

Transcranial Doppler in the Evaluation of Internal Carotid Artery Dissection

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Background and Purpose A subject with dissection of the internal carotid artery (ICA) may present with a variety of symptoms, from headache to stroke. Thus far, it has not been possible to identify the subset of patients at risk for cerebral ischemia. Because the majority of these ischemic events are secondary to embolic phenomena, we used transcranial Doppler (TCD) evaluation with emboli monitoring to study 17 consecutive patients with ICA dissection treated at Harborview Medical Center, Seattle, Wash. during a 2-year period from 1992 until 1994.

Methods Ten patients with ICA dissection secondary to trauma and seven with spontaneous ICA dissection were diagnosed by carotid angiography and studied by TCD from the time of diagnosis through initiation of therapy. Emboli monitoring was performed in the middle cerebral artery (MCA) ipsilateral to the dissection at the initial evaluation and intermittently thereafter to ensure that the emboli stopped with treatment.

Results Emboli were detected in the MCA distal to the dissection in 10 of 17 patients (59%). Patients with microemboli detected by TCD presented with a stroke (70%) much more frequently than those without emboli (14%) ($P=.0498$). The presence of a pseudoaneurysm did not increase the risk of either microemboli or stroke.

Conclusions We have demonstrated a high incidence of intracranial microemboli in the MCA distal to carotid dissections and a significant correlation between the presence of emboli and stroke. TCD can therefore be used as an adjunctive tool to manage patients with suspected carotid dissection and may prove useful in evaluating the efficacy of treatment in reducing microemboli and subsequent stroke. (*Stroke*. 1996;27:1226-1230.)

Key Words • anticoagulation • dissection • stroke • ultrasonics

Dissection of the ICA, both spontaneous and secondary to trauma, has been found with increasing frequency in recent years.¹⁻⁶ In both types of dissection, a false lumen is created and can result in stenosis, occlusion, or pseudoaneurysm of the involved vessel.^{5,7} The presentation of carotid dissection may vary from symptoms as mild as headache, oculosympathetic paresis, and cranial nerve palsies to ischemic symptoms resulting in TIAs and strokes. Hemispheric symptoms may be due to low-flow states after stenosis or occlusion of the ICA; however, most symptoms are likely due to emboli originating in the damaged vessel. Thus, it is important to determine the efficacy of the treatment in eliminating emboli. The morbidity associated with cerebral ischemia is high, and therefore early diagnosis and treatment of carotid dissection are critical. Current treatment options include anticoagulation, antiplatelet therapy, and occasionally surgery when symptoms are refractory to medical therapy.⁴

Carotid angiography is the diagnostic procedure of choice to establish the diagnosis, although MRI, MR angiography, and ultrasound are increasingly used.⁸⁻¹⁰ None of these studies, however, can identify those patients at risk for an ischemic event. In this report we describe 17 patients in whom TCD examination and emboli monitor-

ing were used to aid in the evaluation of carotid dissection. The potential role of emboli monitoring in guiding treatment is discussed.

Subjects and Methods

Seventeen patients with dissection of the ICA were diagnosed and treated at Harborview Medical Center in Seattle, Wash. between January 1992 and January 1994. Medical records, angiograms, CT scans, and TCD studies were reviewed.

All patients underwent aortic arch and three- or four-vessel cerebral angiography on average 3.5 days after presentation of the dissection. The radiological reports as well as the actual films were analyzed. TCD examinations were performed by standard techniques^{11,12} with the use of a 2-MHz Doppler instrument (Transpect Medasonics) (Figure). Systolic, diastolic, and mean BFVs were measured in each vessel. PI was calculated by the following formula: $PI = (\text{Systolic BFV} - \text{Diastolic BFV}) / \text{Mean BFV}$. Emboli monitoring with TCD was performed for 20 to 30 minutes in each MCA ipsilateral to the dissection.^{13,14} High-intensity transient signals were counted on-line by a technician and identified by their characteristic high-frequency chirping sound on the audio output of the TCD equipment and by their specific features on spectral analysis. The embolic pattern is characterized by a very brief (<0.1 second) high-intensity signal occurring randomly through the cardiac cycle. These signals are easily distinguished from noise artifacts that are bidirectional, originate at the baseline, are coincident with movement of the probe, and have a noisy quality on the audio output.¹⁴⁻¹⁶ These signals are subsequently referred to as emboli. The criteria for embolic signals as well as the equipment used remained consistent throughout the study period. Embolic frequency was calculated as the number of emboli per hour. TCD studies were performed in a nonblinded fashion at the time of diagnosis and at regular intervals (1 to 2 days) thereafter with the initiation of therapy. The examinations were discontinued once no emboli were detectable and the patient's clinical status was stable.

