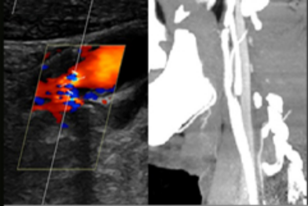
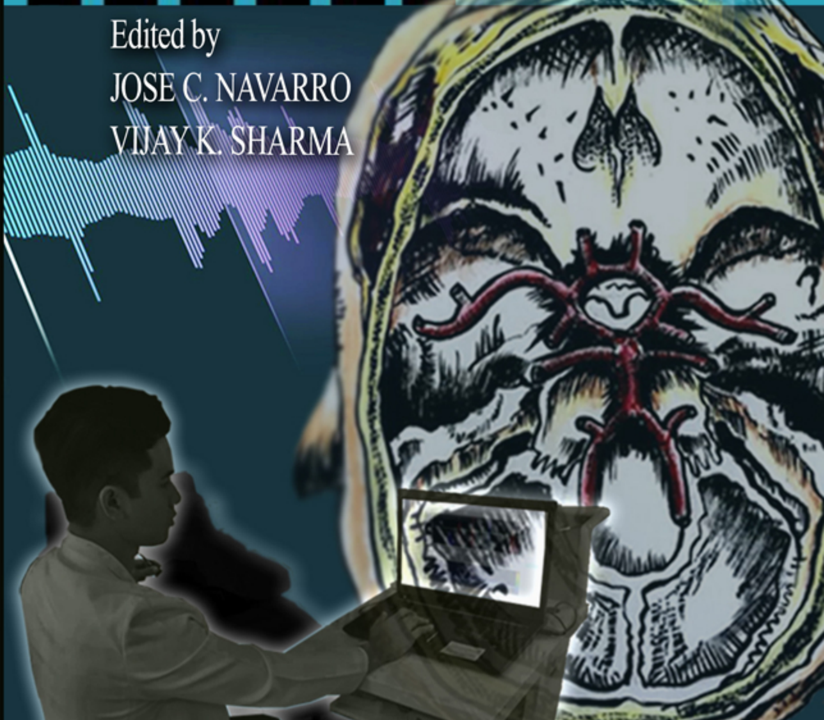
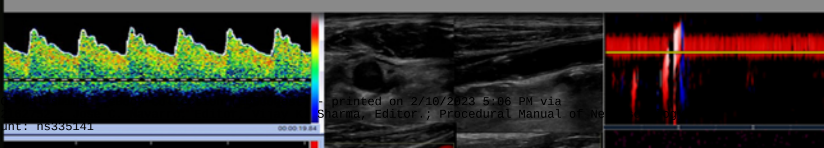


PROCEDURAL MANUAL *of* NEUROSONOLOGY

Edited by
JOSE C. NAVARRO
VIJAY K. SHARMA





Procedural Manual of Neurosonology

Procedural Manual of Neurosonology

Edited by

Jose C. Navarro and Vijay K. Sharma

Cambridge Scholars Publishing

Procedural Manual of Neurosonology

Edited by Jose C. Navarro and Vijay K. Sharma

This book first published 2019

Cambridge Scholars Publishing

Lady Stephenson Library, Newcastle upon Tyne, NE6 2PA, UK

British Library Cataloguing in Publication Data
A catalogue record for this book is available from the British Library

ISBN (10): 1-5275-3426-X
ISBN (13): 978-1-5275-3426-1

TABLE OF CONTENTS

FOREWORD

Stroke systems of care undergo rapid changes and the field of Vascular Neurology faces new challenges. Emphasis on imaging in patient selection for reperfusion therapies placed snapshot assessment of occlusion location and estimates of core and penumbra as key elements in education of next generation of clinicians caring for stroke patients. The use and knowledge of cerebrovascular ultrasound testing decrease leaving these skills a privilege of few.

A stroke clinician should be able not only to determine if a patient is eligible for systemic thrombolysis or endovascular treatment but also to ascertain stroke pathogenic mechanisms across all patients. The key question in Vascular Neurology is not just What? and Where? but **Why?** A normal CT angiogram in a patient with severe or fluctuating symptoms poses more questions. Ultrasound brings a unique option of evaluating cerebral hemodynamics in real time with assessments being repeatable as often as necessary. The ability to perform various cerebrovascular ultrasound tests as an extension of the neurological examination and knowledge how to interpret cerebral and systemic hemodynamic findings distinguishes a stroke clinician capable to attend to needs of the most complex patients and tailor make treatment or secondary prevention solutions based on these individual pathogenic mechanisms. Ultrasound is entirely complementary to CTs, MRIs and catheter angiography, and these vascular tests should be taught in all stroke fellowships worldwide, ideally starting during residency.

