Transcranial Doppler: An Overview of its Clinical Applications

A Alexandrov, M Joseph

Citation

A Alexandrov, M Joseph. *Transcranial Doppler: An Overview of its Clinical Applications*. The Internet Journal of Neuromonitoring. 1999 Volume 1 Number 1.

Abstract

INTRODUCTION

Routine transcranial Doppler (TCD) ultrasound examination of the intracranial arteries was demonstrated to be possible in 1982. One fact that has to be constantly kept in mind when utilizing TCD is that the value obtained for a particular artery is the velocity of blood flowing through the vessel, and unless the diameter of that vessel is established by some other means it is not possible to determine the actual blood flow. Thus TCD is primarily a technique for measuring relative changes in flow. The utility of the technique is now well established for a number of different disease processes.

The American Academy of Neurology technology assessment report published in 1990 stated that TCD has established value in the assessment of patients with intracranial stenosis, collaterals, subarachnoid hemorrhage, and brain death. Recently, a panel of international experts critically reviewed the literature published up to 1998 and ranked the specific clinical applications of TCD based on the strength and quality of published evidence (Table 1).

Figure 1

Table 1: Specific clinical applications of TCD

Applications	Rating	Evidence	
		Quality	Strength
Sickle Cell Disease	Effective	Class I	Type A
Ischemic cerebrovascular disease	Established	Class II	Type B
Subarachnoid hemorrhage	Established	Class II	Type B
Arteriovenous malformations	Established	Class III	Type C
Cerebral circulatory arrest	Established	Class III	Type C
Perioperative monitoring	Possibly useful	Class III	Type C
Meningeal infection	Possibly useful	Class III	Type C
Periprocedural monitoring	Investigational	Class III	Type C
Migraine	Doubtful	Class II	Type D
Cerebral venous thrombosis	Doubtful	Class III	Type D

Recommendations were made by Babikian VL, Feldmann E, Wechsler LR, Newell DW, Gomez CR, Bogdahn U, Caplan LR, Spencer MP, Tegeler CH, Ringelstein EB, Alexandrov AV, and endorsed by the American Society of Neuroimaging . J Neuroimaging 2000;**:***-*** (in press).

SICKLE CELL DISEASE

The Stroke Prevention Trial in Sickle Cell Anemia (STOP) has demonstrated a 90% relative risk reduction of ischemic stroke when the need for prophylactic blood transfusion was determined by TCD in children 2-16 years old. TCD was used to obtain time-averaged maximum mean flow velocity (MFV) in the middle cerebral (MCA) or intracranial internal carotid (ICA) arteries. TCD's were classified as normal (MFV < 170 cm/s), conditional (MFV 170-199 cm/s), abnormal (> 200 cm/s), or inadequate (TCD not interpretable). Children with two abnormal TCD's were randomized to transfusion aimed at the reduction of sickle hemoglobin to < 30% or standard care. Of 1933 children screened at 14 sites 130 were randomized. The trial was stopped because of a large beneficial treatment effect to prevent ischemic stroke (14 strokes in the normal care arm, 1 stroke in the transfusion arm). Among children not included in the trial, 4 strokes occurred in 1304 TCD-normals (a 35-months stroke-free survival 99%), 9 strokes occurred in 339 TCD-conditional children (95% survival), 16 strokes occurred in 183 TCD-abnormals (82% survival), and 3 strokes were diagnosed in 107 children with inadequate TCD studies.

The STOP study is the most successful stroke prevention trial to date and the data provide the strongest evidence for effective clinical application of TCD to prevent ischemic stroke in children with sickle cell anemia.

Potential TCD pitfalls include:

ISCHEMIC CEREBROVASCULAR DISEASE

TCD is indicated in patients with ischemic cerebrovascular disease including ischemic stroke, transient ischemic attack (TIA), or asymptomatic patients at high risk of stroke because it can detect:

TCD can be used in patients with acute (<12 hours) ischemia to identify major arterial occlusion or stenosis, as well as to document reperfusion (Figure 1). A proximal arterial occlusion on TCD is present in 69% of thrombolysis-eligible patients during the first six hours of ischemia. Recanalization occurs rapidly and only 24% of patients have occlusions on TCD within 6-24 hours after stroke onset.

In patients presenting subacutely, TCD helps to identify stroke pathogenic mechanisms and refine other findings (i.e. bilateral stenoses on carotid duplex, flow gap and artifacts on MRA, etc). The presence of collaterals and delayed flow acceleration on TCD usually indicates a hemodynamically significant lesion such as a > 80% ICA stenosis or occlusion. TCD can also unmask hemodynamically significant distal ICA obstructions and tandem ICA lesions.

