

Transcranial Doppler Ultrasonography

Chapter 8



What is TCD-US?

- Christian Johann Doppler first described the Doppler principle in 1843
- TCD is a noninvasive US device that allows real-time evaluation of intracranial cerebral circulation

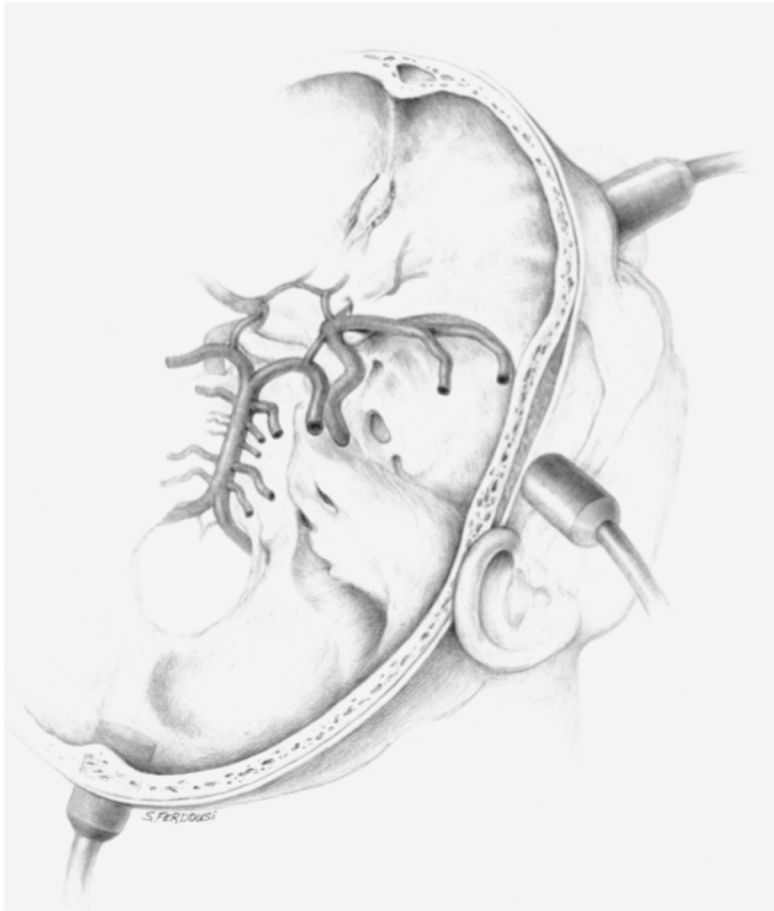


Figure 1. TCD probe positions over different acoustic windows of the skull.

US examination of a vessel by means of TCD is referred to as insonation

TCD probe is placed over different "acoustic windows" that are specific areas of skull where there is a lack of bony covering or the cranial bone is thin

Transtemporal window - MCA, ACA, PCA, and terminal portion of the internal carotid artery

Transorbital window - ophthalmic artery and the internal carotid at the siphon level

Transforaminal (occipital) window - distal vertebral arteries and the basilar artery

Submandibular window - distal portions of the extracranial internal carotid artery

How does it work?



TCD does not allow direct visualization of an insonated vessel like a carotid duplex



Indirect evaluation by means of an US beam (2 MHz frequency) produced from electrically stimulated piezoelectric crystals



This beam bounces off the RBCs within the insonated artery



The reflected signal is received by the transducer and converted into an electrical signal

- Waveform demonstrates blood flow velocities, the direction of flow and allows for calculated parameters to be added to the evaluation
 - Pulsatility Index (PI) – reliable marker of resistance distal to the insonated site
 - $PI = (\text{peak systolic velocity} - \text{end diastolic velocity}) / \text{mean velocity}$

