surgical side. In 14 (64%) of 22 CAS procedures, post-operative brain DW images were unchanged. In eight CAS cases (36%), new hyperintensities were seen on DW images. Among them, two were symptomatic with a major neurologic deficit lasting more than 7 days. Post-treatment brain or retinal major stroke rates were 4% ($n = 1$) for CEA and 14% ($n = 3$) for CAS. Overall symptomatic complication rates were 19.2% and 13.6%, respectively.

CONCLUSION: Rate of ischemic brain lesions was significantly lower after CEA than after CAS, although most of these brain lesions were silent. Also, CEA is a safer procedure carrying a lower risk of post-operative cerebral ischemia. CAS, however, may be a comparable procedure considering the total complication rate and can be a more reliable procedure with advances in neuroprotective means.

Although carotid endarterectomy (CEA) and carotid artery stent placement (CAS) are performed to re-

duce the risk of cerebral ischemia, these procedures have been known to cause temporary or permanent neurologic deficits (1–6). Therefore, low to minimum occurrence of periprocedural neurologic complications and death became the critical issue to both CEA and CAS procedures. Strict criteria for patient selection and meticulous pre- and post-treatment care are mandated to achieve low complication rates.

CEA is now fully accepted as the standard procedure following thorough investigations for its safety based on many retrospective and prospective studies through the past decades. Its clinical application has steadily increased with a low complication rate, and it remains the only revascularization procedure with proved efficacy in symptomatic and asymptomatic ca-

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rotid artery stenosis (7–13). CAS is currently being investigated as an alternative treatment to CEA, and many recent studies suggest that CAS is also effective in the treatment of carotid artery stenosis (3, 4, 11, 14–21).

Diffusion-weighted (DW) imaging is highly sensitive and specific in the diagnosis of ischemic stroke (22–24). Ischemic stroke may occur as a complication of any vascular intervention from the heart to the head. DW imaging is being used in many interventional and surgical procedures as a marker of ischemic complications (1, 2, 5, 22, 25–29).

In the present prospective study, our principal objective was to evaluate the frequency and significance of ischemic brain lesions of embolic origin after CEA and CAS by using DW imaging and to compare imaging findings with post-treatment neurologic status, angiographic morphology of the atheromatous plaque, and severity of stenosis.

Methods

Patient Selection and Data Collection

Forty-eight consecutive procedures were performed in a total of 44 patients (42 men and two women) with moderate or severe ($>40\%$) carotid artery stenosis. Twenty-six CEA and 22 CAS procedures were performed from July 1998 through February 2001 (two patients underwent bilateral CEA and two patients underwent bilateral CAS). CEA was performed by two experienced vascular surgeons: one (B.B.L.) had extensive experience for more than 2 decades in the United States with an American-trained and American board-certified background to be specialized in carotid surgery before establishment of Korea-first Carotid Clinic for stroke prevention; the other was an associate (D-I.K.) who was trained in Korea by the first vascular surgeon. CAS was performed by one experienced (more than 30 cases) interventional neuroradiologist (H.S.B.).

The patients were not randomized to two treatment groups. The appropriate treatment technique was selected based on consensus among the multidisciplinary team members of the Carotid Clinic, which included interventional neuroradiologists (H.G.R., H.S.B., J.W.R., D.G.N., W.J.M.), a vascular surgeon (B.B.L., D-I.K.), cardiologist, and neurologist. Priority was given to CAS for the patients who were considered to be at high surgical risk (i.e., high carotid bifurcation, unstable cardiopulmonary disease, bilateral severe carotid disease) following the full disclosure of the investigational nature of the CAS procedure to the patient as a clinical option to replace CEA. However, CAS was not implemented as a treatment option in patients with a severely tortuous aorta or floating thrombus, although the indications for CAS in general were more liberal than those for CEA.

No patient had an accompanying intracranial lesion that was more significant than the proximal carotid artery stenosis. The degree of stenosis was determined according to the North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria: minimum residual lumen at the point of maximum stenosis referenced to the diameter of the distal lumen of the internal carotid artery (ICA) at the first point at which the arterial walls became parallel, percentage of stenosis = $100 [1 - (\text{minimal residual lumen}/\text{distal lumen})]$ (7, 8, 10, 11). The following information was collected for each patient: age, sex, initial symptoms, stenosis degree of the ipsilateral and contralateral ICAs, and angiographic surface morphology. We also recorded pre- and post-treatment DW imaging findings and any new or worsening neurologic and non-neurologic symptoms occurring within 7 days of the procedure. This study was approved by the institutional review board, and written informed consent was obtained from all patients.

Protocol for CEA

All CEA surgical procedures were performed with the patient under general anesthesia. Transcranial Doppler sonography was added to electroencephalography to provide continuous intraoperative monitoring of brain perfusion and detection of microembolic signals during the procedure. Intravenous heparin (100 U/kg) was routinely administered before the ICA was cross-clamped and converted with protamine following the procedure. To avoid particulate emboli during the surgical procedure, extreme care was given especially during the exposure of the ICA and common carotid artery (CCA). Arteriotomy was fully extended high above the upper extent of the lesion or stenosis to be removed. Intraoperative shunt surgery was performed to maintain the cerebral blood flow during the cross-clamp period, especially when the blood flow velocity in the ipsilateral middle cerebral artery (MCA) was decreased to more than 50% of preclamping values. The shunt was introduced, with extreme care, distally into the ICA first and then proximally into the CCA after arteriotomy. Careful flushing through the shunt was performed to remove all potential source of particulate fragments before shunt surgery was established. After meticulous removal of the plaque, the endarterectomy wound along the carotid bifurcation area was thoroughly inspected to remove all the residual loose fragments of the plaque from the surface of the endarterectomized vessel wall. The arteriotomy was closed with a running 6–0 Prolene suture with or without a patch. Aspirin (100 mg/day) was routinely administered after surgery. The excised plaques were examined macroscopically after surgery to classify them as smooth or ulcerated plaque, depending on the absence or presence of ulcerations and intramural thrombi.