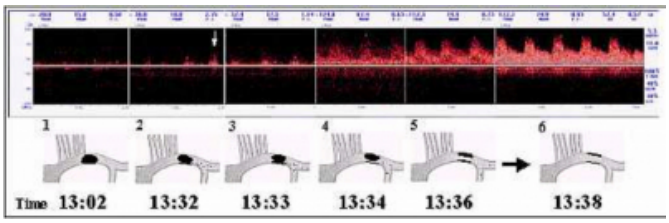
We use a battery of TCD findings described by Wilterdink et al as well as our criteria for P Comm A and compensatory flow increase to identify hemodynamically significant ICA lesions. In our series, TCD has a 94% sensitivity and 97% specificity for hemodynamically significant proximal ICA lesions and an 81% sensitivity and 96% specificity for distal ICA lesions (Demchuk AM et al, J Neuroimaging 2000:January).

These TCD findings are extremely helpful for grading carotid stenosis if carotid duplex shows elongated proximal ICA stenosis or clot with unremarkable velocities, bilateral ICA stenoses, or indirect evidence of a distal ICA lesion. In 26% of acute stroke patients, TCD provides information additional to other vascular imaging tests regarding stenosis severity and stroke pathogenesis. TCD is a useful addition to carotid duplex and MRA in evaluation of acute ischemic stroke.

TCD can be used to detect cerebral microembolic signals (MES) in patients with potential emboligenic sources including cardiac lesions such as atrial fibrillation and patent foramen ovale (PFO), extracranial carotid lesions, and intracranial stenoses. TCD can help to localize the source of embolization with simultaneous bilateral MCA monitoring, or unilateral MCA and ICA monitoring. TCD can be perfomed at bedside to detect PFO. A bolus of 15-20 cc of agitated saline is given intravenously and the patient should perform a Valsalva maneuver 5 seconds after bolus. TCD monitoring should be performed for 2 minutes on the left MCA, or bilaterally. A head-frame should be used to avoid probe movement and related artifacts. If MES arrive within seconds, they indicate functional PFO. If a “shower” or “curtain” of MES is detected, this indicates a large PFO with accuracy comparable to echocardiography. If MES arrive after 1 min, a pulmonary shunt may be present and this route of paradoxical embolism can not be detected by echocardiography.

Figure 2

Figure 1



SUBARACHNOID HEMORRHAGE (SAH)

TCD is indicated in patients with SAH to detect and monitor arterial vasospasm. Most of the patients we monitor have non-traumatic SAH due to ruptured intracranial aneurysm. A baseline TCD study should be performed on the day of diagnostic angiography or immediately after surgery to correlate velocities and vessel identification with angiographic findings. With the day of the ictus as day 0, we perform daily TCD in all grades of SAH during days 3 – 10 after headache onset because:

0.1. Vasospasm most commonly develops during the first week

0.2. Early detection of a significant spasm provides (in most cases) one or two days for the institution of therapy before the onset of ischemic symptoms

0.3. Progression from moderate to severe vasospasm can occur within 24 hours

0.4. Candidates for angioplasty can be identified earlier

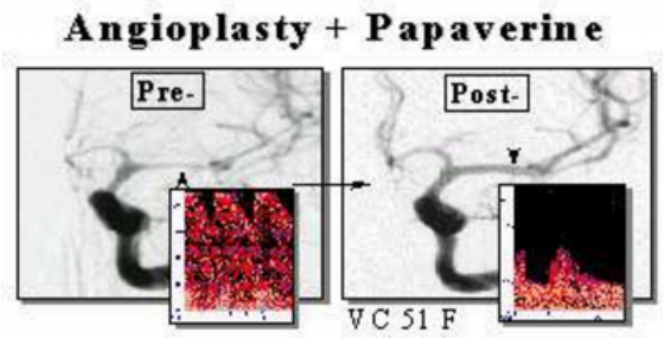
If no vasospasm is detected by day 7-8 in patients with grade I SAH, TCD is usually discontinued. TCD is performed every other day after days 8 through 10 in patients with higher SAH grades and no spasm. If no spasm is found at the end of second week, TCD is discontinued. If moderate or severe vasospasm is present during the second week, TCD monitoring is continued daily until spasm resolves to a mild

or low moderate degree. TCD is discontinued after day 10 when a patient with residual moderate vasospasm tolerates returning to normal blood pressure values.

