Carotid Endarterectomy With Transcranial Doppler and Electroencephalographic Monitoring
A Prospective Study in 130 Operations

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Background and Purpose: We report the results of combined recording of hemodynamic and thromboembolic phenomena during carotid endarterectomy by means of computerized electroencephalography as well as transcranial Doppler ultrasonography. The study focuses on the additional value of transcranial Doppler to detect ischemia during surgery.

Methods: Combined monitoring was performed in 130 consecutive operations, using standard anesthesiological, surgical, and neurophysiological procedures.

Results: A reduction of \( \geq 70\% \) of blood flow velocities in the middle cerebral artery during cross-clamping was measured in 16 patients. In seven of these cases there were no severe electroencephalographic changes and a shunt was not used, but one of the patients developed a subcortical infarct with slight disability. In 55 patients, 75 episodes of embolization were detected by transcranial Doppler. In one of these, with massive embolization after release of the clamp, an intraoperative stroke occurred without changes on cranial computerized tomography or neurological disability on follow-up. In the other 54 patients, intraoperative embolization did not cause clinical or neuroradiological symptoms. Electroencephalographic changes occurred in only two of the 75 episodes. In addition to the two nondisabling strokes during surgery (1.5%), six strokes occurred within 5 days of operation, including one hemorrhage. There was no significant relation between contralateral carotid occlusion and stroke \((p=0.6)\).

Conclusions: During carotid endarterectomy, transcranial Doppler immediately provides information about thromboembolism and hemodynamic changes that are not detected by electroencephalography alone. Acoustic feedback from the transcranial Doppler monitoring unit has a direct influence on the surgical technique. Transcranial Doppler ultrasound may be a useful tool in the study and prevention of intraoperative stroke. \(Stroke, 1993;24:665-669\)

Key Words • carotid endarterectomy • Doppler • electroencephalography • ultrasonics

During the past 25 years carotid endarterectomy was the subject of much debate until the collaborators of the European Carotid Surgery Trial and the North American Symptomatic Carotid Endarterectomy Trial recently published the interim results of their studies.\(^1,2\) They showed that patients who suffered transient ischemic attacks or minor stroke and atherosclerosis of the ipsilateral carotid artery with a stenosis of \( \geq 70\% \) linear reduction substantially benefited from the operation. In the future, patients who fulfill these criteria should be offered carotid endarterectomy.

Efforts have been made to reduce the number of complications of carotid surgery, but comparison of perioperative stroke rates is difficult because they depend not only on operation technique but also on the selection of patients and the definition of complications.\(^3-5\) Moreover, studies evaluating the operative morbidity and mortality of carotid endarterectomy are usually retrospective, and the differentiation between intraoperative and postoperative strokes is often unclear. Most authors describe a neurological deficit in relation to carotid surgery as perioperative. A careful evaluation of the risks and benefits of carotid endarterectomy should separately consider the rate of intraoperative stroke.

In previous studies, we evaluated the reliability of cerebral function monitoring during carotid surgery with an electroencephalographic (EEG) expert system.\(^6,7\) In these studies, 68% of perioperative strokes occurred intraoperatively; 50% of these intraoperative strokes were probably caused by hemodynamic factors. This proportion is significantly higher than in spontaneously occurring stroke.\(^8\) The remaining half of intraoperative strokes was probably of thromboembolic origin and primarily occurred during surgical manipulation of the carotid arteries. Nondisabling cerebral in-
farcts in the subcortical region or in the internal capsule were not detected by the EEG expert system. Recently, Russel et al.9 and Spencer et al.10 demonstrated that transcranial Doppler (TCD) ultrasonography is very sensitive in detecting microemboli. To more reliably monitor cerebral function during carotid endarterectomy and also to record evidence of cerebral embolization, we introduced TCD registration of the blood flow velocities of the ipsilateral middle cerebral artery as an addition to the EEG expert system. The aims of this study were to investigate whether TCD is a useful tool for the detection of cerebral embolization, to assess our current criteria for the selective use of a shunt, and finally, to estimate if the simultaneous use of EEG and TCD monitoring might reduce the number of intraoperative strokes.