Protocol for CAS

Ticlopidine (250 mg twice/day) and aspirin (150 mg/day) were administered orally at least 48 hours before CAS. Vascular access was gained by placement of a 9F sheath in the common femoral artery. A 6F sheath was placed in the femoral vein for a temporary cardiac pacemaker. Heparin (usually 80 IU/kg) was administered intravenously to achieve an activated clotting time of 2–3 times greater than normal. With use of a hydrophilic guidewire (0.038- or 0.035-inch Terumo) with an exchange length (260 or 300 cm), the 5F neuroangiographic catheter was advanced into the ipsilateral external carotid artery. The 5F catheter was withdrawn, and the 7F, 90-cm guiding sheath (Shuttle Flexor; Cook, Inc., Bloomington, IN) was then advanced in the CCA over the exchange wire. For very tortuous vessels, a 0.038-inch extra-support Amplatz wire (Boston Scientific/Medi-Tech, Newton, MA) was used with considerable success. After positioning the tip of the 7F guiding catheter 3–4 cm below the carotid bifurcation, a baseline carotid angiogram was obtained, and the interventional neuroradiologist estimated the length and severity of the lesion and the length and size of the stent required. The critical measurement was the diameter of the CCA just proximal to the bifurcation, which was used to size the Easy Wallstent (Boston Scientific/Schneider, Minneapolis, MN) to be deployed. In most cases, the stenotic lesion was crossed with a 0.038- or 0.035-inch Terumo wire without great difficulty. In one difficult case, a microcatheter and 0.014-inch wire were used. The stenotic lesion was predilated with a 4-mm diameter angioplasty balloon (Ultra-thin Diamond, Boston Scientific/Medi-Tech, Newton, MA) except in three cases with moderate stenosis and two cases with severe stenosis. After this, or as a primary procedure, a self-expanding Easy Wallstent was positioned across the lesion and deployed. Once the stent was deployed, the lesion was dilated with an appropriately sized balloon (usually 5- or 6-mm diameter, Ultra-thin Diamond) to permit full expansion of the stent within the ICA and CCA. Up to two balloon inflations—no more than 10 seconds each—were permitted. After angioplasty

and stent deployment, a completion angiographic examination was performed.

MR Imaging Examination

Pretreatment MR imaging was performed as close to treatment as possible within 7 days (mean, 2.3 days [CEA, 2.4 days; CAS, 2.2 days]; range 0–7 days), with the post-treatment imaging performed a mean of 3.1 days (CEA, 3.3 days; CAS, 2.9 days) after the procedure (range 1–7 days). There was no specific difference in time to MR imaging between the two groups. All preoperative MR imaging in the CEA group was performed after transfemoral carotid arteriography for exclusion of angiography-related infarction. Imaging was performed with a 1.5-T apparatus (Signa Horizon or Signa CV/I; GE Medical Systems, Milwaukee, WI) equipped with a head coil. The pre- and post-treatment MR imaging routinely included the following: axial spin-echo T1-weighted, fast spin-echo T2-weighted, fluid-attenuated inversion-recovery (FLAIR), DW, perfusion-weighted, and post-contrast spin-echo T1-weighted imaging. The DW images were acquired with an echo-planar sequence. An isotropic sequence was used (6500/97/1 TR/TE/NEX, field of view 280 mm, matrix 128 × 128), with b values of 0 and 1000 s/mm². The DW images were then evaluated by two neuroradiologists (H.G.R., H.S.B.) blinded to the clinical status of the patients. Any presence of new hyperintensity in the brain was interpreted as a sign of new ischemic lesions after CEA or CAS.

Statistical Analysis

The new hyperintensities on DW images and overall complications of the patients in the CEA and CAS groups were compared by using the Fisher exact test. Significance was defined as $P < .05$.

Results

Recanalization of ICA stenosis by CEA or CAS was technically successful in all 48 procedures. There was no death associated with the procedures. The mean age of the patients at the time of the procedures was 63.4 ± 8.5 years (range, 37–80 years). Of the 48 carotid artery stenoses cases, 37 (77%) were symptomatic lesions (eight with transient ischemic attacks or amaurosis fugax and 29 with cerebral or retinal infarcts) and 11 (23%) were asymptomatic. The contralateral ICA was occluded in nine (19%) of 48 procedures and severely stenotic in six (12%). Table 1 shows the baseline demographics and clinical characteristics of the two treatment groups. Table 2 shows neurologic and other non-neurologic complications after treatment.

CEA Series

In 25 (96%) of 26 CEA procedures, post-operative brain DW images were unchanged. A new, small, asymptomatic hyperintense signal was observed in only one patient (4%), in the striatocapsule of the surgical side (Fig 1). This patient had ipsilateral moderate stenosis (46%) of the right ICA with plaque ulceration and contralateral severe stenosis (70%) at the same time. There were five cases with nonischemic complications (19%). Four lower cranial nerve injuries, two temporary and another two permanent,

TABLE 1: Baseline characteristics of the treatment groups

Characteristic	CEA Procedures (n = 26)	CAS Procedures (n = 22)
Age (y), mean (range)	64.9 (48–77)	61.5 (37–80)
Sex, no (%)		
Male	22 (92)*	20 (100)*
Female	2 (8)	0 (0)
Presenting symptom, no. (%)		
Transient ischemic attack and/or amaurosis fugax	5 (19)	3 (14)
Cerebral and/or retinal infarction	14 (54)	15 (68)
Asymptomatic	7 (27)	4 (18)
Pretreatment carotid artery		
Ipsilateral stenosis, no. (%)		
Mean percentage stenosis $\pm$ SD	77.9 $\pm$ 13.2	76.3 $\pm$ 14.0
Moderate (40–70%)	7 (27)	8 (36)
Severe (>70%)	19 (73)	14 (64)
Ipsilateral plaque ulceration, no. (%)	21 (80)	18 (82)
Contralateral stenosis, no. (%)		
Mean percentage stenosis $\pm$ SD	20.4 $\pm$ 30.5	59.2 $\pm$ 42.1
None or <20% stenosis	17 (62)	6 (27)
Mild (20–40%)	4 (15)	1 (4)
Moderate (40–70%)	3 (12)	3 (14)
Severe (>70%)	2 (8)	4 (18)
Occlusion	1 (4)	8 (36)

* Two patients underwent bilateral CEA and two patients underwent bilateral CAS.