The patients were treated with either antiplatelet therapy with ASA or anticoagulation with heparin followed by 3 to 6 months

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Selected Abbreviations and Acronyms
 ASA = acetylsalicylic acid (aspirin)
 BFV = blood flow velocity
 ICA = internal carotid artery
 MCA = middle cerebral artery
 PI = pulsatility index
 TCD = transcranial Doppler ultrasonography
 TIA = transient ischemic attack

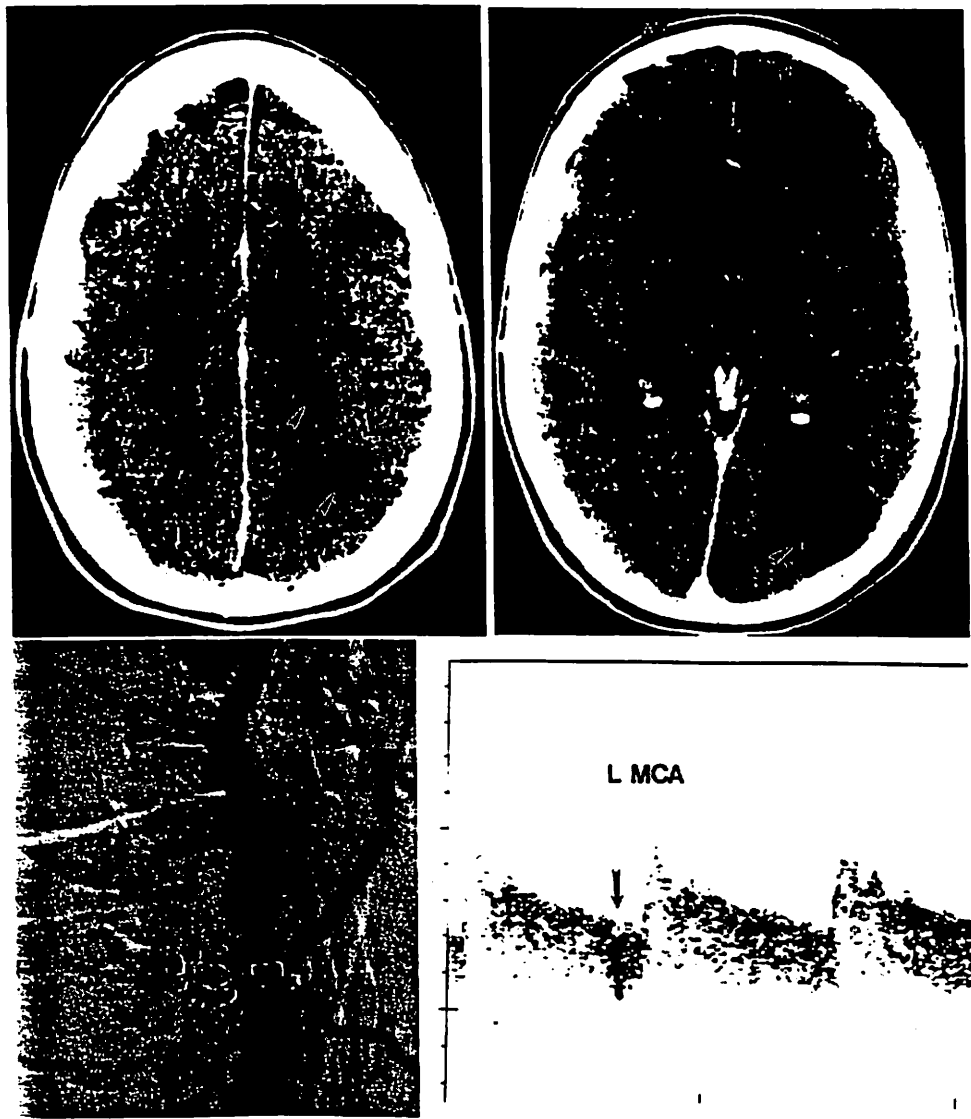
of warfarin. The choice of treatment depended on the clinician's preference, the status of the patient, and whether there were any contraindications to full anticoagulation, such as associated trauma.

