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## Silent Cerebral Ischemia Detected by Diffusion-Weighted MRI After Carotid Endarterectomy

Alain Barth, MD; Luca Remonda, MD; Karl-Olof Lövblad, MD; Gerhard Schroth, MD; Rolf W. Seiler, MD

- **Background and Purpose**—Small emboli arising from a friable plaque during carotid endarterectomy (CEA) constitute an important risk of perioperative ischemic complications. To evaluate the incidence and significance of silent cerebral ischemic lesions of embolic origin after CEA, we prospectively examined a series of surgical patients with high-grade carotid stenosis by using diffusion-weighted MRI (DWI). We also tried to correlate postoperative ischemic lesions with the occurrence of sonographic cerebral embolic signals, the presence of plaque ulcerations, and the use of intraoperative shunting.
- *Methods*—Of a consecutive series of 53 patients undergoing elective CEA for high-grade carotid stenosis, 48 patients with unchanged postoperative neurological status were prospectively studied with DWI of the brain the day before and the day after the operation. The magnetic resonance images were analyzed by 2 neuroradiologists blinded to the clinical result of the operation. Any new hyperintense signal was interpreted as a postoperative ischemic lesion.
- *Results*—Forty-six (95.8%) of 48 patients had unchanged postoperative brain DWI. In 2 patients (4.2%), a new single asymptomatic hyperintense signal was observed on the side of the operation. Both lesions were small and presumably of embolic origin. They were not related to sonographic embolic signals, plaque ulcerations, or intraoperative shunting.*Conclusions*—These results suggest that the incidence of silent ischemic brain lesions of embolic origin after CEA is low
- and does not correlate with the occurrence of intraoperative sonographic microemboli. They confirm that CEA is a safe procedure that carries a low risk of postoperative cerebral events. (*Stroke*. 2000;31:1824-1828.)

Key Words: carotid endarterectomy ■ carotid stenosis ■ cerebral embolism ■ magnetic resonance imaging, diffusion-weighted

C arotid endarterectomy (CEA) is currently considered the best treatment for stroke prevention in patients with high-grade carotid stenosis.<sup>1</sup> The usefulness of CEA is dependent on a low incidence of perioperative neurological complications and death. Current standards postulate a surgical morbidity/mortality rate <6% for symptomatic stenoses and <3% for asymptomatic stenoses.<sup>2,3</sup> Strict patient selection criteria and a very high quality of surgical care are mandatory if such low complication rates are to be achieved. Institutions performing CEA are requested to keep an exact documentation of their results to maintain high performance standards and correct possible deviations from the accepted norms.<sup>2</sup>

Most neurological complications occurring during or immediately after CEA are said to result from cerebral embolism.<sup>4–6</sup> Intraoperative hypoperfusion should rarely be a problem because brain perfusion can be maintained by collateral channels or selective shunting. In contrast, small emboli arising from a friable plaque during arterial dissection and cross-clamping constitute an unavoidable risk of perioperative ischemic complications. Several studies using transcranial Doppler (TCD) monitoring have measured a prevalence of embolic signals as high as 95% during CEA.<sup>5,7–13</sup> It has been suggested that increased cerebral microembolization may positively correlate with postoperative deterioration of cognitive function or the appearance of lacunar infarcts on MRI.<sup>7,8</sup> However, the clinical relevance of sonographic embolic signals remains unclear because most of them are not associated with focal neurological deficits.<sup>14,15</sup> Whether embolic signals represent an increased risk of postoperative silent brain ischemia also remains to be determined.

Recent developments in MRI technology have greatly improved the quality and rapidity of the diagnosis of cerebral ischemia. Diffusion-weighted MRI (DWI) is a quick, noninvasive, and reliable tool for the detection of acute ischemic lesions of the brain.<sup>16–19</sup> Small ischemic areas not exceeding 3 mm in diameter can be demonstrated only on DWI sequences. DWI can be performed early after an operation with little inconvenience to the patients. It is a suitable new

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method for improving quality control in cerebrovascular interventions.

In the present prospective study, we performed preoperative and postoperative DWI of the brain in a consecutive series of 48 CEA patients who did not show any new neurological deficit after the operation. The principal objective was to evaluate the incidence and significance of silent ischemic brain lesions of embolic origin after CEA. We also tried to correlate ischemic events with possible risk factors, including intraoperative sonographic cerebral emboli, ulcerations in the removed atheromatous plaque, and use of an intraoperative shunt device.