TABLE 2: Posttreatment results in the two groups

Posttreatment Result	CEA (n = 26)	CAS (n = 22)
Unchanged DW image	25 (96)	14 (64)
New hyperintensity on DW image	1 (4)	8 (36)
Symptomatic hyperintensity on DW image	0 of 1 (0)	2 of 8 (25)
Neurological and/or visual deficit	1 (4)	3 (14)
Nonischemic complication	5 (19)	0 (0)
Total symptomatic complications	5 (19)	3 (14)

Note.—Data are number (%) of procedures.

occurred in three patients. Also, two hyperperfusion syndromes developed. Of four cranial nerve paralyses, hypoglossal nerve injury was noted in one patient, vagus nerve injury in one patient, and both glossopharyngeal and hypoglossal nerve injuries in one patient. All three patients were assessed to have a high carotid bifurcation above C2 level preoperatively and subsequently required mandibular subluxation or dislocation to have adequate exposure to carry out the procedure properly. Of the two cases with hyperperfusion syndrome, one had only severe transient headache with no neurologic deficit and the other had significant neurologic deficit and seizure lasting more than 7 days with cerebral edema and hemorrhage (Fig 2).

CAS Series

In 14 (64%) of 22 CAS procedures, post-treatment brain DW images were unchanged. New, small hyperintense signals were observed in eight cases (36%). However, only two of them were symptomatic with

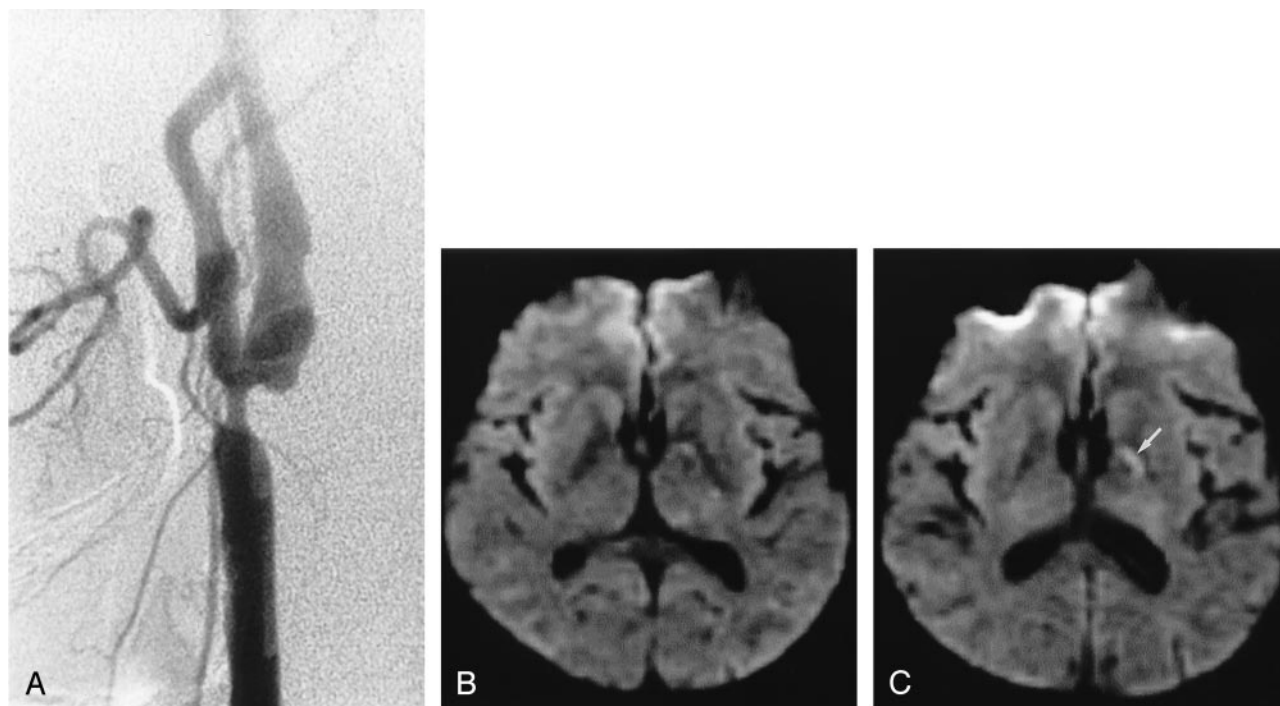


Fig 1. Case of a 65-year-old man with symptomatic moderate stenosis (49%) of the left carotid bifurcation.

A–C, Pre-CEA angiogram (A), pre-CEA DW image (B), and post-CEA DW image. Post-CEA DW image demonstrates a single, new, small hyperintensity (arrow) in the left striatocapsule near the small old lacune. This lesion was silent.

neurologic deficit lasting more than 7 days after CAS. All new lesions measured several mm in diameter, but were smaller than 1 cm. There was no evidence of major artery occlusion or territorial infarction. In one case, intraarterial thrombolysis of the central sulcal branch of the right MCA was performed with urokinase after CAS because the patient claimed left upper extremity weakness immediately after stent placement. DW images and findings at neurologic examination after CAS and thrombolysis were normal. There were two cases with visual field defect due to retinal infarction: one with symptomatic hyperintensities in the ipsilateral brain on DW images (Fig 3), the other with no new hyperintensity. So, the total number of cases with post-treatment neurologic or retinal deficit was three (14%). Non-neurologic complications were not observed in this series. There was no obvious trend in the frequency of ischemic lesions on DW images with regard to whether or not plaque ulceration and severe contralateral stenosis or occlusion were present. In the subgroup who underwent CAS without predilatation ($n = 5$), new hyperintense signals were observed in two cases (40%).