The primary goal of TCD is to identify early development of a significant (high moderate or severe) vasospasm. These patients can then receive appropriate treatment (HHH therapy). If vasospasm increases, intra-arterial balloon angioplasty can be performed to restore vessel patency. Vasodilators may also used to improve distal spasm, although their effect is temporary. TCD may be used to assess the hemodynamic effects of interventions (Figure 2).

Figure 3

Figure 2



Pre-angioplasty MCA MFV 302 cm/s; post-angioplasty MCA MFV 106 cm/s.

The signs of resolving proximal vasospasm on TCD are:

The signs of resolving distal vasospasm on TCD include:

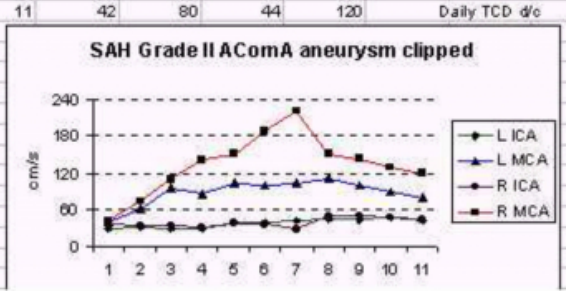
Potential TCD pitfalls include:

CASE REPORT

TCD velocity changes in a 47 y.o. female with Grade II SAH and anterior communicating artery aneurysm

Figure 4

Days	L ICA	L MCA	R ICA	R MCA	Comments
1	30	40	35	42	First day after headache onset
2	32	62	34	74	First day post-op
3	30	95	34	110	
4	30	85	32	140	
5	40	104	38	150	First day of HHH-therapy
6	40	100	36	189	
7	42	104	28	220	
8	46	112	49	150	First day after angioplasty
9	44	100	50	142	
10	48	89	48	129	
11	42	80	44	120	Daily TCD d/c



Arterio-Venous Malformation (AVM)

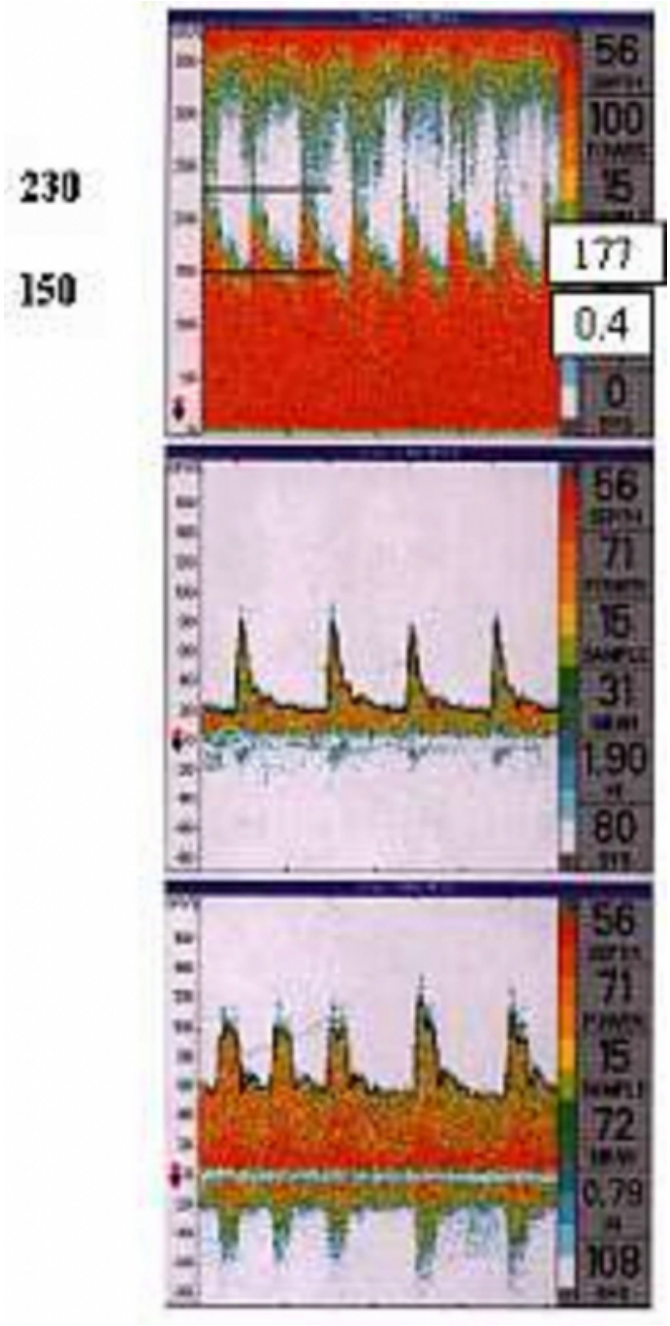
TCD can detect abnormal flow velocities and pulsatility in an AVM-feeder: an artery that partially or solely supplies an AVM and bears a signature of an extremely low resistance flow. Although TCD should not be used as a screening tool to detect an AVM, it can be used for patient follow-up during staged embolization