#### **Subjects and Methods**

#### **Patient Selection**

Forty-eight patients with unchanged neurological status after CEA were included in the study. They were part of a consecutive series of 53 patients who underwent elective CEA for high-grade carotid stenosis between November 1998 and December 1999. Five patients were excluded from the study: 1 with a symptomatic postoperative brain infarct after acute thrombosis of the operated carotid bifurcation, 2 with pacemakers, and 2 with claustrophobia. No deaths occurred during the study. The diagnosis of high-grade carotid stenosis was made by ultrasonography and confirmed by magnetic resonance angiography.20 The degree of stenosis was determined with the North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria<sup>21</sup>: percentage of stenosis=100×[1-(minimal residual lumen/distal lumen)]. Age, sex, vascular risk factors, and neurological history and status of each patient were recorded before surgery. The risk factors considered included smoking, diabetes mellitus, arterial hypertension, hyperlipidemia, obesity, and a positive family history. We asked about any history of coronary artery disease or bypass surgery, as well as peripheral vascular occlusive disease, as additional risk factors for carotid surgery. The surgical risks were evaluated by using the 4-grade classification of Sundt and colleagues<sup>22,23</sup> and Meyer et al<sup>24</sup>: grade I, patients aged <70 years with no medical or neurological risks; grade II, patients with angiographically determined risks, including contralateral carotid stenosis, intracranial atherosclerosis, or high common carotid bifurcation; grade III, patients with a major medical risk (in most circumstances, severe coronary artery disease); and grade IV, patients neurologically unstable, with persistent transient ischemic attacks or crescendo transient ischemic attacks despite adequate anticoagulation.

#### **Carotid Endarterectomy**

The operation was performed with the patient under general anesthesia. Neuroprotective measures during cross-clamping included moderate hypothermia (34°C to 35°C) and propofol administration until the appearance of burst suppression on EEG. TCD sonography enabled continuous intraoperative monitoring of brain perfusion and detection of microembolic signals. Intravenous heparin (100 U/kg) was administered before the internal carotid artery (ICA) was exposed. To avoid particulate emboli during surgical preparation, care was taken to obtain exposure of the ICA high above the upper extent of the stenosis. Intraoperative shunting was performed only when blood flow velocities in the ipsilateral middle cerebral artery (MCA) decreased to <40% of preclamping values.<sup>25</sup> If indicated, the shunt (Sundt Carotid Endarterectomy Shunts, Heyer-Shulte Neurocare Group) was introduced proximally into the common carotid artery after arteriotomy, then rinsed to remove all possible particulate fragments, and finally inserted distally into the ICA. After gross removal of the plaque, the carotid bifurcation was inspected through the operating microscope, and any loose residual fragments of the plaque were carefully removed from the vessel wall. The arteriotomy was closed with a running 6-0 Prolene suture without any venous or artificial patch. Aspirin (100 mg) was routinely administered 6 hours after the operation. Full inhibition of thrombocytic aggregation was restarted on the first postoperative day.

The excised plaques were examined macroscopically after the operation. They were classified as smooth or ulcerated depending on the absence or presence of ulcerations and intramural thrombi.

#### Intraoperative TCD Monitoring

Continuous intraoperative TCD monitoring was performed with a 2-MHz pulsed-Doppler transducer fixed with a head strap over the temporal bone ipsilateral to the operation (Multi Dop X4, DWL Elektronische Systeme GmbH). The mean maximal blood flow velocity in the MCA was measured at an insonation depth of 45 to 55 mm. Microemboli in the MCA were identified by use of the emboli detection program provided by Multi Dop X4 DWL-apparatus (DWL Elektronische Systeme GmbH) with a decibel threshold of 9 dB. Microemboli were defined as transient (<300 ms), highintensity, unidirectional signals within the Doppler velocity spectrum accompanied by an audible snap on the auditory output.<sup>26,27</sup> By the method of Spencer,5 brief high-intensity signals occurring exclusively at clamp release were considered to represent air bubbles and were not counted as cerebral emboli. Only sustained auditory and spectral signals representing true particulate matter emboli were considered to be significant. TCD microemboli detection was performed during the different phases of the operation, defined as follows: preoperative (from intubation to skin incision), intraoperative (from skin incision to skin closure), early postoperative (from skin closure to recovery from anesthesia), and late postoperative (3 hours after awakening). The preoperative, early, and late postoperative insonation phases lasted 20 to 45 minutes. The intraoperative insonation phase was uninterrupted and lasted 80 to 120 minutes.

