Detection of Middle Cerebral Artery Emboli During Carotid Endarterectomy Using Transcranial Doppler Ultrasonography

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The purpose of our study was to define the signal characteristics and clinical circumstances associated with emboli detected in the middle cerebral artery using 2-MHz pulsed transcranial Doppler ultrasound in patients undergoing carotid endarterectomy. Signals designating emboli were transients displaying harmonic qualities the signatures of which were clearly different from those of mechanical and electronic artifacts. We reviewed the audio/video tape recordings from 91 patients for signals of air bubble emboli occurring upon release of common carotid artery crossclamps; recordings from 35 patients (38%) demonstrated air bubble emboli. Transients with signatures identical to those of air bubble emboli were also discovered when bubbles in the bloodstream were improbable; we defined these transients as representing formed-element emboli. Such signals were found in recordings from 24 patients (26%), and they occurred before (both spontaneously and upon common carotid artery compression), during, and after surgical dissection. Signals indicating formed-element emboli were associated with intraluminal platelet thrombus, with ulcerations in the carotid artery, and with transient ischemic attacks or stroke. Most postoperative formed-element emboli did not cause symptoms but, when persisting for hours, they were associated with strokes and cerebral infarction. This Doppler ultrasound method of detecting emboli will be useful in the study of stroke mechanisms and as a clinical test to guide the medical and surgical treatment of patients at risk of stroke. (Stroke 1990;21:415-423)

It is generally recognized that arterial embolism from the heart or a carotid artery to the brain represents a major mechanism for stroke. Atherosclerotic plaque in the carotid arteries can produce emboli by two mechanisms, by the rupture of its contents into the blood stream and by the breaking off of a thrombus formed on an ulcerated surface or in the blood stream when flow distal to the plaque is slowed.

A method that detects the passing of emboli into the brain would be valuable in the study of stroke mechanisms, and if sufficiently sensitive such a method might be useful as an early warning system heralding the advent of transient ischemic attacks (TIAs), brain infarction, and stroke. The use of Doppler ultrasound in detecting gas emboli in the blood was developed in 1968 and has been used as a practical early warning system in the study of decompression sickness as well as in the development of safer decompression tables for divers and caisson workers. Gas emboli produce characteristic signals because of their high reflectivity and large size compared with blood cells.

Padayachee et al noted that 17 of 19 patients monitored with transcranial Doppler ultrasound (TCD) during carotid endarterectomy had Doppler signals of abnormally high amplitudes upon commencement of shunting. The authors ascribed these high-amplitude signals to turbulent blood flow or microscopic air bubbles introduced when the shunt was inserted and observed that such signals had no correlation with adverse effects in the patients.

Our experiences in the detection of decompression gas emboli and the use of TCD for intraoperative monitoring during surgical manipulations of the carotid artery have led to many observations of Doppler signals associated with the introduction of bubbles into the artery and with circumstances producing conglomerates of formed elements not gaseous in nature. We relate our methods, experiences,
and conclusions using TCD in the detection of signals of arterial emboli in patients undergoing carotid endarterectomy. No hypothesis is tested here; rather, we report our discovery of a new technique for the detection and study of arterial emboli and provide preliminary observations on their clinical correlates.

**Subjects and Methods**

We used 2-MHz pulsed-wave Doppler equipment with a sample length of 18 mm, gated to focal depths of 4.5-5.5 cm. The probe was placed over the temporal bone to insonate the middle cerebral artery (MCA) or its branches ipsilateral to the operated carotid artery. The diameter of the sample volume, determined by the focus pattern of the probe and an unknown degree of distortion by the temporal bone, was estimated to be >2 mm to include an entire segment of the MCA. A specially designed headgear fixed the probe to the patient's head with provisions for adjusting the probe's position and angle.

We monitored 196 patients with high degrees of stenosis of the internal carotid artery (ICA), either with cerebrovascular symptoms or asymptomatic, during carotid endarterectomy. Continuous recordings of Doppler fast Fourier-transform (FFT) spectra, as well as separate audio signals of Doppler ultrasound and of voice annotations, were made using a videotape recorder with dual audio channels. Usually, the recorder ran continuously from the time the surgeon exposed the carotid artery until closure of the arteriotomy. Recordings were also occasionally made the day before surgery, the day of surgery before induction of anesthesia, and in the recovery room of the operating suites. Occasionally, recordings were made while manual compression of the common carotid artery (CCA) was performed before surgery to assess the availability of collateral blood flow.