AVM feeders may also be accidentally found during routine TCD examination and sonographers should be able to identify these flow patterns.

Hassler and Burger suggested the following classification (in Newell D, Aaslid R, Transcranial Doppler, 1992):

Partial AVM feeders may have end-diastolic velocities greater than 80 cm/s and low PI values of 0.4-0.6.

Figure 5
Figure 3



1 hour after symptom onset:

A 23 year old male had a motor seizure and was drowsy. CT showed an intracerebral hemorrhage.

TCD showed aliasing at the mid M1 depths of insonation. The left M1 MCA MFV is 177 cm/s, PI 0.45. End-diastolic velocity exceeded 140 cm/s indicating an exclusive AVM feeder.

2 hours after symptom onset:

The patient became comatose and was taken for an immediate surgical clot removal.

Consistent with the clinical findings of raising ICP, TCD showed a high resistance flow pattern in the right MCA on the side contralateral to the AVM correlating with the clinical picture of a mass-effect.

At this time, MFV in the AVM feeder decreased to 72 cm/s and PI increased to 0.78 indicating compression of the AVM by an expanding intracerebral hematoma.

CEREBRAL CIRCULATORY ARREST

TCD is used as a confirmatory test to document reverberating flow in patients which indicates brain death due to cerebral circulatory arrest. TCD can also be used as a screening tool to determine the timing for other confirmatory tests such as nuclear brain scanning or angiography. It is of particular value in patients with high barbiturate levels when clinical examination and electro-encephalography are unreliable.

A variety of clinical conditions may lead to brain death. TCD is indicated when the clinical examination indicates brain death or is unreliable. The test may determine positive end-diastolic flow and rule out cerebral circulatory arrest, or may demonstrate reverberating flow and confirm clinical findings. It is extremely important to document arterial blood pressure during TCD examination to rule out transient arrest of cerebral circulation due to hypotension. Once reverberating flow is identified, it should be confirmed in both MCA's and the BA, and monitored for 30 minutes to exclude effects of transient intracranial pressure increase.

Patients at risk of brain death may need baseline TCD examination before clinical changes become apparent. A baseline TCD will help to establish the presence of temporal windows and end-diastolic flow. Subsequent TCD studies can be performed hours or days later with greater confidence, and intracranial flow changes will be easier to interpret. For example, absent end-diastolic flow in both MCA's and the BA in a patient with clinical brain death may indicate incomplete arrest (extremely high resistance to flow with some residual brain perfusion) or complete arrest (vessel wall distention during systoli with no brain perfusion). In other words, TCD can be performed either too early or too late. Although velocities may be minimal with the latter, confusion may arise if temporal windows were not established beforehand. In this situation, it is prudent to wait and repeat TCD 1-2 hours later. Progression to arrest will

manifest as reverberating flow, and a complete arrest will make identification of any signals more difficult (asonic arteries).

OTHER CLINICAL APPLICATIONS

Patients with ICA occlusive disease may be at risk of hemodynamic stroke if collaterals fail to adjust to various stimuli. TCD can be used to determine vasomotor reactivity (VMR) of the intracranial vessels. MCA flow velocity decreases during hypocapnia since constriction of arterioles increases resistance to flow, increases PI and dampens proximal flow velocity. Hypercapnia produces arteriolar vasodilation, decreased resistance and results in increased flow velocities and low PI's. The relative change from hyper- to hypo- over normocapnic state is referred to as VMR reserve capacity:

VMR = [1 – (MFVhyper – MFVhypo) / MFVnormo] x 100%.