#### **Magnetic Resonance Examination**

MRI was performed on a 1.5-T Siemens Vision apparatus equipped with a head coil (Siemens Medical Systems). The preoperative workup routinely included first-pass gadolinium-enhanced magnetic resonance angiography of the neck vessels and MRI of the brain.<sup>20</sup> In addition, DWI was prospectively performed the day before and the day after CEA after obtaining the patient's written consent. Wholebrain diffusion-weighted images were acquired with an echo-planar sequence. An isotropic sequence was used (repetition time 4000 ms, echo time 133 ms, field of view 210 mm, matrix  $128 \times 128$ , and number of excitations 4), with *b* values of 0 and 972 s/mm<sup>2</sup>. The DWIs were then evaluated by 2 experienced neuroradiologists (L.R., K.-O.L.) blinded to the clinical status of the patients. Any presence of new hyperintensities on the brain scans was interpreted as a sign of new embolic ischemic lesions after CEA.

#### Results

The mean age of the 48 studied patients was  $66.7\pm7$  years (range 46 to 83 years). There were 15 women (31%) and 33 men (69%). Cigarette smoking (62%) and arterial hypertension (62%) were the most frequent vascular risk factors, followed by hyperlipidemia (48%), positive family history (23%), obesity (19%), and diabetes mellitus (8%). A history of coronary artery disease or bypass surgery was present in 33% of the patients, and a history of peripheral vascular occlusive disease was present in 25%. Thirty-two patients (66%) had symptomatic carotid stenosis (15 with transient ischemic attacks and 17 with brain infarcts). Sixteen patients (34%) had asymptomatic carotid stenosis. The contralateral internal carotid artery was occluded in 4 patients (8%) and highly stenotic in 2 (4%). One patient (2%) was Sundt grade I, 18 patients (37%) were grade II, 21 patients (44%) were grade III, and 8 patients (17%) were grade IV.

Preoperative MRI of the brain was normal in 29 patients (60%). MRI showed old or subacute ipsilateral brain infarcts



**Figure 1.** A, Preoperative brain DWI of a 63-year-old man with a left carotid artery stenosis presenting with aphasia and parietal brain infarction. B, Postoperative brain DWI demonstrating a single new hyperintense lesion in the ipsilateral parietal occipital cortex (arrow). This lesion was compatible with a small perioperative ischemic area of embolic origin.

in 11 patients (23%) with symptomatic carotid stenosis. Eight patients (17%) had acute ischemic lesions evident on preoperative DWI. By selection, all 48 patients were neurologically unchanged after the operation. Forty-six of them (95.8%) also had unchanged postoperative cerebral DWI. A new postoperative asymptomatic ischemic lesion was observed in 2 patients (4.2%).

The first patient was a 63-year-old man operated on for a left-sided symptomatic high-grade carotid stenosis with parietal brain infarction and residual aphasia. No particulate emboli were detected at TCD monitoring during the intervention. No shunt was used because an adequate collateral circulation was present. The removed plaque showed extensive ulcerations with intramural thrombi. In this patient, the wound had been reopened at the end of the operation because of persistent venous oozing. The operated carotid bifurcation was not manipulated again, and awakening was uneventful. On postoperative brain DWI, a new small hyperintense lesion was observed in the ipsilateral parietal occipital cortex (Figure 1). The size of this lesion was  $\approx 6$  mm, and the morphology suggested a small peripheral branch embolus.

The second patient was a 74-year-old woman operated on for an asymptomatic high-grade carotid stenosis on the right side. Operation and recovery were uneventful. Because the temporal bone windows were absent, no TCD monitoring could be performed, and a temporary shunt was inserted during the operation. Morphologically, the removed plaque was smooth without ulcerations or thrombi. On postoperative brain DWI, a new single hyperintensity <4 mm in diameter was detected in the ipsilateral subcortical temporal region (Figure 2). The morphology of this minute silent lesion was also highly suggestive of a perioperative embolus.