Illustrative Cases

Case 1: Traumatic Dissection

A 47-year-old woman was involved in a motor vehicle accident, after which she required a cricothyroidotomy at the scene to establish an open airway. She was unconscious at the scene and subsequently transported to Harborview Medical Center, where she was found to have facial lacerations and abrasions, rib fractures, and long

bone fractures but no spinal injury. She was medically paralyzed at the time of arrival, and CT scan of her head revealed no parenchymal abnormalities. She was taken to the operating room for repair of her extremity fractures and then to the intensive care unit while intubated. On emergence from anesthesia and sedation, she was noted to have a complete left hemiplegia. A second head CT scan revealed multiple small right-sided hypodensities, consistent with embolic strokes. A cerebral angiogram was performed and revealed a right ICA dissection with 70% stenosis of the vessel, a mild right vertebral artery dissection, and branch occlusions of right temporoparietal MCA branches. The patient was begun on heparin and was fully anticoagulated when TCD was performed the next day. TCD examination showed equal velocities in both MCAs with no evidence of intracranial crossover. There were three emboli per hour noted in the right MCA. Over the next several days, the patient was converted from heparin to warfarin and followed with serial TCD examinations. She did not deteriorate clinically, and TCD failed to detect any further emboli 3 days after initiation of anticoagulant treatment. Subsequent monitoring of the right MCA revealed no further emboli.



Spontaneous left ICA dissection in a 37-year-old postpartum woman. Top left and right, Head CT scan with contrast reveals multiple left-sided embolic strokes (arrows). Bottom left, Left common carotid angiogram shows mild extracranial ICA stenosis with a large pseudoaneurysm at the C1-C2 level. Bottom right, TCD examination with emboli monitoring of the left (L) MCA reveals 60 embolic signals per hour (arrow).

Case 2: Spontaneous Dissection

A previously healthy 37-year-old woman underwent cesarean section for delivery of her full-term infant and 10 days later underwent emergency dilatation and curettage for removal of the retained placenta. The following day she developed a right-sided hemiparesis. A CT scan revealed multiple cortical and subcortical hypodensities on the left side. Laboratory investigations for vasculitis and autoimmune disorders were negative, as was an echocardiogram. Carotid duplex revealed high-grade (80% to 99%) left ICA stenosis. She was begun on low-dose heparin, and TCD performed the next day revealed normal intracranial velocities. TCD with emboli monitoring revealed 60 emboli per hour in the left MCA. A heparin infusion was begun, and therapeutic levels were maintained. A carotid angiogram revealed fibromuscular dysplasia of the right ICA and a pseudoaneurysm of the left ICA with a 20% narrowing just proximal to it. The patient remained stable neurologically, and TCD performed 5 days later, with full warfarin anticoagulation, revealed no emboli. She was treated with warfarin for 8 months and then converted to ASA; she had significant recovery of her hemiparesis (Figure).

Results

A total of 17 patients with carotid artery dissection were treated between January 1992 and January 1994. The group included 8 males (47%) and 9 females (53%), ranging in age from 10 to 50 years, with an average age of 31 years (Table 1). The cause of the dissection was traumatic in 10 patients (7 motor vehicle accidents, 2 gunshot wounds, and 1 intraoperative injury) and spontaneous in 7 patients; there was no difference in the sex distributions in the two categories. Of the 9 patients with a traumatic carotid dissection (excluding the intraoperative dissection), 5 also suffered a traumatic brain injury, 6 had facial and/or basilar skull fractures, 1 had a cervical spine fracture, and 1 had cervical soft tissue injury. One patient with a traumatic dissection had no evidence of head or neck injury.