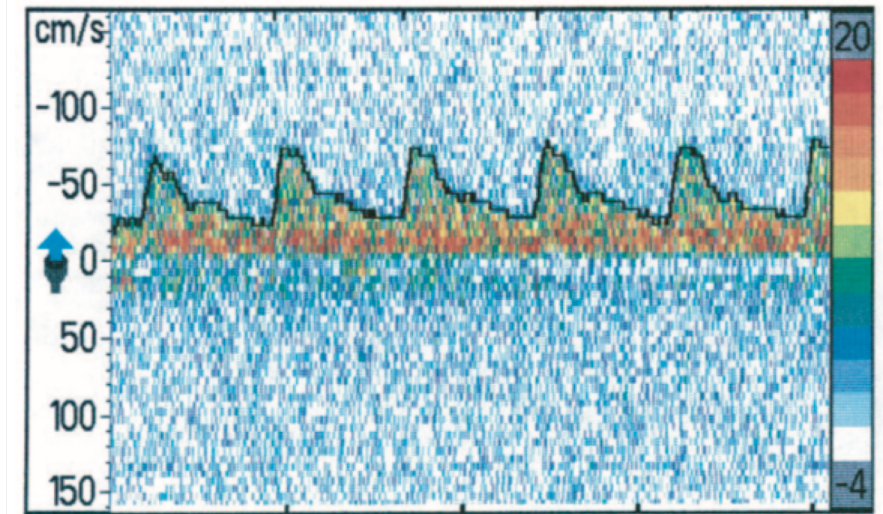


Figure 2. TCD screen waveform with velocity scale on the left.

Basic Cerebrovascular Hemodynamics



Ohm's Law: $\text{flow} = \text{pressure} / \text{resistance}$



$\text{CBF} = \text{CPP} / \text{CVR}$



$\text{CPP} = \text{MAP} - \text{ICP}$



CVR is influenced in physiologic states by constriction and dilation of small vessels in the brain



In pathologic states, focal changes in resistance can be seen immediately behind an area of significant stenosis



The main goal of brain hemodynamics is to keep CBF stable despite alterations in CPP and CVR

TCD Examination & Interpretation

Table 1. Accepted Guidelines for a Normal TCD Study

Artery	Window	Depth (mm)	Direction	Mean Flow Velocity
MCA	Temporal	30 to 60	Toward probe	55 ± 12 cm/s
ACA	Temporal	60 to 85	Away	50 ± 11 cm/s
PCA	Temporal	60 to 70	Bidirectional	40 ± 10 cm/s
TICA	Temporal	55 to 65	Toward	39 ± 09 cm/s
ICA (siphon)	Orbital	60 to 80	Bidirectional	45 ± 15 cm/s
OA	Orbital	40 to 60	Toward	20 ± 10 cm/s
VA	Occipital	60 to 80	Away	38 ± 10 cm/s
BA	Occipital	80 to 110	Away	41 ± 10 cm/s

TCD, transcranial Doppler; MCA, middle cerebral artery; ACA, anterior cerebral artery; PCA, posterior cerebral artery; TICA, terminal internal carotid artery; ICA, internal carotid artery; OA, ophthalmic artery; VR, vertebral artery; BA, basilar artery.

- Different depth range, flow direction, and normal age-related flow velocity (**FV**) ranges are established for each vessel

TCD measurements are influenced by different physiologic and pathologic factors, also by vasoactive medications

Table 2. Effects of Different Physiological States on TCD Flow Velocity

Physiologic Factor	Effects
Increase age	Decrease FV
Increase CSF pressure	Decrease FV
Increase central venous pressure	Decrease FV
Increase PaCO ₂	Increase FV due to vasodilatation
Increase cardiac output	Decrease FV to maintain normal CBF
Increased blood viscosity	Decrease FV
Anemia	Increase FV
Drugs with vasodilatation properties	Increase FV
Drugs with vasoconstriction properties	Decrease FV

TCD, transcranial Doppler; CSF, cerebrospinal fluid; FV, flow velocity; CBF, cerebral blood flow.