In the complete series of 48 examined patients, useful TCD monitoring was performed in 42 patients. Significant cerebral particulate emboli were detected in 7 patients (17%) during the perioperative period. Ulcerations with or without intramural thrombi were observed macroscopically in 21 patients (44%). A shunt was used selectively in 12 patients (25%). Because of the small sample size and the rarity of observed cerebral events, no evident relationship could be established



**Figure 2.** A Preoperative brain DWI of a 74-year-old woman with an asymptomatic high-grade carotid stenosis on the right side. B, Postoperative brain DWI demonstrating a single new hyperintensity in the ipsilateral subcortical temporal region (arrow), which was highly suggestive of a perioperative embolus.

between these postulated risk factors and the occurrence of silent embolic brain lesions on DWI.

#### Discussion

In this consecutive series of 53 patients undergoing elective CEA for high-grade carotid stenosis, the stroke rate was 1.9% and the mortality rate was 0%. The incidence of silent postoperative brain ischemia demonstrated by DWI was 4.2%, corresponding to 2 new lesions found in the 48 patients examined. Both new acute lesions were small in size and would probably not have been detected by conventional brain CT or MRI. Despite the small sample size, these results can be considered excellent compared with previously published reports. Older studies using CT scan demonstrated new asymptomatic cerebral infarcts in 0% to 6% of operated patients.<sup>7,28-32</sup> CT scan was usually performed 3 to 5 days after the operation, when infarcted areas were sufficiently demarcated. The significance of these studies was obviously limited by the low sensitivity of CT scanning for acute ischemic changes. More recent studies applied conventional MRI techniques, such as T1- and T2-weighted images, fluid-attenuated inversion recovery, and proton density sequences. The rate of postoperative silent infarction was higher than in CT-based reports and ranged from 0% to 24%.8,12,13,33 Still higher rates of silent ischemia would probably have been demonstrated in these MRI studies if they had been supplemented by diffusion-weighted sequences. In the present series, the probability of missing postoperative ischemic lesions was particularly small because DWI was performed in the first 24 hours after the operation to detect any short-lived cerebral changes.

A high percentage of the patients enrolled in the present study had considerable preoperative risk factors. Seventeen percent were considered Sundt grade IV, and 44% were considered Sundt grade III. Twelve percent had a contralateral ICA occlusion or high-grade stenosis. Acute ischemic brain lesions were evident in 17% of preoperative DWI examinations. Despite this relatively unfavorable patient selection, the demonstrated rate of postoperative silent ischemia was quite low. This small study from a single center may not be representative of the general results of CEA; however, it adds accurate and valuable information about the cerebral safety that can be reached during the operation.

The significance of microembolic signals detected by TCD monitoring during and after the operation remains unclear.<sup>5</sup> It has been shown that perioperative cerebral embolism is responsible for 70% to 80% of stroke after CEA.4-6 In our series, one patient suffered partial MCA territory infarction after acute thrombosis of the operated carotid bifurcation. The embolic mechanism was evident in this case. In addition, both new cerebral lesions detected in the unchanged patients were small and probably corresponded to asymptomatic embolic foci. However, they were not related to sonographic microembolic signals, which were observed in 17% of 42 monitored patients. It is known that the rate of detection of microemboli with TCD monitoring depends strongly on the technique used. Very high percentages (up to 95%) have been reported by several groups of investigators.<sup>5,7-13</sup> Exclusion of the sonographic signals that occur at and immediately after declamping greatly reduces the overall rate of detected microemboli. In one study considering only emboli occurring at flow restoration, a rate as low as 5% was reported along with what were considered to be flow turbulence signals in 40% of cases.<sup>34</sup> Several studies have found that the occurrence of postoperative cerebral ischemia is correlated with increased numbers of sonographic microemboli.8,10,11 Given the small sample size of the present study and the relatively low rate of observed microemboli, our results do not allow any conclusion concerning the number of sonographic embolic signals and the occurrence of radiologically detectable cerebral lesions.