My dear friends and colleagues Jose Navarro and Vijay Sharma have put together a practical manual of most commonly used cerebrovascular ultrasound tests and assembled the group of contributing experts that represent leading stroke specialists from Asia. This is a welcome contribution that complements existing literature mostly from European and US experts. A perspective for countries where advanced multi-modal imaging for stroke is not universally available makes this book a valuable resource particularly for clinicians facing challenges in caring for stroke patients with limited resources. In addition, residents and fellows should find this informative to learn tests and key applications. Existing

ultrasound laboratories and technologists should also have updated scanning protocols and operation manuals, all good reasons to have this book.

Andrei V. Alexandrov, MD
Semmes Murphey Professor and Chairman,
Department of Neurology
Director,
Neurosonology Examination
President,
American Society of Neuroimaging

PREFACE

In spite of the tremendous advances in the field of vascular neuroimaging, the role of ultrasound (Transcranial Doppler, Transcranial Color-Coded Duplex, and Carotid ultrasonography) in the diagnosis and management of cerebrovascular disorders can still be relied upon. The accuracy of neurovascular ultrasound against vascular imaging is quite acceptable. Certain ultrasound tests can even be considered superior to other modalities. The detection and monitoring of microembolic signals in real-time can be achieved only with ultrasound. Vasomotor reactivity of brain vasculature demonstrates the downstream effects of a significant arterial stenosis and can be performed at the bedside by TCD. This can aid the clinicians in a confident risk stratification, especially for patients undergoing major surgical procedures. TCD monitoring during intravenous thrombolysis may identify the site of arterial occlusion, recanalization and re-occlusion in real-time. Furthermore, its portability and being a non-invasive test make it advantageous as compared to other vascular imaging techniques. TCD has acceptable accuracy parameters for test performance and reproducibility.

A group of neurosonologists, mostly from Asia pooled together their talent, time and effort to create this manual. It is not intended to be a textbook or a reference book of neurosonology. The main purpose of this manual is to have an immediate access for clinicians and sonographers, trainees and students performing neurosonological evaluations. Chapters are written by several authors to reflect diverse expertise, experience and techniques put together in a concise manner.

We wish to express our deepest gratitude to our families for support given to us while working on this project. It also goes to our patients, trainees and colleagues in our respective departments. Lastly, we thank Cambridge publishing for believing in us and providing the opportunity to have this manual published.

Jose C. Navarro, MD, MSc
Vijay K. Sharma, MD

Applied Ultrasound Physics and Instrumentation

Amit Batra, Jose C. Navarro, Komal Kumar, and Vijay K Sharma

For a better understanding of a technology, users should know the basic principles, components, simple operations and interpretations of various research findings. Cervical duplex (CDU) and transcranial Doppler (TCD) ultrasonography are routinely performed for hemodynamic evaluations in patients with cerebrovascular ischemia. Both CDU and TCD are essential components of comprehensive stroke centers.

Ultrasound physics is considered quite difficult. Often, it is said that "if you do not want someone to learn clinical ultrasonography, teach him physics in detail. He will certainly run away! In this chapter, we describe some of the basic principles and the relevance of ultrasound physics. Although, this is not comprehensive, this chapter contains all the essentials needed by a beginner. Readers interested in becoming experts or planning to take the credentialing examinations are advised to understand ultrasound physics in greater detail.

1. **What is ultrasound?**
   A normal human ear can hear sound frequencies from 20-Hz to 20,000- Hz. Sounds with frequencies of more than 20,000 Hz are inaudible to the human ear and called ultrasound waves.

2. **What are the basic characteristics of sound?**
   Sound is a longitudinal, mechanical, and pressure wave that travels in a straight line. It requires a medium for its travel. The speed of sound is determined by the medium through which it propagates (and has no relationship with the frequency, amplitude, power, or any other variable). The speed of the propagation of sound in air is 330 meters per second, while it becomes 1,540 meters per second (1 mile per second) in soft tissues.

Ultrasound frequencies emitted by the transducers used in the commercial ultrasound machines used for medical purposes are between 2- and 22-MHz. While a lower frequency ultrasound travels for longer distances (2-MHz ultrasound travels up to 150 mm), the imaging resolution is poor. On the other hand, higher frequency ultrasound transducers can image only superficial tissues but with a high resolution. For example, excellent tissue resolution needed for carpal-tunnel imaging uses 22-MHz ultrasound. The commonly performed cervical duplex ultrasonography uses transducers with frequencies ranging from 5- to 12-MHz.