Comparison of Complications of CEA and CAS

Rates of post-treatment brain lesions on DW images were 4% and 36% in the CEA and CAS series, respectively. Frequency of ischemic brain lesions of embolic origin after CEA was significantly lower than that of CAS ($P = .005$). Post-treatment brain or retinal stroke rates were 4% ($n = 1$) and 14% ($n = 3$), and the overall symptomatic ischemic and nonisch-

emic complication rates were 19% ($n = 5$) and 14% ($n = 3$), respectively. Although strokes were more common after CAS than after CEA, overall complications were less common in CAS. However, these differences were not significant statistically ($P = .21$ and $P = .27$, respectively).

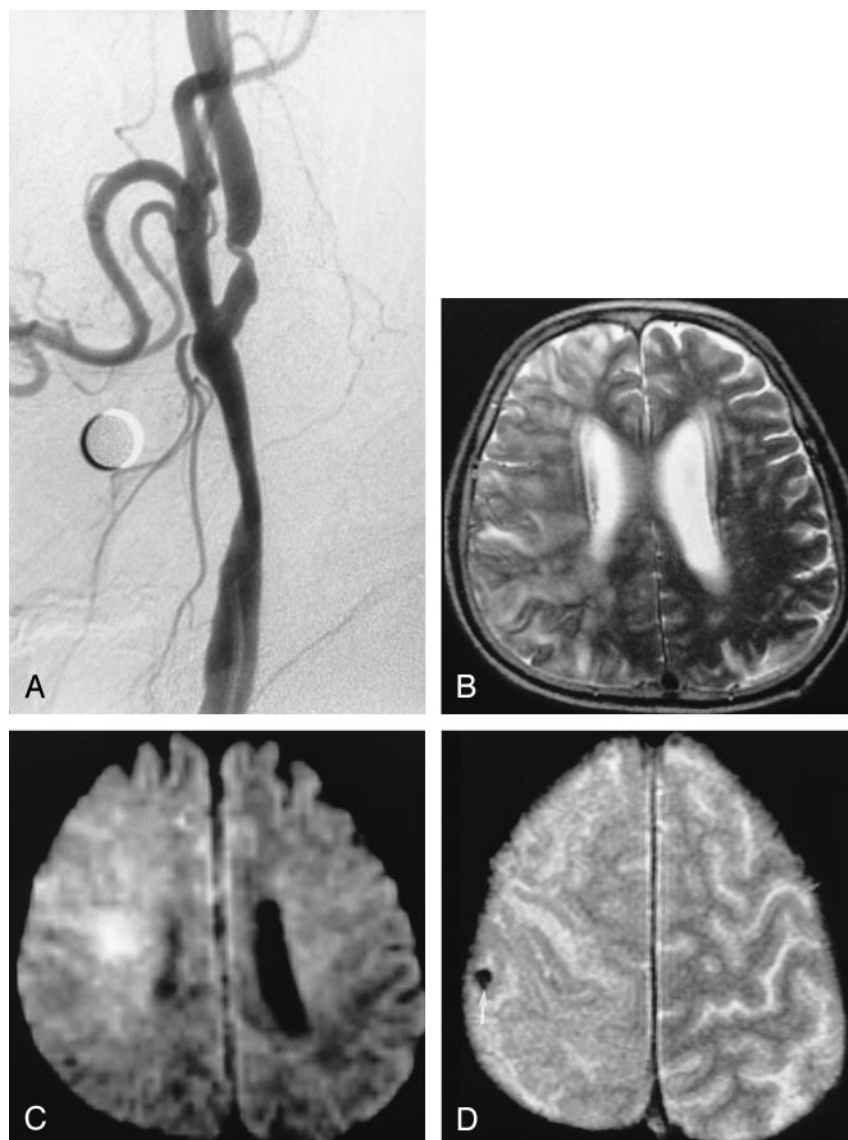
Because of the small sample of this prospective study and the rarity of observed cerebral events, no evident statistical relationship could be established between angiographic variables (such as plaque ulceration and contralateral stenosis or occlusion) and the occurrence of embolic brain lesions on DW images (Table 3).

Discussion

The treatment of ICA stenosis has significantly evolved during the last 40 years. The NASCET and Asymptomatic Carotid Atherosclerosis Study (ACAS), for example, have proved the absolute or relative benefit of CEA in reducing the stroke risks for symptomatic (7, 10, 13) and asymptomatic patients with significant carotid artery stenosis (8). Subsequently, the traditional surgical treatment with CEA became the standard of care in treating cervical carotid artery stenosis. However, it was found to have certain limitations. In the initial results from NASCET, 5.8% of the patients had perioperative major stroke or death (7). In the ACAS, the perioperative stroke rate was 2.3% (8). Recently, further results from NASCET showed 6.5% perioperative stroke and death, 2.0% permanently disabling stroke and death, 9.3% wound complication, and 8.6% cranial nerve paralysis (10).

FIG 2. Case of a 68-year-old man with severe stenosis (91%) of the right ICA, who claimed severe unilateral headache after the third post-CEA day.

A–D, Pre-CEA angiogram (A), post-CEA T2-weighted MR image (B), post-CEA DW image (C), and post-CEA gradient-echo MR image (D). Post-CEA MR images show severe vasogenic edema with focal hemorrhage (arrow) in the right cerebral hemisphere.



The European Carotid Surgery Trial, similar in size to that of NASCET, reported final results with comparable perioperative rates of stroke and death (13). Among the 1745 patients who received CEA, there were 122 nonfatal major strokes or death (7.0%); the death rate at 30 days was 1.0%, the disabling stroke rate 2.5%, and the non-disabling stroke rate 3.5% (13). Neck dissection and retraction necessary to reach the carotid artery may injure cranial nerves during CEA. Cranial and cervical nerve palsies have been reported in up to 27% of patients (7, 10, 17, 30–33), even in recent studies. There were four cranial nerve injuries in three patients (11.5%) in our CEA series although two were temporary and all developed among the patients with a high carotid bifurcation. Independent predictors of adverse outcome after CEA include contralateral occlusion, previous ipsilateral CEA, and combined coronary and carotid artery disease, although ongoing debates exist (10, 34–38).