Subjects and Methods
From January 1990 until July 1991 we studied 130 consecutive carotid endarterectomies performed in 105 men and 25 women (mean age, 64 years; range, 43–82 years). Eighty-two patients (63%) had recent carotid symptoms (amaurosis fugax, transient ischemic attack, or nondisabling stroke). Forty-eight patients (37%) were operated on for asymptomatic stenosis, 20 of them in combination with a coronary bypass procedure. All patients underwent neurological examination before and after the operation, as soon as the level of consciousness allowed a reliable examination of neurological function. As in the earlier study,6 when the patients exhibited the same preoperative neurological status the procedure was considered uncomplicated. New deficits corresponding to the side of the operation were recorded as intraoperative stroke, and complications that developed after the first postoperative examination were classified as postoperative. A distinction was made between disabling strokes (disability exceeding grade 3 on the modified Rankin scale11 after 2 weeks) and nondisabling strokes. All patients underwent preoperative duplex scanning and subtraction angiography of both carotid and vertebral arteries. All patients had cranial computed tomography (CT) before the operation. When intraoperative embolization was detected or clinical deficits occurred, a CT scan was performed between the third and fifth postoperative day. In case of doubt, a magnetic resonance (MR) scan was performed additionally. Anesthesia was given with a standardized regimen, using nitrous oxide and halothane 0.25–0.50% to avoid a depth of anesthesia that would interfere with reliable EEG monitoring.

The intraoperative EEG monitoring system provides automatic acquisition and recording of the EEG as well as data concerning blood pressure, anesthetics used, and the impact of cross-clamping on cerebral function. Relevant parameters are extracted from these data, and abnormal activity results in automatic alarm. Asymmetry between the hemispheres is continuously calculated by comparison of the number of zero-crossings of the EEG signal on both sides and is numerically displayed. Asymmetry >15% was considered severe enough to justify the use of a shunt (Javid-Bard shunt).

For the TCD registration we used a pulsed Doppler transducer (EME TC 2-64), gated at a focal depth of 45–60 mm and placed over the temporal bone to insonate the main stem of the middle cerebral artery. The TCD transducer was fixed with a head strap. Ipsilateral carotid artery compression was performed to ensure the correct insonation of the middle cerebral artery. The Doppler audio signal was made audible in the operating theater throughout the entire procedure. Special attention was devoted to changes in blood flow velocities when cross-clamping the common carotid and internal carotid arteries as well as after release of the clamps.

We also took great interest in embolization signals and registered the number and duration of such events on the TCD monitor. According to Spencer,12 embolization signals must be defined as short transients, <0.1 seconds, ranging 3–60 dB above the background Doppler blood velocity spectrum. They are unidirectional, their duration in the spectrum is inversely proportional to their velocity, and they occur at random within the cardiac cycle. Probably the most important characteristic is that they sound like harmonic chirps, whistles, or clicks, depending on velocity, making emboli easily recognizable to the listener.

A standardized surgical procedure was applied in all cases. After dissection of the artery and intravenous heparinization, three cross-clamps were applied to the common, internal, and external carotid arteries. In cases of severe EEG asymmetry a Javid shunt was inserted and a classic endarterectomy was performed. When EEG asymmetry was <15%, endarterectomy was performed without the use of a shunt. The reduction of blood flow velocities in the middle cerebral artery was not used as a criterion to shunt. After endarterectomy the artery was flushed with heparin solution to remove emboli and subsequently sutured. The external and common carotid arteries were unclamped before the internal carotid artery to remove residual emboli via the extracerebral circulation.

Continuous video recording of the spectral waveforms and audio Doppler signals was made from the moment that the patient was intubated until the end of anesthesia. Peak and mean blood flow velocities in the ipsilateral middle cerebral artery, blood pressure, anesthetics used, and surgical manipulations such as dissection of the vessel, cross-clamping, and removal of the clamps were recorded on a standard list. The tapes were reviewed immediately after surgery in cases of concern.

Results
Eighty-one patients had a carotid stenosis of 90–99%, 34 had a stenosis of 65–90%, and 15 had a stenosis of <65%. Twenty-five patients had an occluded contralateral internal carotid artery. Twelve patients had a significant (>50%) stenosis of one subclavian artery, and eight patients showed a significant stenosis (>50%) of one vertebral artery. In eight patients one vertebral artery was occluded.

TCD monitoring was technically possible throughout the entire operation in 123 cases (95%). Of the seven patients in whom TCD monitoring was not feasible, five did not have a suitable temporal window. In two patients TCD recording was not possible for practical reasons. The most important TCD findings have been divided into hemodynamic and thromboembolic phenomena for the sake of clarification.
**Hemodynamic Changes**