3. What is the Doppler effect?

Whenever a relative motion occurs between the sound source and the observer, the frequency heard by the observer is different from the originally produced sound frequency. Accordingly, when the distance between the source and the observer increases, the latter hears a lower frequency (negative Doppler shift). On the other hand, the observer hears a frequency higher than the original if the distance between him and the source is reduced (positive Doppler shift). This phenomenon was described by Christian Doppler in 1842. In general, the Doppler shift is calculated with the following formula:

$$f_D = \frac{2f_o \, v \cos \theta}{c}$$

Where f_D is frequency shift, f_o is original frequency, v stands for velocity of sound, cosθ is the angle of insonation, and c is a constant.

The constant (c) changes with the ultrasound frequency. Therefore, the Doppler frequency shift is independent of the originally produced frequency. This explains why the flow velocities (derived from the frequency shift) are the same whether one uses a transducer with a frequency of 4-MHz or 8-MHz.

According to this formula, the angle of insonation is an important determinant of the Doppler shift. Sonographers should remember at least the following:

a. Cosine of 0 degree = 1
b. Cosine of 180 degree = -1
c. Cosine of 60 degrees = 0.5

These numbers mean that if an arterial segment is insonated parallel to the blood flow (the angle of insonation is 0 or 180 degrees), the Doppler shift represents the real flow velocity. On the other hand, if an artery is insonated at 60 degrees to the blood flow, the Doppler shift (hence, the flow velocity) measured by the machine would be about 50 percent lower than the real. This is important since most of the flow velocities obtained during cervical duplex sonography are obtained at 60 degrees of insonation.

4. How is ultrasound produced?

Ultrasound is produced when a small amount of current is passed through a piezoelectric crystal (lead zirconate titanate). Note that '*piezo'* means pressure. The crystals are very stable and have a very long life. Their piezoelectric nature gets destroyed only with hard physical impact. Therefore, sonographers should always avoid dropping the transducer (see figure 1) on the floor.

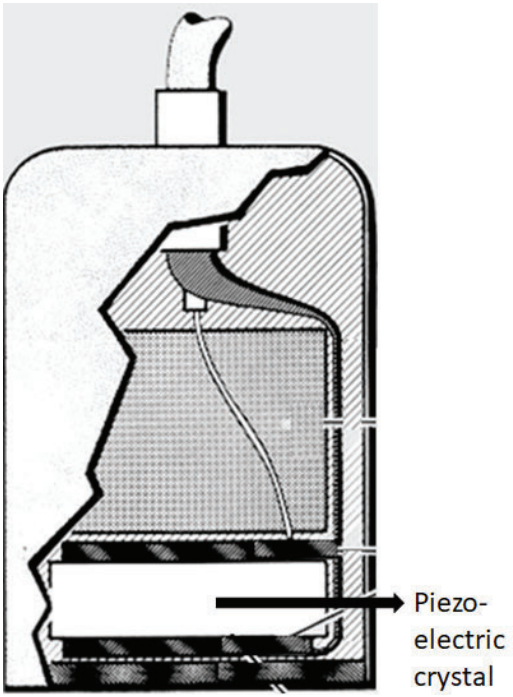


Figure 1.

5. What is the difference between 'continuous wave' and 'pulsed wave' ultrasound?

Initial ultrasound machines used the continuous wave (CW) ultrasound, in which one transducer continuously emitted ultrasound waves, while the other transducer received the waves reflected back from various tissue interfaces. Therefore, the instruments were bulky. Although, these machines could detect the flow and its direction, they were unable to tell the distance (or depth) between the source and the reflector. Therefore, CW ultrasound are "range ambiguous."

In pulsed wave (PW) ultrasound systems, instead of emitting continuously, the transducer emits ultrasound waves in a burst or bundle only for a fraction, and then starts working as a receiver for the reflected waves. Most of the diagnostic transducers emit ultrasound for 1% of the time (also called duty factor) and listens for 99% of the time. Since the time of emission of ultrasound and the time of receiving are known, the distance from where the waves are reflected can be assessed. Therefore, the PW ultrasound systems are "depth discriminant."