CAS has recently emerged as a rational alternative

to CEA, especially to the high surgical risk group, although CAS has not been completely out of investigational status. CAS, compared with CEA, is now well accepted for its advantages and benefit because CAS can avoid surgical incision, requires only local anesthetic in the groin, and may be more cost-effective because of a shorter hospital stay and the reduced use of expensive intensive care unit (20, 39, 40). Furthermore, CAS may have advantages over CEA in specific clinical subgroups such as patients with substantial coexistent morbidities (e.g., severe cardiopulmonary disease), contralateral carotid occlusion, post-endarterectomy restenosis, radiation-induced stenosis, and surgically inaccessible (high, low, or tandem) lesions. CAS is not limited to the cervical portion of the carotid artery (4, 16, 40).

Since 1996, there have been many individual CAS series (4, 6, 9, 14, 15, 24, 39–45). Comparative analysis of these studies is difficult because of the heterogeneity in the sample populations, lesion characteristics, endovascular techniques, and outcome data.

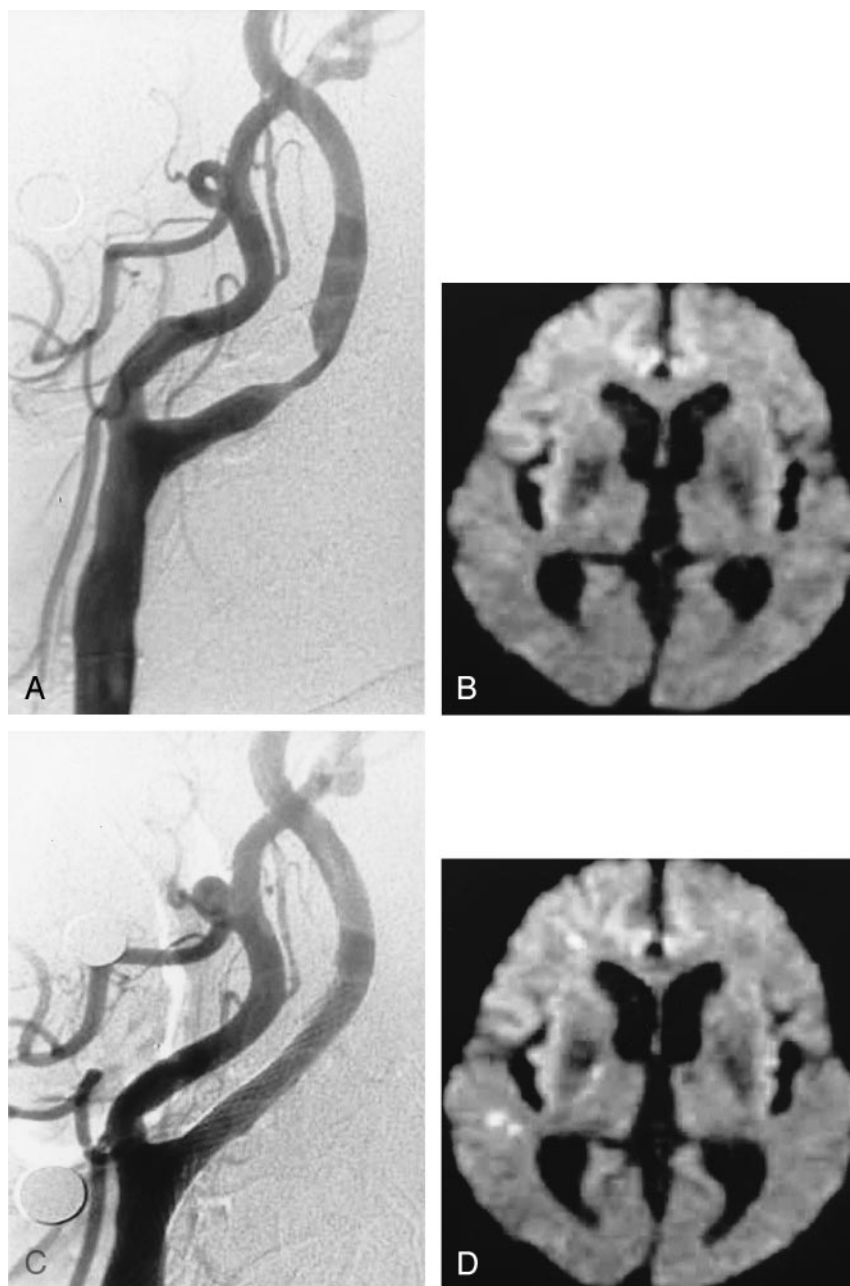


FIG 3. Case of a 58-year-old man with symptomatic moderate stenosis (69%) of the right ICA.

A–D, Pre-CAS angiogram (A), pre-CAS DW image (B), post-CAS angiogram (C), post-CAS DW image (D). Post-CAS DW image demonstrates several small hyperintensities in the right cerebral hemisphere including the striatocapsule. Mild left-sided weakness and visual field defect occurred after CAS.

TABLE 3: Rate of new hyperintensity on DW image according to the angiographic variables

Angiographic Variables	CEA (n = 26)	CAS (n = 22)
Ipsilateral ICA stenosis		
Severe	0 (0 of 19)	29 (4 of 14)
Moderate	1 (1 of 7)	50 (4 of 8)
Contralateral ICA stenosis		
Occlusion or severe	33 (1 of 3)	33 (4 of 12)
Normal to moderate	0 (0 of 23)	40 (4 of 10)
Ipsilateral ICA plaque ulcer		
Present	5 (1 of 21)	33 (6 of 18)
Absent	0 (0 of 5)	50 (2 of 4)

Note.—Data are percentage (number) of procedures in which a new hyperintensity was noted for that angiographic variable.