The time from the traumatic event until presentation of the dissection ranged from 0 to 6 days, with an average of 2.1 days. In this group, 5 of 10 patients (50%) presented with a stroke and 2 of 10 (20%) presented with a transient neurological deficit (TIA). Two patients (20%) developed oculosympathetic palsy secondary to the dissection, and in 1 patient the dissection was an incidental finding on an angiogram performed after aneurysm clipping. In the spontaneous group, 2 of 7 patients (29%) presented with TIAs and another 3 of 7 (43%) with stroke. One patient presented with an oculosympathetic palsy, and a single patient presented with a pharyngeal mass without other symptoms. Fifty-seven percent of these patients complained of headaches.

Angiography revealed evidence of dissection in the right ICA in 7 patients, the left ICA in 9 patients, and both ICAs in 1 patient, for a total of 18 arteries. Stenosis was found in 11 of 18 vessels (61%) and occlusion in 5 of 18 vessels (28%); 2 of 18 arteries (11%) had no evidence of narrowing but contained a pseudoaneurysm. Overall, 9 of 18 dissected arteries (50%) contained a pseudoaneurysm: 5 of 10 in the traumatic group and 4 of 8 in the spontaneous group. A single patient in the traumatic group developed a carotid-cavernous fistula secondary to the dissection. In-

TABLE 1. Summary of Findings in 17 Patients With Carotid Dissection

	Traumatic	Spontaneous	Total
No.	10	7	17
Presentation			
Stroke	5	3	8 (47%)
TIA	2	2	4 (24%)
Homer's syndrome	1	1	2 (12%)
Cranial nerve palsy	1	0	1 (6%)
Angiogram*			
Occlusion	3	2	5 (28%)
Stenosis	6	5	11 (61%)
Aneurysm	5	4	9 (50%)
CT scan			
Stroke	6	3	9 (53%)
TCD			
Emboli	6	4	10 (59%)

*n=18 arteries.

tracranial branch occlusions were noted distal to the dissection in 4 cases. Evidence of fibromuscular dysplasia was found in the contralateral carotid artery in 2 of 17 patients (12%): 1 in the traumatic and 1 in the spontaneous group (Table 1).

CT revealed strokes in 9 of 17 patients (53%); these were small infarcts secondary to emboli in 6 of 17 patients (35%) and infarcts secondary to major branch occlusion in 3 of 17 patients (18%). Two patients had evidence only of traumatic brain injury, and 6 of 17 (35%) had normal brain parenchyma by CT scan.

Doppler ultrasound evaluation correctly identified ICA stenosis in 9 of 18 vessels (50%) and occlusion in 5 of 18 vessels (28%). By TCD, the mean PI in the MCA ipsilateral to the dissection was 0.66 compared with 0.91 in the contralateral MCA. The mean MCA velocity was 82 cm/s distal to the injured carotid artery and 83 cm/s in the contralateral MCA. This lowered PI most likely reflects lowered resistance in the vessels distal to the dissection, a change that may not be reflected in the velocity. Evidence of collateral circulation through the circle of Willis was identified in 10 of 17 patients (59%) and was absent in the remaining 7.

Emboli monitoring performed in the MCA distal to the carotid injury revealed emboli in 10 of 18 vessels (56%), ranging from 2 to 60 signals per hour. In the traumatic group, 4 of 10 patients (40%) had emboli on the initial TCD examination, and 2 of 10 (20%) had emboli detected on subsequent monitoring 4 and 5 days later. In the spontaneous group, all 4 of 7 patients (57%) with emboli had them on the first TCD examination. Of the 9 dissected arteries containing a pseudoaneurysm, 5 (56%) had evidence of emboli in the ipsilateral MCA; of the 8 arteries without an aneurysm seen on angiogram, 5 (63%) had emboli documented in the MCA. There was no statistical correlation by χ^2 analysis between the presence of a pseudoaneurysm and either evidence of emboli by TCD examination or stroke. (Table 2). There was also no correlation by χ^2 analysis between the presence of emboli and low PI or low velocity in the ipsilateral MCA.