The operators should understand the important concept of "aliasing". When the Doppler shift is more than half of the pulse repetition frequency (PRF, i.e., the number of ultrasound bundles emitted per second), PW ultrasound systems are unable to detect the flow correctly (remember that the blades of a fan appear as moving counterclockwise slowly when the fan attains fast speed). This is called aliasing, and it is defined by the Nyquist limit. This is one of the major limitations of PW ultrasound systems. On the other hand, the CW systems are not limited by aliasing (the Nyquist limit is maximum). Aliasing may be observed on color flow imaging (figure 2) as well as on Doppler spectra (figure 3).

Current, commercially available equipment use PW ultrasound.

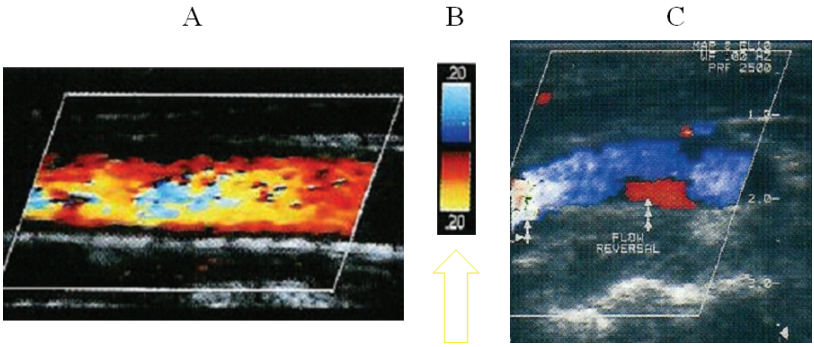


Figure 2. Panel A shows aliasing in color mode. Panel B shows the color scale. Shades of red are in one direction, while the shades of blue lie in the opposite direction. Importantly, the center of this color scale is black. It means that if there is a change (reversal) in the direction of blood flow, it has to go from red to black to blue or the other way around (panel C). If there is no black color in between the colors on the two sides of the color scale, this becomes aliasing (A).

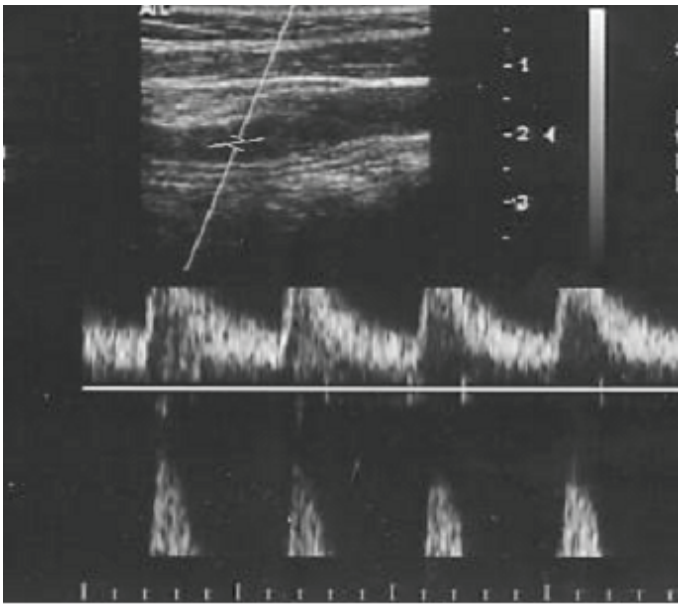


Figure 3. Aliasing on Doppler spectra. Due to the lower pulse repetition frequency (or low scale), the machine cannot measure the peak velocities. The top of the spectra appear as cut from the top and pasted below.

Some methods of correcting Aliasing (figure 4):