However, the overall reported rate of technical success is greater than 93%, procedure-related mortality rates (including cardiac deaths) are 0–4.5%, and stroke rates are 0–13%. The reported stroke and death rates after CAS were heterogeneous, and complication rates in many reports were higher than the postulated limit of 6% for symptomatic stenoses and 3% for asymptomatic stenoses (7, 8, 19, 24).

In 1996, Diethrich et al (39) reported two deaths (1.8%), seven strokes (6.3%), and five transient neurologic events (4.5%) after CAS. The authors concluded that the frequency of periprocedural neurologic complications was excessive. Diethrich, in an accompanying editorial, suggested that CAS be re-

stricted to cases of carotid restenosis after prior CEA, instances in which the internal carotid stenosis was anatomically higher than readily treated by CEA, and patients with radiation-induced stenosis (46). However, in a larger prospective study of 271 CAS procedures in 231 patients reported by Mathur et al (45) in 1998, there was one death, two major strokes (0.7%), and 17 (6.2%) minor strokes. Roubin et al (43) updated their previous report with inclusion of 604 procedures in 528 patients. The overall 30-day stroke and death rate was 7.4%.

Recently, results of an initial randomized multicenter clinical trial of Carotid and Vertebral Artery Transluminal Angioplasty Study were reported (20). Carotid angioplasty with or without stent placement had similar major risks and effectiveness at preventing stroke during 3 years compared with CEA. These results suggested that CAS might be comparable to CEA.

The efficacies of CEA and CAS in preventing stroke depend on the ability of the vascular surgeon and interventional neuroradiologist to achieve complication-free results. Ongoing refinements and advances in the techniques and devices are aimed at reducing the occurrence of neurologic complications. Most neurologic complications occurring during or immediately after CEA and CAS are said to result from cerebral embolism. Principally, three mechanisms have been implicated in stroke complicating CEA. The first is cerebral embolization during dissection of the carotid arteries and from the endarterectomy surface (47–49); the second, stroke as a result of hypoperfusion during clamping of the carotid arteries (49); and the third, stroke due to cerebral hemorrhage following clamp release and hyperperfusion (49, 50). Intraoperative hypoperfusion should rarely be a problem because brain perfusion can be maintained by collateral channels or selective shunt surgery. In contrast, small emboli arising from a friable plaque during arterial dissection and cross clamping may constitute an unavoidable risk of perioperative ischemic complications. There was one post-operative ischemic lesion on DW images and two hyperperfusion syndromes in our CEA series.

During CAS, while the risk of hypoperfusion is likely to be less, the frequency of embolization has been demonstrated to be much greater. For CAS of a tight carotid stenosis, a guidewire, balloon catheter, and stent introducer must be passed across the narrowing point and the plaque dilated or have a stent placed. This process is associated with a high frequency of cerebral embolization demonstrated on transcranial Doppler sonograms of the MCA (51). Long or multiple stenoses were suggested as independent predictors of stroke during CAS (45).

In these consecutive 48 procedures of 44 patients undergoing elective CEA or CAS, all new acute lesions were small and most could not be seen with conventional brain MR imaging. Older studies that used CT demonstrated new asymptomatic cerebral infarcts in 0–6% after CEA (52–54). The significance of those studies was obviously limited by the low sensitivity of CT scanning for acute ischemic changes.

In studies that used conventional MR imaging techniques, such as T1- and T2-weighted, FLAIR, and proton density-weighted sequences, the rate of post-operative silent infarction was higher than that in CT-based reports and ranged from 9–24% (55, 56). Recently, several DW imaging studies about embolic infarction after CEA have been reported (1, 2, 5, 57), and the rate of ischemic lesion on DW images ranged from 0–4.2%.

Our study has some limitations, including a relatively small sample and rarity of stroke in the two groups, relatively inconsistent time window for DW imaging even within one week, and different patient characteristics between the two groups. Angiographic variables such as plaque ulceration, degree of ipsilateral carotid artery stenosis, and contralateral carotid artery stenosis or occlusion could not be compared statistically with the occurrence of embolic brain lesions on DW images. Although DW imaging would provide a more specific and sensitive evaluation of acute ischemic lesion in comparison with prior CT and MR imaging studies, it may not demonstrate all ischemic lesions. We might have missed transiently or permanently reversible, short-lived ischemic lesions in some cases (58, 59). In the CAS series of 22 procedures, 54% (n = 12) had a contralateral ICA occlusion or severe stenosis; however, only 11.5% (n = 3) occurred in the CEA series of 26 procedures. Because of this relatively unfavorable patient selection, this small study from a single center may not be representative of the general results of CEA and CAS.

Conclusion

In this small series, the frequency of ischemic brain lesions of embolic origin following CEA was significantly lower than that of CAS, and therefore, CEA in general seems to be a safer procedure with a lower risk of post-operative cerebral or retinal ischemia, as long as meticulous surgical technique, reliable intraoperative monitoring, and neuroprotective measures are consistently applied. However, most hyperintensities on DW images were silent, and the overall symptomatic complications were higher in the CEA series than in the CAS series. CAS may be able to become a comparable procedure to CEA in view of its relatively low total complication rate and can be a more reliable procedure with continued advances in neuroprotective devices in the near future.