Sixteen patients had a reduction of blood flow velocities of ≥70% at cross-clamping. Nine of these patients developed a severe (>15%) EEG asymmetry; a shunt was used in eight of them. The remaining patient with a >70% reduction of blood flow velocities and a severe EEG asymmetry underwent carotid endarterectomy and a coronary bypass procedure simultaneously. The introduction of a shunt was not possible for technical reasons, and therefore the endarterectomy was performed during deep cooling. Seven patients who showed ≥70% reduction of blood flow velocities at cross-clamping had no severe asymmetry of the EEG and were not shunted. Two of them developed only moderate (10–15%) asymmetry, and one of these two was later found to have a subcortical infarct (patient A, see below); five patients showed no asymmetry. Surprisingly, in two of these five patients the TCD signal completely disappeared on cross-clamping. Conversely, one patient had a blood flow velocity reduction of 26% on cross-clamping and yet developed severe EEG asymmetry; this patient was shunted. In eight of the remaining 106 patients a shunt was used because of recent cerebral infarction, and in one additional case a shunt was used because of accidental intra-arterial injection of lidocaine, administered in an attempt to block the baroreceptors in the glomus caroticum. This gave rise to diffuse and asymmetrical slow-wave activity on the EEG and raised the possibility of a dissection caused by this maneuver. In all shunted patients the blood flow velocities were immediately restored to the preclamp level.

The TCD provided immediate information regarding the correct functioning of the shunt.

One of the shunted patients had a complete attenuation of the TCD signal during test clamping. After release of the clamps, the TCD displayed high blood flow velocities of >150% of the preoperative blood flow velocity values. The blood pressure was unstable, ranging from 120/75 mm Hg to 220/130 mm Hg, and 5 days later the patient developed an intracerebral hematoma in a previous infarct.

**Thromboembolism**

In the 123 successfully performed TCD registrations, embolization was detected in a total of 75 episodes in 55 patients (45%). Embolization signals varied from one single embolus during insertion of a needle for stump pressure measurement to massive embolization for 5 minutes after release of the clamps. Embolization during manipulation of the artery before cross-clamping was observed in 10 cases. In these cases, the duration of embolization was usually short (one single signal or total duration <10 seconds). Embolization after endarterectomy and release of the clamp was detected in 39 cases and often lasted longer (in two cases >20 seconds, and in one case as long as 5 minutes). Emboli were detected in five cases when fluid was introduced into the blood stream: lidocaine, heparin (in two cases), and radiopaque contrast medium (in two cases). In eight cases embolization was detected on release of the external carotid artery clamp. In three cases the use of a shunt caused embolization (19% of all shunts), primarily during the introduction of the shunt. Embolization was detected six times immediately after introduction of a needle for stump pressure measurement. One episode of embolization was detected after the operation, when the nurse pressed an adhesive plaster on the wound, and in another case several emboli were detected when the patient was tilted from the operating table. Two short signals indicated spontaneous occurrence of emboli, without manipulation of the clamps, the vessel, or the patient.

Embolization was followed by clinical deficits in only one case (patient B, see below). In this case, the EEG did not show asymmetry. In the case in which accidental injection of lidocaine into the blood stream occurred, TCD showed embolization signals, and in the EEG, transient bilateral slow-wave activity was seen with an asymmetry of >15%. Another patient with relatively massive embolization on TCD showed a similar transient diffuse and severe EEG asymmetry. Neither patient developed neurological deficits. No other cases with cerebral embolization were detected by the EEG expert system.

**Complications**

Two patients developed an ischemic complication during operation. In both cases the stroke was nondisabling. In the first patient (A), the blood flow velocities of the middle cerebral artery diminished by 75% on cross-clamping of the internal carotid artery. The endarterectomy was completed without shunting because the EEG asymmetry was judged not severe enough to warrant the use of a shunt (asymmetry 10–12%). On first neurological examination we found motor dysphasia, which completely disappeared within 2 weeks. CT and MR scanning showed two small subcortical infarcts in the left insular region (Figure 1). In the second patient (B), EEG and TCD demonstrated no hemodynamic insufficiency on cross-clamping, and the endarterectomy was performed without the use of a shunt. On release of the internal carotid artery clamp, massive embolization occurred for 5 minutes. Weakness of the contralateral arm was found on postoperative examination. CT scanning after 3 days showed no changes. The patient made a complete recovery within 14 days.

Five patients developed ischemic complications postoperatively, with intervals ranging between 1 hour and 5 days. After 2 weeks, two of these strokes were classified as disabling and three as nondisabling. In four of the five patients a postoperative CT scan showed a fresh infarct in a border zone between two vascular territories, suggesting a hemodynamic origin, and in one patient a small subcortical infarct in the territory of the middle cerebral artery was detected. In three patients an occlusion of the operated vessel was demonstrated with duplex scanning and digital subtraction angiography 12–72 hours after surgery.

One of these seven intraoperative and postoperative strokes occurred in a patient with a contralateral carotid occlusion (n=25), and six occurred in the remaining 105 patients with a patent contralateral carotid artery.