Seven of the 10 patients (70%) with TCD documentation of emboli presented with a stroke, whereas only 1 of 7 patients (14%) without emboli did so. By Fisher's exact test this result was statistically significant, with

TABLE 2. Summary of Findings in Patients With Embolic Signals on TCD

	Patients With Emboli by TCD		
	Traumatic	Spontaneous	Total
Stroke	4/5	3/3	7/8
TIA	1/2	0/2	1/4
ICA occlusion	2/3	2/2	4/5
ICA stenosis	3/6	3/5	6/11
Pseudoaneurysm	3/5	2/4	5/9

The denominator reflects the total number of patients with each finding; the numerator reflects the same group who also had emboli by TCD.

95% confidence ($P=.0364$ [one-sided] and $P=.0498$ [two-sided]).

A total of 11 of 17 patients (65%) were treated initially with ASA, and 7 of these had embolic phenomena on TCD 1 to 3 days after initiation of therapy. Three patients taking ASA were ultimately changed to heparin because of persistent emboli; of these, 2 were asymptomatic and the third suffered a stroke on ASA. Nine of 17 patients (53%) received heparin followed by warfarin for 3 to 6 months as the definitive treatment. In both ASA and heparin/warfarin treatment groups, emboli monitoring was performed serially until no embolic events were detectable and the patient's clinical status was stable.

Discussion

First described much earlier, more detailed descriptions of carotid dissection were reported by Yamada et al in 1967¹⁷ and Fisher et al in 1978.⁵ Since then, multiple series of patients with both spontaneous and traumatic dissection and their presenting signs and symptoms have been described.^{1,3,18-20} Mokri¹ reviewed 70 patients with spontaneous dissections and found that the most common presenting symptom was headache in 84%, followed by focal cerebral ischemic symptoms (TIA or stroke) in 61% and oculosympathetic paresis in 53%. Watridge et al⁶ described 24 patients diagnosed with traumatic dissections at the time of injury, 12 (50%) of whom developed CT scan evidence of infarction. Although cerebral ischemia is more frequent in traumatic dissections, it can also occur with spontaneous dissections,¹ and those patients who present with cerebral ischemia appear to have greater morbidity.^{1,2,6,21} Bogousslavsky et al²¹ analyzed 1200 patients presenting with stroke, 30 (2.5%) of which were due to carotid dissection. Of these selected patients, the outcome was decidedly worse, with only 40% making a good recovery. Our results, indicating a high incidence of intra-arterial microemboli, support the hypothesis that distal embolism can occur frequently in carotid dissection.

The diagnosis of carotid dissection has typically been made by angiography.^{4,5,7} Common angiographic findings include luminal stenosis, occlusion, pseudoaneurysm, intimal flap, and distal branch occlusion.^{4,5} More recently, MRI and MR angiography have been used to accurately diagnose carotid dissection.^{4,10,22} Duplex sonography can identify carotid dissection when associated stenosis is greater than 50%.⁹ None of these modalities, however, is able to identify patients at risk for cerebral ischemia.

In a prior report in which TCD examination was used, 6 patients with ICA dissection revealed abnormalities in the intracranial circulation, including decreased PI and decreased BFV ipsilateral to the dissection.⁹ Achtereekte et

al²³ also described a patient in whom the diagnosis of dissection was made initially by TCD. We are not aware, however, of any reported uses of TCD for emboli monitoring in carotid dissection in the literature.

In the 17 cases of carotid dissection in this report, TCD was performed to assess the hemodynamics of the intracranial circulation and also to detect embolization from the dissected vessel. The traumatic dissections in our series, like those in the literature, had a high incidence of cerebral ischemia (70%), most commonly stroke. Unlike other series, however, the spontaneous dissections also presented most commonly with ischemia (86%), although the high incidence may reflect the small sample size. The angiographic findings in the present series, including the incidence of pseudoaneurysms, are similar to those described in previous reports.^{1,2,4}

TCD identified hemodynamic changes in the cerebral circulation distal to the dissection as well as the collateral flow pattern to the affected hemisphere. The differences in MCA velocities and PI reflect the varied compensatory responses in the circulation, which depend on change in perfusion pressure above the lesion. These hemodynamic indices are potentially useful in detection of hemodynamic insufficiency secondary to dissection, a rare situation in which surgical therapy (extracranial-intracranial bypass or reconstruction of the artery) may be used. TCD may also be used to follow patients noninvasively to assess resolution of the dissection.