References

1. Forbes KPN, Shill HA, Britt PM, Zabramski JM, Spetzler RF, Heiserman JE. **Assessment of silent embolism from carotid endarterectomy by use of diffusion-weighted imaging: work in progress.** *AJNR Am J Neuroradiol* 2001;22:650–653
2. Barth A, Remonda L, Lövgren KO, Schroth G, Seiler RW. **Silent cerebral ischemia detected by diffusion-weighted MRI after carotid endarterectomy.** *Stroke* 2000;31:1824–1828
3. Vitek JJ, Roubin GS, Al-Mubarek N, New G, Iyer SS. **Carotid artery stenting: technical considerations.** *AJNR Am J Neuroradiol* 2000;21:1736–1743

4. Shawl F, Kadro W, Domanski MJ, et al. **Safety and efficacy of elective carotid artery stenting in high-risk patients.** *J Am Coll Cardiol* 2000;35:1721-1728
5. Feiwell RJ, Besmertis L, Sarkar R, Saloner DA, Rapp JH. **Detection of clinically silent infarcts after carotid endarterectomy by use of diffusion-weighted imaging.** *AJNR Am J Neuroradiol* 2001;22:646-649
6. Qureshi AI, Luft AR, Janardhan V, et al. **Identification of patients at risk for periprocedural neurological deficits associated with carotid angioplasty and stenting.** *Stroke* 2000;31:376-382
7. North American Symptomatic Carotid Endarterectomy Trial Collaborators. **Beneficial effect of carotid endarterectomy in symptomatic patients with high-grade carotid stenosis.** *N Engl J Med* 1991;325:445-453
8. Executive Committee for the Asymptomatic Carotid Atherosclerosis Study. **Endarterectomy for asymptomatic carotid artery stenosis.** *JAMA* 1995;273:1421-1428
9. Dietz A, Berkefeld J, Theron JG, et al. **Endovascular treatment of symptomatic carotid stenosis using stent placement: long-term follow-up of patients with a balanced surgical risk/benefit ratio.** *Stroke* 2001;32:1855-1859
10. Ferguson GG, Eliasziw M, Barr HWK, et al., for the North American Symptomatic Carotid Endarterectomy Trial (NASCET) Collaborators. **The North American Symptomatic Carotid Endarterectomy Trial: surgical results in 1415 patients.** *Stroke* 1999;30:1751-1758
11. Biller J, Feinberg WM, Castaldo JE, et al. **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
12. Bettmann MA, Katzen BT, Whisnant J, et al. **Carotid stenting and angioplasty: a statement for healthcare professionals from the Councils on Cardiovascular Radiology, Stroke, Cardio-Thoracic and Vascular Surgery, Epidemiology and Prevention, and Clinical Cardiology, American Heart Association.** *Stroke* 1998;29:336-338
13. European Carotid Surgery Trialists' Collaboratory Group. **Randomised trial of endarterectomy for recently symptomatic carotid stenosis: final results of the MRC European Carotid Surgery Trial (ECST).** *Lancet* 1998;351:1379-1387
14. Theron JG, Payelle GG, Coskun O, Huet HF, Guimaraens L. **Carotid artery stenosis: treatment with protected balloon angioplasty and stent placement.** *Radiology* 1996;201:627-636
15. Roubin GS, Yadav S, Iyer SS, Vitek J. **Carotid stent-supported angioplasty: a neurovascular intervention to prevent stroke.** *Am J Cardiol* 1996;78:8-12
16. Phatouros CC, Higashida RT, Malek AM, et al. **Carotid artery stent placement for atherosclerotic disease: rationale, technique, and current status.** *Radiology* 2000;217:26-41
17. Wholey MH, Wholey M, Mathias K, et al. **Global experience in cervical carotid artery stent placement.** *Catheter Cardiovasc Interv* 2000;50:160-167
18. Malek AM, Higashida RT, Phatouros CC, et al. **Stent angioplasty for cervical carotid artery stenosis in high-risk symptomatic NASCET-ineligible patients.** *Stroke* 2000;31:3029-3033
19. Joint Officers of the Congress of Neurological Surgeons and the American Association of Neurological Surgeons. **Carotid angioplasty and stent: an alternative to carotid endarterectomy.** *Neurosurgery* 1997;40:344-345
20. CAVATAS Investigators. **Endovascular versus surgical treatment in patients with carotid stenosis in the Carotid and Vertebral Artery Transluminal Angioplasty Study (CAVATAS): a randomised trial.** *Lancet* 2001;357:1729-1737
21. Hobson RW II, Lal BK, Chaktoura E, et al. **Carotid artery stenting: analysis of data for 105 patients at high risk.** *J Vasc Surg* 2003;37:1234-1239
22. Lovblad KO, Laubach HJ, Baird AE, et al. **Clinical experience with diffusion-weighted MR in patients with acute stroke.** *AJNR Am J Neuroradiol* 1998;19:1061-1066
23. Beauchamp NJ Jr, Barker PB, Wang PY, vanZijl PC. **Imaging of acute cerebral ischemia.** *Radiology* 1999;212:307-324
24. van Everdingen KJ, van der Grond J, Kappelle LJ, Ramos LMP, Mali WPTM. **Diffusion-weighted magnetic resonance Imaging in acute stroke.** *Stroke* 1998;29:1783-1790
25. Rordorf G, Bellon RJ, Budzik RE Jr, et al. **Silent thromboembolic events associated with the treatment of unruptured cerebral aneurysms by use of Guglielmi detachable coils: prospective study applying diffusion-weighted imaging.** *AJNR Am J Neuroradiol* 2001;22:5-10
26. Muller M, Reiche W, Langenscheidt P, Hassfeld J, Hagen T. **Ischemia after carotid endarterectomy: comparison between transcranial Doppler sonography and diffusion-weighted MR imaging.** *AJNR Am J Neuroradiol* 2000;21:47-54
27. Jaeger HJ, Mathias KD, Drescher R, et al. **Diffusion-weighted MR imaging after angioplasty or angioplasty plus stenting of arteries supplying the brain.** *AJNR Am J Neuroradiol* 2001;22:1251-1259
28. Britt PM, Heiserman JE, Snider RM, Shill HA, Bird CR, Wallace RC. **Incidence of postangiographic abnormalities revealed by diffusion-weighted MR imaging.** *AJNR Am J Neuroradiol* 2000;21:55-59
29. Bendszus M, Koltzenburg M, Burger R, Warmuth-Metz M, Hofmann E, Solymosi L. **Silent embolism in diagnostic cerebral angiography and neurointerventional procedures: a prospective study.** *Lancet* 1999;354:1594-1597
30. Forssell C, Kitzing P, Bergqvist D. **Cranial nerve injuries after carotid artery surgery: a prospective study of 663 operations.** *Eur J Vasc Endovasc Surg* 1995;10:445-449
31. Maroulis J, Karkanevatos A, Papakostas K, Gilling-Smith GL, McCormick MS, Harris PL. **Cranial nerve dysfunction following carotid endarterectomy.** *Int Angiol* 2000;19:237-241
32. AbuRahma AF, Choueiri MA. **Cranial and cervical nerve injuries after repeat carotid endarterectomy.** *J Vasc Surg* 2000;32:649-654
33. Zannetti S, Parente B, De Rango P, et al. **Role of surgical techniques and operative findings in cranial and cervical nerve injuries during carotid endarterectomy.** *Eur J Vasc Endovasc Surg* 1998;15:528-531
34. Baker WH, Howard VJ, Howard G, Toole JF. **Effect of contralateral occlusion on long-term efficacy of endarterectomy in the Asymptomatic Carotid Atherosclerosis Study (ACAS).** *ACAS Investigators.* *Stroke* 2000;31:2330-2334
35. Gasecki AP, Eliasziw M, Ferguson GG, Hachinski V, Barnett HJ. **Long-term prognosis and effect of endarterectomy in patients with symptomatic severe carotid stenosis and contralateral carotid stenosis or occlusion: results from NASCET.** *North American Symptomatic Carotid Endarterectomy Trial (NASCET) Group.* *J Neurosurg* 1995;83:778-82
36. Rihal CS, Gersh BJ, Whisnant JP, et al. **Influence of coronary heart disease on morbidity and mortality after carotid endarterectomy: a population-based study in Olmsted County, Minnesota (1970-1988).** *J Am Coll Cardiol* 1992;19:1254-1260
37. Mackey WC, O'Donnell TF Jr, Callow AD. **Cardiac risk in patients undergoing carotid endarterectomy: impact on perioperative and long-term mortality.** *J Vasc Surg* 1990;11:226-233
38. Salenius JP, Harju E, Riekkinen H. **Early cerebral complications in carotid endarterectomy: risk factors.** *J Cardiovasc Surg* 1990;31:162-167
39. Diethrich EB, Ndiaye M, Reid DB. **Stenting in the carotid artery: initial experience in 110 patients.** *J Endovasc Surg* 1996;3:42-62
40. Brown MM. **Carotid angioplasty and stenting: are they therapeutic alternatives [Suppl]?** *Cerebrovasc Dis* 2001;11:112-118
41. Criado FJ, Wellons E, Clark NS. **Evolving indications for and early results of carotid artery stenting.** *Am J Surg* 1997;174:111-114
42. Yadav JS, Roubin GS, Iyer S, Vitek J, King P, Jordan WD, Fisher WS. **Elective stenting of the extracranial carotid arteries.** *Circulation* 1997;95:376-81
43. Roubin GS, New G, Iyer SS, et al. **Immediate and late clinical outcomes of carotid artery stenting in patients with symptomatic and asymptomatic carotid artery stenosis: a 5-year prospective analysis.** *Circulation* 2001;103:532-537
44. Al-Mubarak N, Roubin GS, Gomez CR, et al. **Carotid artery stenting in patients with high neurologic risks.** *Am J Cardiol* 1999;83:1411-1413
45. Mathur A, Roubin GS, Iyer SS, et al. **Predictors of stroke complicating carotid artery stenting.** *Circulation* 1998;97:1239-1245
46. Diethrich EB. **Indications for carotid artery stenting: a preview of the potential derived from early clinical experience.** *J Endovasc Surg* 1996;3:132-139
47. Riles TS, Imparato AM, Jacobowitz GR, et al. **The cause of perioperative stroke after carotid endarterectomy.** *J Vasc Surg* 1994;19:206-214
48. Smith JL, Evans DH, Fan L, et al. **Interpretation of embolic phenomena during carotid endarterectomy.** *Stroke* 1995;26:2281-2284
49. Spencer MP. **Transcranial Doppler monitoring and causes of stroke from carotid endarterectomy.** *Stroke* 1997;28:685-691
50. Dalman JE, Beenackers IC, Moll FL, Leusink JA, Ackerstaff RG. **Transcranial Doppler monitoring during carotid endarterectomy helps to identify patients at risk of postoperative hyperperfusion.** *Eur J Vasc Endovasc Surg* 1999;18:222-227