Tape recordings from 91 patients were satisfactory for the analysis of signals indicating bubble emboli occurring upon release of the carotid crossclamps after arteriotomy closure; tape recordings from 63 of these 91 patients were also searched for signals of emboli before and after surgery. Because of time and technical constraints, not all tape recordings could be analyzed. Also, before it became clear that formed-element emboli could be detected, complete and extended monitoring was not employed. Most of our positive findings came to light recently, when more complete recordings were available. Even so, it was not possible to perform all the monitoring needed because of the urgent schedule during which most of these patients were available for study. Doppler signals were analyzed off-line by replaying the videotapes and reconstructing the FFT frequency spectra from the Doppler audio channel. Tape recordings from 18 patients in whom embolization was suspected or in whom an ulcer was reported by the surgeon were also analyzed. The relative signal amplitudes were determined by color spectral analysis, which displayed four levels separated by 10 dB.

**Results**

All signals analyzed 1) were transient (lasting 0.01-0.1 second), 2) occurred randomly, and 3) had amplitudes ≥10 dB greater than the background Doppler signal. To the ear, signals defined as indicating emboli were harmonic in tone, with a chirping or whistling quality and distinctly different from those indicating probe motion artifacts. Probe motion artifacts did not have a harmonic quality, were coincident with motion of the probe or electronic switching, and had a noisy "banging" quality with a broad frequency spectrum, the highest energies of which extended to the lowest frequency ranges (Figure 1). Probe motion artifacts were invariably bidirectional.

Bubble emboli signals were the most easily recognized (Figure 2) by their qualities similar to those of decompression bubbles as well as by correlating with an iatrogenic source. Bubble emboli signals had very high amplitudes (up to 60 dB greater than the background Doppler signal). Their harmonic quality correlated with a relatively narrow spectrum that, however, changed in frequency during the time displayed. When occurring during diastole, bubble emboli signals had a lower frequency and lasted longer than when occurring during systole (Figure 3).

Bubble emboli signals were always associated with circumstances requiring invasion of the artery wall, predominantly release of carotid crossclamps after arteriotomy closure. Puncture of an artery with a needle for measuring pressure or manipulations for inserting a shunt from the CCA to the distal ICA also produced bubble emboli signals. The signals occurred at their greatest frequency and amplitude immediately following the release of carotid crossclamps and then randomly tapered off to no further signals within 2 minutes (Figure 4). Of 91 patients undergoing carotid endarterectomy in whom observations were made immediately upon opening of the carotid crossclamps, 57 had bubble emboli signals. The bubble emboli could not always be counted individually because of superposition, with many signals appearing within the Doppler spectrum at the same time or in rapid succession. The number of bubbles was, however, estimated by the cumulative duration of signals after crossclamp release. Thirty-five patients (38%) displayed no bubble emboli signals; 14 (15%) displayed 0.1-0.5, 11 (12%) displayed 0.5-1, 18 (20%) displayed 1-5, and 13 (14%) displayed 5-25 cumulative seconds of bubble emboli signals. In seven patients, bubble emboli signals occurred in the MCA after the external carotid artery (ECA) was unclamped and before the ICA was unclamped. The bubble emboli presumably passed retrograde through the ophthalmic artery to the circle of Willis and its distribution. After the bubbles passed through the ECA, opening of the ICA distributed more to the MCA territory.

In only one patient were postoperative cerebrovascular symptoms associated with bubble emboli sig-
nals. Fourteen cumulative seconds of bubble emboli signals passed through the ECA to the MCA before the ICA crossclamp was removed. No emboli signals were detected before arteriotomy or during two subsequent TCD examinations (one in the recovery room and the other 1 day after surgery). Stump pressure and blood velocities were adequate during crossclamping without shunting (49 mm Hg and 44/22 cm/sec, respectively). This patient's symptoms consisted of dysphasia for 24 hours and left hemiparesis that gradually improved over the 1 week of hospitalization. Upon discharge, the patient was walking and had no special complaints.