Problems arising at placement of an intraoperative shunt or during manipulations of an ulcerated plaque are most often related to surgical technique. For shunt insertion, we adopted the method of Sundt et al,<sup>35</sup> who recommend first introducing the shunt proximally into the CCA, then rinsing all possible particulate fragments, and finally inserting the shunt distally into the ICA. No increase of silent brain lesions was observed on DWI in our series when a shunt was used, as happened in 25% of the patients. Similarly, silent cerebral lesions were not related to the ulcerated character of the removed plaque in our series, although ulcerations and intramural thrombi were observed in no fewer than 44% of the patients. This fact once again indicates the necessity of gentle handling of the vessels during carotid preparation.

The results of CEA must be compared with those of new endovascular therapeutic modalities, such as percutaneous carotid angioplasty and stenting. At present, only a small series of studies involving endovascularly treated carotid stenoses have been published. The available results are heterogeneous, and complication rates are generally higher than the postulated limit of 6% for symptomatic stenoses and 3% for asymptomatic stenoses.<sup>36–42</sup> Although it is attractive because it is less invasive than surgery, carotid angioplasty seems to have a much higher risk of periprocedural cerebral embolism.<sup>43</sup> The risk of particulate emboli to the brain during catheterization is increased in older patients with friable atherosclerotic vessel walls and tortuous stenoses at the carotid bifurcation. However, in a few series of studies containing a small number of patients, a benefit of angio-

plasty and stenting has been reported in high-risk patients presenting with a contralateral carotid occlusion or in patients who experienced restenosis after CEA.<sup>44,45</sup> Few DWI studies have examined the issue of silent cerebral ischemia after endovascular procedures. A recent study by Bendszus et al<sup>46</sup> examined the occurrence of silent embolism in diagnostic and interventional cerebral angiography. Among 100 consecutive angiographic procedures, brain DWI showed 42 hyperintense lesions consistent with cerebral embolism in 23 patients. All the lesions were asymptomatic. In a series of 19 patients undergoing carotid angioplasty and stenting for high-grade stenosis, Lövblad et al<sup>47</sup> observed new hyperintensities on DWI in 4 patients (21%). Two patients presented new neurological deficits, and 2 remained asymptomatic.

In conclusion, our results suggest that the incidence of silent ischemic brain lesions of embolic origin after CEA is low and does not correlate with the occurrence of intraoperative sonographic microemboli. These results confirm that the operation is safe for the brain as long as meticulous surgical technique, reliable intraoperative monitoring, and neuroprotective measures are consistently applied.

#### References

- Gorelick PB. Carotid endarterectomy: where do we draw the line? Stroke. 999;30:1745–1750.
- Moore WS, Barnett HJM, Beebe HG, Bernstein EF, Brener BJ, Brott T, Caplan LR, Day A, Goldstone J, Hobson RW II, et al. Guidelines for carotid endarterectomy: a multidisciplinary consensus statement from the ad hoc committee, American Heart Association. *Stroke*. 1995;26: 188–201.
- Biller J, Feinberg WM, Castaldo JE, Whittemore AD, Harbaugh RE, Dempsey RJ, Caplan LR, Kresowik TF, Matchar DB, Tooke JF, 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.
- Krul JMJ, van Gijn J, Askerstaff RGA, Eikelboom BC, Theodorides T, Vermeulen FEE. Site and pathogenesis of infarcts associated with carotid endarterectomy. *Stroke*. 1989;20:324–328.
- Spencer MP. Transcranial Doppler monitoring and causes of stroke from carotid endarterectomy. *Stroke*. 1997;28:685–691.
- Ferguson GG, Eliasziw M, Barr HWK, Clagett GP, Barnes RW, Wallace MC, Taylor DW, Haynes RB, Finan JW, Hachinski VC, 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.
- Gaunt ME, Martin PJ, Smith JL, Rimmer T, Cherryman G, Ratliff DA, Bell PRF, Naylor AR. Clinical relevance of intraoperative embolization detected by transcranial Doppler ultrasonography during carotid endarterectomy: a prospective study of 100 patients. *Br J Surg.* 1994;81: 1435–1439.
- Ackerstaff RGA, Jansen C, Moll FL, Vermeulen FEE, Mamerlijnck RPHM, Mauser HW. The significance of microemboli detection by means of transcranial Doppler ultrasonography monitoring in carotid endarterectomy. *J Vasc Surg.* 1995;21:963–969.
- Gavrilescu T, Babikian VL, Cantelmo NL, Rosales R, Pochay V, Winter M. Cerebral microembolism during carotid endarterectomy. *Am J Surg.* 1995;170:159–164.
- Lennard N, Smith J, Dumville J, Abbott R, Evans DH, London NJM, Bell PRF, Naylor AR. Prevention of postoperative thrombotic stroke after carotid endarterectomy: the role of transcranial Doppler ultrasound. J Vasc Surg. 1997;26:579–584.
- Levi CR, O'Malley HM, Fell G, Roberts AK, Hoare MC, Royle JP, Chan A, Beiles BC, Chambers BR, Bladin CF, et al. Transcranial Doppler detected cerebral microembolism following carotid endarterectomy: high microembolic signal loads predict postoperative cerebral ischemia. *Brain*. 1997;120:621–629.
- Gaunt ME. Transcranial Doppler: preventing stroke during carotid endarterectomy. Ann R Coll Surg Engl. 1998;80:377–387.