51. Markus HS, Clifton A, Buckenham T, Taylor R, Brown MM. **Improvement in cerebral hemodynamics after carotid angioplasty.** *Stroke* 1996;27:612-616
52. Habozit B. **The silent brain infarct before and after carotid surgery.** *Ann Vasc Surg* 1990;4:485-489
53. Sise MJ, Sedwitz MM, Rowley WR, Shackford SR. **Prospective analysis of carotid endarterectomy and silent cerebral infarction in 97 patients.** *Stroke* 1989;20:329-332
54. Street DL, O'Brien MS, Ricotta JJ, et al. **Observations on cerebral computed tomography in patients having carotid endarterectomy.** *J Vasc Surg* 1988;7:798-801
55. Mericle RA, Kim SH, Lanzino G, et al. **Carotid artery angioplasty and use of stents in high-risk patients with contralateral occlusions.** *J Neurosurg* 1999;90:1031-1036
56. Cantelmo NL, Babikian VL, Samaraweera RN, Gordon JK, Pochay VE, Winter MR. **Cerebral microembolism and ischemic changes associated with carotid endarterectomy.** *J Vasc Surg* 1998;27:1024-1030
57. Enevoldsen EM, Torfing T, Kjeldsen MJ, Nepper-Rasmussen J. **Cerebral infarct following carotid endarterectomy: frequency, clinical and hemodynamic significance evaluated by MRI and TCD.** *Acta Neurol Scand* 1999;100:106-110
58. Kidwell CS, Alger JR, Di Salle F, et al. **Diffusion MRI in patients with transient ischemic attacks.** *Stroke* 1999;30:1174-1180
59. Li F, Liu KF, Silva MD, et al. **Transient and permanent resolution of ischemic lesions on diffusion-weighted imaging after brief periods of focal ischemia in rats: correlation with histopathology.** *Stroke* 2000;31:946-954