Formed-element emboli signals were recognized by the same harmonic, chirping qualities as bubble emboli signals but had lower amplitudes (≤40 dB greater than the Doppler background signal). The spectral distribution was similar to that of bubble emboli signals (Figure 5); however, formed-element emboli signals occurred either before invasion of the
FIGURE 3. Transcranial Doppler spectrograms. Top: Bubble embolus signal passing through right middle cerebral artery (RMCA) during late systole after crossclamp (XCL) release, displaying short duration consistent with high blood velocity and ultrasonic sample length. Bottom: Bubble embolus signal passing through RMCA during diastole after XCL release, with corresponding lower velocity and longer duration. Some aliasing of bubble signals is seen in both panels due to high amplitude, approximately 40 dB above blood flow velocity amplitudes. Horizontal white line at top of each panel, 100 cm/sec or 2.5 kHz; time base, 1 second; CLV, patient file designation.

artery wall or several hours after arteriotomy closure and were always discrete and seldom close to one another. Formed-element emboli signals were recognized when no transient artifact signals were present and were found in tape recordings from 24 of the 91 patients examined for bubble emboli signals.

Eleven tape recordings disclosed formed-element emboli signals during the preoperative checkout or during dissection of the carotid arteries before arteriotomy. When more than one signal was identified, an ulceration was invariably found at the carotid bifurcation. If signals occurred during surgical manipulations of the carotid arteries, the surgeon was alerted; the signals ceased when the manipulation stopped.

Formed-element emboli signals were observed in two patients during CCA compression (Figure 6). No cerebrovascular symptoms were noted after showers of fewer than six such signals. Among the 11 patients with formed-element emboli signals detected before arteriotomy, 10 presented with either a TIA or a stroke appropriate to the operated/monitored side. The other patient was asymptomatic and was oper-
ated on because of a 75% diameter stenosis of the ICA with poor collateral circulation.

Patient 190, who presented with attacks of amaurosis fugax, displayed multiple formed-element emboli signals in the MCA on the symptomatic side both the day before and immediately before surgery. The ipsilateral ICA was occluded, and there was retrograde blood flow in the ophthalmic artery. Upon opening of the carotid bifurcation, two deep ulcerations were found, one at the end of the CCA and the other at the origin of the stenotic ECA. This patient illustrates that not only can formed-element emboli be associated with amaurosis fugax, but they also can pass to the MCA territory through branches of the ECA from ulcerations at the bifurcation of the CCA.

Formed-element emboli signals were detected during extended postoperative periods in two patients, (105 and 41), both of whom sustained severe strokes.

Patient 105 underwent a left carotid endarterectomy for an asymptomatic 90% diameter stenosis of
the ICA. Carotid crossclamping in the operating room reduced MCA blood velocity from 78/25 to 50/23 cm/sec and mean stump pressure to 50 mm Hg. Crossclamp time was 26 minutes; no shunt was used. Only 1.5 cumulative seconds of bubble emboli signals were detected in the MCA upon release of the crossclamp. The first signal suspected of representing a formed-element embolus occurred 12 minutes after closure of the arteriotomy. Upon awakening with initially normal function, this patient developed progressive right body weakness and a speech deficit. Formed-element emboli signals were recorded from the MCA, and cervical Doppler ultrasonographic studies indicated that the operated ICA remained patent but also demonstrated formed-element emboli signals (Figure 5). The patient was returned to the operating suite for a second operation, during which continuous TCD monitoring again demonstrated formed-element emboli signals occurring up to the time of the second carotid crossclamping (Figure 7). Upon the second carotid crossclamping, formed-element emboli signals ceased except for one detected approximately 2 seconds after crossclamping. When the CCA was reopened, at the site of the first CCA crossclamping the surgeon found a fractured intima with some dissection and to which a large platelet thrombus was adherent. This lesion was corrected by extending the endarterectomy proximally. The arteriotomy was closed, the crossclamps were removed, and no further formed-element emboli signals were detected during the remainder of the procedure nor during a repeat TCD examination 2 days later. Computed tomography (CT) immediately after surgery revealed only mild cortical atrophy. However, repeat CT 48 hours later revealed a nonhemorrhagic infarct involving the left MCA distribution, including the parietooccipital watershed area but not the basal ganglia. The patient received anticoagulant therapy and began intensive physical therapy. On the day of discharge 5 weeks after surgery, the patient's speech was semifunctional with word-finding difficulties, he had no function in his right upper extremity, and he walked with a cane. It seems clear that this patient's stroke resulted from repeated formed-element emboli arising from the CCA thrombus and passing through the MCA into its distribution. Though no shunt was used, the stroke was probably not due to hypoperfusion because both the stump pressure and the MCA blood velocity indicated adequate perfusion during crossclamping. The possibility that bubble emboli were significant seems unlikely because the cumulative duration of such signals was only 2.3 seconds.