The most valuable aspect of the TCD examination in the present series of patients was the ability to detect and follow microemboli in the MCA distal to a carotid dissection. In normal control subjects, TCD does not detect embolic phenomena.²⁴ After carotid dissection, we found a high incidence of microemboli (59%). We typically monitored patients for 20 to 30 minutes for microemboli. With longer monitoring times, it is possible that more positive studies would have been found; in the future, automated emboli detection systems may provide more accurate data. The presence of emboli by TCD is not only a useful diagnostic sign of carotid dissection but appears to correlate with patients at high risk for stroke and may prove useful in guiding therapy. The mechanism of cerebral ischemia in carotid dissection has been hypothesized to be most frequently embolic in nature.^{1,18} Although the presence of distal branch occlusion on angiography is diagnostic, this finding was present in only 4 of 17 patients. There is a significant correlation between the presence of emboli on TCD and stroke: 70% of patients with TCD evidence of emboli presented with stroke as opposed to 14% of those without. This finding was statistically significant despite the small size, indicating a strong correlation between the presence of microemboli and stroke in these patients.

The definitive therapy for spontaneous or traumatic arterial dissection is not clear. The most common treatment has been heparinization followed by oral anticoagulation with warfarin for 3 to 6 months.^{4,18} Other authors have used antiplatelet therapy, especially in traumatic cases in which there may be a contraindication to anticoagulation.¹ Surgical therapy, including endarterectomy, resection of the injured segment, carotid ligation, and extracranial-intracranial bypass, has been used for patients with hemodynamic insufficiency or persistent ischemia secondary to emboli.^{1,25,26} Because of the increased risk of stroke in patients with TCD microemboli, we advocate anticoagula-

tion with heparin, followed by warfarin, in these patients unless there is a contraindication.

Emboli monitoring was also performed after initiation of anticoagulation or antiplatelet therapy in all patients. Although daily TCD examinations were performed until disappearance of embolic signals was noted, continuous bedside monitoring was not available. From this small series, it appears that ASA is not always adequate therapy to prevent embolization, emphasizing the importance of heparin/warfarin anticoagulation in these patients. In the future, continuous monitoring could potentially be used to adjust the level of anticoagulation until the embolic signals resolved. The optimal duration of treatment is not certain; however, emboli monitoring after cessation of therapy could then be used to ensure that treatment was successful. Also of interest was the lack of correlation between the presence of a pseudoaneurysm and emboli on TCD or stroke. The absence of a pseudoaneurysm may not be a valid reason to omit anticoagulation therapy in the acute setting. Pseudoaneurysms are still the most likely nidus for emboli that cause delayed cerebral ischemia.^{1,27} Adequate follow-up was not available in this study to confirm this. The absence of emboli by TCD does not eliminate the risk of stroke, particularly since the limited examination time may not have been sufficient to detect all microemboli. This source of error may be reduced by multiple, serial exams or continuous monitoring. In a larger series, it may be possible to evaluate this more fully.

In conclusion, although carotid dissection (especially spontaneous) may carry a benign prognosis, the risk of stroke is still significant. TCD is a new modality that can be used as an adjunctive tool in cases of suspected carotid dissection. The value of TCD to monitor therapy requires further investigation. In this initial series, the presence of microemboli by TCD may be a marker for patients at high risk for stroke; given the retrospective nature of this series, it is not possible to determine the true predictive value of this finding. Because our patients were treated with either antiplatelet or anticoagulation therapy, it is unclear what the natural history would have been: One patient treated with ASA had persistent emboli and a subsequent stroke; the emboli resolved with heparin anticoagulation. Thus, given the high incidence of embolic phenomena on TCD, we believe that carotid dissection should be treated aggressively with anticoagulation and that efficacy of treatment should be followed with TCD monitoring. In the future, it may be possible to stratify patients without any embolic signals into a lower risk group that may be treated with ASA. According to this strategy, it may be possible to reduce the incidence of stroke secondary to carotid dissection by early identification of dissections and institution of early treatment, particularly in high-risk patients.

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