- 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;27:1024–1031.
- Gregoretti S, Bradley EL Jr. Regarding 'The significance of microemboli detection by means of transcranial Doppler ultrasonography monitoring in carotid endarterectomy.' J Vasc Surg. 1996;23:734–736.
- Müller M, Behnke S, Walter P, Omlor G, Schimrigk K. Microembolic signals and intraoperative stroke in carotid endarterectomy. *Acta Neurol Scand.* 1998;97:110–117.
- Warach S, Chien D, Li W, Ronthal M, Edelman RR. Fast magnetic resonance diffusion-weighted imaging in acute stroke. *Neurology*. 1992; 42:1717–1723.
- Sorensen AG, Buonano FS, Gonzalez RG, Schwamm LH, Lev MH, Huang-Hellinger FR, Reese TG, Weisskoff RM, Davis TL, Suwnwely N, et al. Hyperacute stroke: evaluation with combined multisection diffusion-weighted and hemodynamically-weighted echo-planar MR imaging. *Radiology*. 1996;199:391–401.
- Lutsep HL, Albers GW, De Crespigny A, Kamat GN, Marks MP, Moseley ME. Clinical utility of diffusion-weighted magnetic resonance imaging in the assessment of ischemic stroke. *Ann Neurol.* 1997;41: 574–580.
- Lövblad KO, Laubach HJ, Baird AE, Curtin F, Schlaug G, Edelman RR, Warach S. Clinical experience with diffusion-weighted MR in patients with acute stroke. *AJNR Am J Neuroradiol*. 1998;19:1061–1066.
- Remonda L, Heid O, Schroth G. Carotid artery stenosis, occlusion, and pseudo-occlusion: first-pass, gadolinium-enhanced, three-dimensional MR angiography: preliminary study. *Radiology*. 1998;209:95–102.
- NASCET Collaborators. Beneficial effect of carotid endarterectomy in symptomatic patients with high-grade carotid stenosis. N Engl J Med. 1991;325:445–453.
- Sundt TM Jr, Sandok BA, Whisnant JP. Carotid endarterectomy: complications and preoperative assessment of risk. *Mayo Clin Proc.* 1975; 50:301–306.
- Sundt TM Jr, Whisnant JP, Houser OW, Fode NC. Prospective study of the effectiveness and durability of carotid endarterectomy. *Mayo Clin Proc.* 1990;65:625–635.
- Meyer FB, Meissner I, Fode NC, Losasso TJ. Carotid endarterectomy in elderly patients. *Mayo Clin Proc.* 1991;66:464–469.
- Halsey JH. Risks and benefits of shunting in carotid endarterectomy: the International Transcranial Doppler Collaborators. *Stroke*. 1992;23: 1583–1587.
- Spencer MP. Detection of cerebral arterial emboli with transcranial Doppler. In: Newell DW, Aaslid R, eds. *Transcranial Doppler*. New Haven, NY: Raven Press Ltd; 1992:215–230.
- Consensus Committee of the Ninth International Cerebral Hemodynamics Symposium. Basic identification criteria of Doppler microembolic signals. *Stroke*. 1995;26:1123.
- Berguer R, Sieggreen MY, Lazo A, Hodakowski GT. The silent brain infarct in carotid surgery. J Vasc Surg. 1986;3:442–447.
- Street DL, O'Brien MS, Ricotta JJ, Ekholm SE, Ouriel K, Green RM, DeWeese JA. Observations on cerebral computed tomography in patients having carotid endarterectomy. *J Vasc Surg.* 1988;7:798–801.
- Sise MJ, Sedwitz MM, Rowley WR, Shakford SR. Prospective analysis of carotid endarterectomy and silent cerebral infarction in 97 patients. *Stroke*. 1989;20:329–332.
- Habozit B. The silent brain infarct before and after carotid surgery. Ann Vasc Surg. 1990;4:485–489.