Patient 41 had a 90% diameter stenosis in the right ICA associated with occlusion of the left ICA. Symptoms consisted of lightheadedness, slurred speech, left facial weakness, a film-like effect in the left eye, and one such episodic film-like effect in the right eye. During endarterectomy, a Javid shunt was employed because poor collateral circulation was identified upon release of the carotid crossclamp by both a low stump pressure and a low MCA blood velocity. Paco2 was maintained at 40 mm Hg. Total crossclamp time, including both placement and removal of the shunt, was 5.6 minutes. Cumulative bubble emboli signal time following release of the crossclamp was 3 seconds. Formed-element emboli signals were detected in the MCA soon after closure of the arteriotomy and at each TCD examination in the cardiac care unit for 2 subsequent days. The patient was not returned to surgery because this was our first observation of continuing formed-element emboli signals and their meaning at that time was not clear. This patient sustained a severe bilateral stroke, becoming quadri-
Discussion

Evidence that the transient Doppler signals defined in this study represent emboli is based on both the circumstances associated with their occurrence and on their unique characteristics, which are clearly different from those of Doppler blood flow signals or transient artifacts. All bubble emboli signals were associated with invasions of the carotid arteries. Formed-element emboli were so designated because their signals (otherwise identical to those of bubbles) arose when there was no invasion of the vasculature. It has been shown that both bubble and
formed-element emboli can pass retrograde through the ECA to the MCA if the ICA is occluded.

Calculations from the duration and velocity of the embolic signals (duration in seconds × velocity in centimeters per second) agree well with the known sample length of the ultrasonic pulse. This result is expected if the signals truly represent discrete reflective particles moving through the sample volume (Figure 3).

It is clear that not all detected emboli signals produced symptoms. Even when many bubble signals occurred, stroke was rare. Also, since visual deficits were never noted postoperatively, even when bubble emboli obviously had passed through the ophthalmic artery, the retina must be relatively unaffected by them. These observations, along with animal experiments (unpublished data) demonstrating that arterial gas emboli to the brain can be detected in its venous drainage, indicate that bubble emboli probably pass through the vasculature of the brain. The findings of Padayachee et al agree with ours that air bubble emboli do not necessarily produce symptoms. This observation may encourage the use of bubbles of air or other foreign agents as ultrasonic contrast media.

A few formed-element emboli to the MCA territory can also be sustained without overt symptoms,
but if the emboli persist for several hours after surgery, they can produce a stroke. When more than one embolus was detected before surgery, an ulcerated plaque was found at the carotid bifurcation. It seems possible that these clinically silent emboli (now given a voice by TCD) may be responsible for silent infarctions.  

No doubt the positive result of finding embolic signals will be more useful than the negative result of not finding them. In the future, detection of cerebral emboli with improved monitors, allowing application over more extended periods, may be used clinically for the diagnosis of arterial ulceration or cardiac sources of emboli. Medical therapy may be adjusted according to the incidence of embolic signals, and surgical techniques may be improved by documentation of embolic sources. Arterial and cardiac sources of emboli may be distinguished by simultaneously monitoring the MCA and ascending aorta.  

Other future applications of this technique include detection of embolized material during cementation in hip arthroplasty and detection of embolism from deep vein thrombosis and transcatheter angioplasty.  

In conclusion, the Doppler signatures of bubble emboli and formed-element emboli from thrombus or plaque material are defined, and embolic signals are separated from artifactual transients. This investigation establishes TCD as a sensitive technique for the detection of cerebral emboli and suggests that it has a future as a sensitive detector of embolic sources and as an early warning system for stroke. Monitoring the MCA with TCD provides a means of evaluating techniques for preventing and limiting cerebral embolism. Additional studies are indicated to define further the clinical and pathologic correlates of formed-element emboli.

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References


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