To perform a CO2 response test, normal velocity values should be obtained at rest (end-expiratory PCO2 36-40 mm Hg). MFVhypo is determined during hyperventilation (decrease to 30-20 mm Hg). MFVhyper is measured during inhalation of a mixture of 5-7% CO2 and air with PCO2 raising up to 50-60 mm Hg. Normal VMR values are 86 + 16% and the response decreases with progression of carotid artery stenosis to occlusion. A decrease to 30% or less may indicate exhausted VMR. Klieser and Widder suggested another classification of VMR based on percent velocity changes for 1 vol% CO2 changes: sufficient > 10% during hyper- and hypocapnia; diminished < 10% during hypercapnia; and exhausted < 5% during hypercapnia, and < 10% during hypocapnia. Potential pitfalls include large between individual variations and the need for patient cooperation during the test.

To avoid CO2 inhalation and gas concentration measurement, an intravenous injection of 1 g acetazolamide (diamox) can be used to induce vasodilation. TCD recording should be obtained once the velocity reaches a plateau in approximately 20 minutes. CBF typically starts to increase in 15 minutes after the injection and reaches maximum at 25 minutes. To avoid velocity variations during breathing cycles, Doppler spectra from up to 20 cardiac cycles can be averaged.

To perform VMR testing, the transducer should be maintained at a fixed angle during all phases using a head-frame. A constant angle of insonation is required to make velocity measurements comparable. However, precise cerebral blood flow calculations from velocity values are impossible since the MCA diameter may be changing and remains unknown during examination. Impaired vasomotor reactivity is found in patients with hemodynamically significant ICA stenoses or occlusions and there is a potential association with an increased risk of stroke. However, prospective multicenter studies are necessary to determine the value of VMR to identify high risk patients.

TCD can also be used for monitoring during procedures, particularly during cardiopulmonary bypass (CABG), carotid endarterectomy (CEA), carotid angioplasty and stenting, balloon occlusion, etc. These special applications of TCD may be potentially useful since TCD allows real time assessment of:

Brain microembolization;
Brain hypoperfusion;
Thrombosis.

Although the cumulative count of microembolic signals (MES) was associated with neuro-psychological deficits after CABG, individual MES detected by TCD are asymptomatic due to their microscopic size. Evidence for microembolization helps to identify the source and to select safe surgical maneuvers to minimize air or particulate brain embolization.

If the MCA MFV decreases to less than 30% of baseline values after clamping the ICA during CEA, it indicates a significant reduction in cerebral blood flow. If the MCA MFV does not recover within 1-2 minutes, it means that collateral channels are failing to compensate for flow reduction and this patient may need shunting. On the contrary, if a surgeon performs shunting routinely, an unexpected MCA MFV drop may help to detect a kink or thrombosis of the shunt.

TCD monitoring is a developing field that requires tight probe fixation devices, operator independent technology and prospective multicenter studies to determine the value of real-time physiological assessment of brain perfusion.

The STAT Neurosonology Service, UT-Houston has adopted the following clinical indications for TCD examinations at Hermann Hospital and outpatient clinic:

Figure 6

Table 2: Indications for TCD

Indications	Service	Follow-up TCD
Sickle Cell Disease	Elective schedule	Normal TCD: Yearly Conditional: 6 months Abnormal: 3-6 months
Acute (< 12 hours) Ischemia	STAT bedside	Thrombolysis: Continuous monitoring Stroke progression: 12 – 48 hours
Subacute Ischemia	Same day	Stroke progression: 24 – 72 hours Emboligenic source: 1 hour emboli monitoring Intracranial stenosis 3 – 6 months Outpatient follow-up variable
Subarachnoid hemorrhage	Daily Alternate day Daily Variable	All grades: Baseline TCD (by day 1-2) Days 3-7 No spasm: Grade 1 - discontinue TCD Other grades - every other day upto day 15

Figure 7

		Spasm: Daily until spasm diminishes to low moderate or less, then alternate day to day15 Residual spasm: continue if necessary
Arteriovenous malformations	Elective schedule	Staged AVM embolization: variable
Cerebral circulatory arrest	STAT bedside	Positive EDV: Repeat TCD in 3-4 hours Daily TCD if necessary Absent EDV: Repeat TCD in 1-2 hours Reverberating flow: 30 min TCD monitoring
Other (i.e. head trauma, meningeal infection, etc)	Same day	Repeat TCD: when necessary

References

r-0.
r-1.
r-2.
r-3.
r-4.
r-5.
r-6.
r-7.
r-8.
r-9.
r-10.
r-11.
r-12.
r-13.
r-14.

Author Information

Andrei V Alexandrov, MD, RVT*
Assistant Professor, Director of the STAT Neurosonology Service

Mathew Joseph, MD
Research Associate, Departments of Neurology-Stroke Service* and Neurosurgery, Health Science Center Medical School, The University of Texas-Houston