- Alimi Y, Kalle K, Poncet M, Fabre D, Doddoli C, Barthelemy P, Juhan C. Silent brain infarct after carotid artery surgery: incidence and prevention. *Ann Vasc Surg.* 1995;9(suppl):S76–S80.
- 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.
- Gossetti B, Martinelli O, Guerricchio R, Irace L, Benedetti-Valentini F. Transcranial Doppler in 178 patients before, during, and after carotid endarterectomy. *J Neuroimaging*. 1997;7:213–216.
- 35. Sundt TM Jr, Houser OW, Sharbrough FW, Messick JM Jr. Carotid endarterectomy: results, complications, and monitoring techniques. In: Thompson RA, Green JR, eds. Advances in Neurology. New York, NY: Raven Press Publishers; 1977;16:97–119.
- Naylor AR, Bolia A, Abbott RJ, Pye IF, Smith J, Lennard N, Lloyd AJ, London NJ, Bell PR. Randomized study of carotid angioplasty and stenting versus carotid endarterectomy: a stopped trial. J Vasc Surg. 1998;28:326–334.
- Jordan WD Jr, Voellinger DC, Fisher WS, Redden D, McDowell HA. A comparison of carotid angioplasty with stenting versus endarterectomy with regional anesthesia. J Vasc Surg. 1998;28:397–402.
- Henry M, Amor M, Masson I, Henry I, Tzvetanov K, Chati Z, Khanna N. Angioplasty and stenting of the extracranial carotid arteries. *J Endovasc Surg.* 1998;5:293–304.
- Abrahamsen J, Roeder OC, Justesen P, Enevoldsen E. Percutaneous transluminal angioplasty in selected patients with severe carotid artery stenoses: the results of a consecutive series of 24 patients. *Eur J Vasc Endovasc Surg.* 1998;16:438–442.
- Chastain HD II, Gomez CR, Iyer S, Roubin GS, Vitek JJ, Terry JB, Levine RL. Influence of age upon complications of carotid artery stenting: UAB Neurovascular Angioplasty Team. *J Endovasc Surg.* 1999; 6:217–222.
- Leisch F, Kerschner K, Hofman R, Bibl D, Engleder C, Bergmann H. Carotid stenting: acute results and complications. Z Kardiol. 1999;88: 661–668.
- Qureshi AI, Luft AR, Janardhan V, Suri MFK, 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.
- Jordan WD Jr, Voellinger DC, Doblar DD, Plyushcheva NP, Fisher WS, McDowell HA. Microemboli detected by transcranial Doppler monitoring in patients during carotid angioplasty versus carotid endarterectomy. *Cardiovasc Surg.* 1999;7:33–38.
- Mericle RA, Kim SH, Lanzino G, Lopes DK, Wakhloo AK, Guterman LR, Hopkins LN. Carotid artery angioplasty and use of stents in high-risk patients with contralateral occlusions. *J Neurosurg*. 1999;90:1031–1036.
- Lanzino G, Mericle RA, Lopes DK, Wakhloo AK, Guterman LR, Hopkins LN. Percutaneous transluminal angioplasty and stent placement for recurrent carotid artery stenosis. *J Neurosurg*. 1999;90:688–694.
- 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.
- Lövblad KO, Plüschke W, Remonda L, Gruber-Wiest D, Do DD, Barth A, Kniemeyer HW, Bassetti C, Mattle HP, Schroth G. Diffusion-weighted MRI for monitoring neurovascular interventions. *Neuroradiology*. 2000; 42:134–138.