Different Vascular Scenarios

Pure focal narrowing at the site of insonation will cause an increase in FV

Narrowing or obstructing lesions proximal to the insonation site will cause a decrease in FV observed at the insonation site

Downstream (distal) decrease in vascular resistance (as in the presence of AVM) will increase FV and decrease PI at the site of insonation

Downstream increased vascular resistance (as in stenosis or obstruction) will decrease FV and increase PI proximal to the lesion

Clinical Applications



Sickle Cell Disease



Intracranial Vasospasm



Arterial Stenosis and Occlusion



Monitoring for Sources of Emboli and Cardiac Shunts



Brain Death



Testing for Cerebrovascular Autoregulation

Sickle Cell Disease

SCD associated with progressive occlusion of large intracranial arteries (most frequently ICA and MCA)



Total occlusion of any one of these arteries will lead to massive stroke

Mean FV up to 170 cm/s = normal

171 – 199 cm/s = conditional

≥ 200 cm/s = abnormal, require transfusions; accompanied by stroke risk of 40% within the next 3 years

Intracranial Vasospasm



- Constriction of cerebral blood vessels due to presence of blood in the subarachnoid space after trauma or rupture of aneurysm
- Vasospasm if severe can produce ischemia to the brain; increase in mortality 1.5 to 3 fold during the first two weeks after SAH
- Vasospasm typically occurs within 3-21 days after SAH and may last for 12 – 16 days
- Pathophysiology is poorly understood but may include changes within vessel walls themselves, alteration of vasoactive substances, immune response, inflammation, oxidative damage



**SAH occurs in
25,000 –
30,000
people in the
US / year**



**Can be
identified 1-2
days before it
becomes
clinically
symptomatic**



**Vasospasm
that follows
SAH causes
increased FV
inside
intracranial
vessels**



**This can be
detected by
TCD
indicating the
need for
treatment
prior to signs
of ischemia**

Table 3. TCD's Mean Flow Velocity Criteria for Cerebral Vasospasm

Severity of Vasospasm	MFV Value cm/s	MCA/ICA Ratio
Normal	<85	<3
Mild	<120	<3
Moderate	120 to 150	3 to 5.9
Severe	151 to 200	>6
Critical	>200	>6

TCD, transcranial Doppler; MFV, mean flow velocity; MCA, middle cerebral artery; ICA, internal carotid artery.

Arterial Stenosis & Occlusion



TCD measurements that correlate with stenosis would be increase FV at the stenotic site



In the case of total occlusion, there should be no flow signal from the occluded site



Increased velocity and/or reversed flow in the collateral vessels may also be seen

Sources of Emboli and Cardiac Shunts

Microemboli traveling along an insonated vessel appear as high intensity transient signals (HITS) on TCD

- May ID high risk status for clinical stroke

Can detect presence of R->L shunt e.g. PFO in patients with contraindications to TEE

- + for R -> L shunt if a shower of HITS is detected in the MCA by TCD 5-10 seconds after IV injection of 10mL of agitated saline
- If shower emboli is detected after a minute of injection it might indicate the presence of a pulmonary shunt!

Brain Death



TCD evaluation in brain death → reverberating or oscillating pattern of flow

Normal arterial blood flow in systole and reversed flow in diastole 2/2 high distal brain resistance in brain dead patients



Useful adjunct test!

Testing for Cerebrovascular Autoregulation



Cerebral autoregulation is the ability to maintain the CBF despite minute-to-minute variation in cerebral perfusion rate



In normal state, this is achieved by arteriolar control over the cerebral peripheral vascular resistance



Breath holding and acetazolamide are the two most commonly used maneuvers to manipulate cerebral autoregulation

Advantages & Limitations of TCD

Advantages

- TCD is relatively inexpensive, noninvasive, portable, and easy to use
- Allows for frequent repeated measurements, continuous monitoring
- Immediate real time detection of changes in CV hemodynamics

Limitations

- Blind procedure: accuracy relies on the knowledge and experience of trained technician and interpreter
- Limited ability to detect distal branches of intracranial vessels
- In 5-10% of cases, sufficient penetration of the bone window cannot be achieved