The intraoperative stroke rate in this series is 1.5% (95% confidence interval, 0.2–5.5%). The postoperative stroke rate, including one patient with a hematoma in a previous infarct, is 4.6% (95% confidence interval, 1.7–9.8%). This results in an overall perioperative stroke rate of 6.1% (95% confidence interval, 2.7–11.8%). There was no patient mortality.
Discussion

TCD monitoring combined with the EEG expert system during carotid endarterectomy provided additional insight into hemodynamic changes and unique information about intraoperative embolization. The TCD registration was technically successful in the vast majority (95%) of the patients and did not interfere with the anesthesiological or surgical procedures.

Cerebral embolization was detected in 45% (55 of 123) of all operations. During dissection of the carotid bifurcation, microemboli usually resulted in very short artifacts (<0.2 seconds) in the Doppler signal, which were almost certainly caused by formed elements that were dislodged from the vessel wall. We detected this in 10 cases (8%).

Emboli recorded on release of the clamps probably consisted of a combination of small air bubbles and formed elements, such as platelet aggregates and atheromatous debris. In these cases, the embolization signals usually lasted longer, in three cases >20 seconds. We observed this type of embolization to the brain in 39 cases (32%) of all patients. Only one of these patients developed an intraoperative nondisabling stroke after a shower of microemboli lasting 5 minutes on release of the clamp of the internal carotid artery, without changes on the postoperative CT scan. The remaining 54 patients with evidence of cerebral embolization on TCD did not show signs of cerebral ischemia either on clinical examination or on the postoperative CT scan. In only two cases, both with several hundreds of emboli, the EEG showed transient but diffuse slow-wave activity with a maximum of delta activity in the ipsilateral hemisphere. The other episodes of cerebral embolization were not detected by the EEG expert system. In eight cases emboli were detected in the middle cerebral artery on release of the clamp of the external carotid artery and before the internal carotid artery was unclamped. In these cases, the emboli probably entered the middle cerebral artery via the transorbital collateral circulation. The risk of dislodging emboli by insertion of a shunt was found to be low (19%). The occurrence of emboli during stump pressure measurements should alert the surgeon to this danger. The impact of this measurement on the surgical procedure must be weighed against the risk of embolization. The introduction of emboli on injection of different types of fluids did not cause severe ischemia. It is apparent that a fluid-filled syringe always contains small air bubbles, even after meticulous desufflation.

TCD monitoring also detected hemodynamic changes that were not reflected by severe abnormalities of the EEG (five of these seven cases had a normal EEG, two had a moderate asymmetry). In these instances a shunt was not inserted. That the EEG remains (nearly) normal despite very low velocities in the middle cerebral artery probably reflects perfusion of the cerebral cortex via leptomeningeal anastomoses. However, in one of these patients infarction occurred in the subcortical white matter. In this patient with severe multivessel disease (ipsilateral carotid artery 90–99% stenosis, contralateral carotid artery 50–65% stenosis, contralateral subclavian artery 50–80% stenosis, and contralateral vertebral artery >50% stenosis) the blood flow velocities in the middle cerebral artery decreased by 75%, while the EEG revealed a moderate asymmetry of 10–12%. Because the combination of TCD and EEG monitoring assesses both the blood flow velocities in the main stem of the middle cerebral artery and the perfusion of the cortex of the brain, the TCD data should be taken into account when decisions are made regarding the selective use of a shunt. Our study did not reveal silent brain infarcts, in contrast to other reports. In the group with a contralateral carotid artery occlusion (n=25), one patient developed a stroke. This stroke rate was not significantly different from that of the patients with a patent contralateral carotid artery (p=0.6, Fisher’s exact test).

We conclude that TCD monitoring during carotid endarterectomy can be performed successfully, provides additional information regarding the cerebral circulation during operation, and will, therefore, lead to a better understanding and prevention of intraoperative stroke. The direct acoustic feedback to the surgeon also provides instantaneous information. In regard to embolization, it has been our experience that the surgeon has been guided by these signals and that an attempt was made to avoid embolization in this way, which may have made the operation safer. After the addition of TCD
monitoring to our EEG expert system, the intraoperative stroke rate has decreased. In our earlier series of 230 carotid endarterectomies, 11 (4.8%) intraoperative strokes occurred (five disabling and six nondisabling). In the present series of 130 carotid endarterectomies, only two (1.5%) nondisabling intraoperative strokes occurred. Not one disabling stroke took place.

Because this comparison involved two consecutive series without concurrent and randomized controls, we cannot prove that TCD monitoring made the operation safer because the possibility that other factors were also responsible for this change must be taken into account. However, we have not been able to identify such factors.

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