a. Increase PRF (scale)
b. Move baseline up or down
c. Use the CW ultrasound

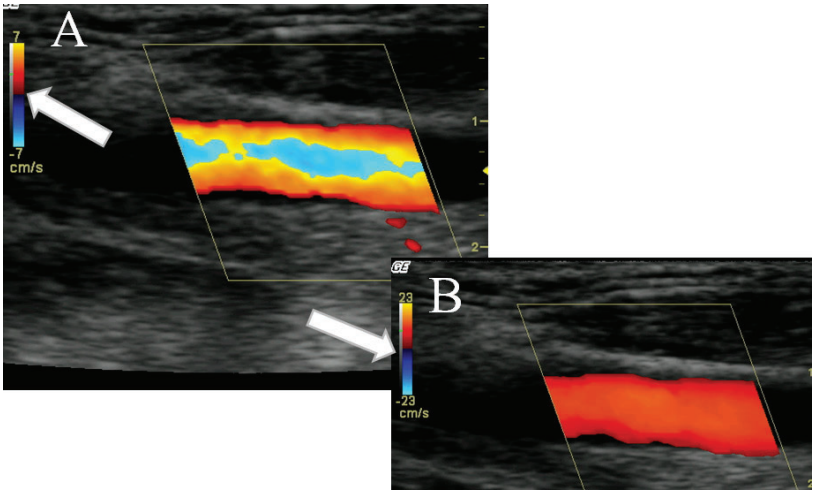


Figure 4. Methods of correcting. Panel A shows aliasing on color-flow (due to low scale setting). Aliasing gets corrected (un-aliasing) on Panel B as the scale is increased.

6. What are the basic principles of fluid dynamics?

All principles of fluid dynamics presume that a flow moves in a straight tube and the stenosis (when it occurs) is concentric (or axis symmetric). Some important concepts are:

a. Blood flows in layers (laminar) and the central layer is fastest. On the other hand, the flow is minimal along the arterial wall (parabolic). Figure 5 shows this phenomenon.

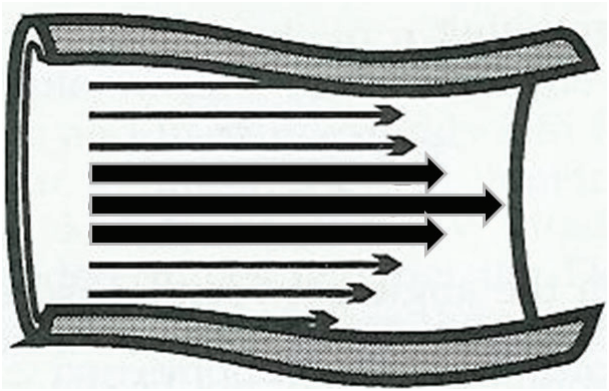


Figure 5. Laminar and parabolic flow. The slow flow along the arterial wall serves as a protection from shearing stress and reduces wear and tear.

b. Poiseuille's law

$$Q = \frac{\Pi(P1 - P2)r^4}{8L\eta}$$

Where Q = Blood flow

P1-P2 = pressure difference

r = radius

L = length

η = viscosity

The most important clinical applications of this formula are:

1. The blood-flow volume is directly proportional to the fourth power of radius. Therefore, reduction of the radius to half will decrease the flow volume by sixteen times (½ X ½ X ½ X ½).
2. The flow volume reduces with increasing length of the stenosis.

7. What are the basic components of an ultrasound machine?
The components of the current commercial diagnostic ultrasound systems are:

A. Transducer probe
B. Transducer pulse controls
C. Central processing unit
D. Display
E. Keyboard/cursor
F. Disk storage device
G. Printer

Therefore, an ultrasound equipment is just a specialized computer.

8. What is the resolution of an ultrasound machine?
Resolution is the ability of the machine to differentiate between two adjacent structures. It is of two types: axial and lateral (figure 6).

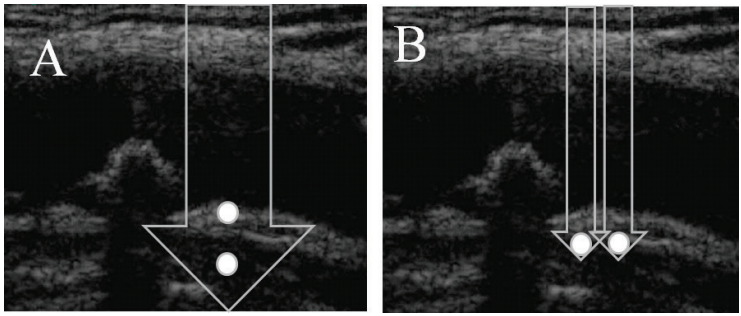


Figure 6. Axial resolution (A) differentiates between superficial and the deeper structures. It is directly dependent on the frequency of the ultrasound transducer (higher frequency = higher axial resolution). Lateral resolution (B) is the ability of the equipment to differentiate two adjacent structures. It depends on the beam width.

9. Which transducer frequency is good for me?
A higher frequency ultrasound has higher axial resolution. However, it does not penetrate deeply. On the other hand, a lower frequency ultrasound can penetrate deeply but has low resolution.

Thus, a sonographer compromises the resolution of the ultrasound by its ability to scan deeper structures. This compromise is called "trade-off."

10. What is ALARA?

ALARA stands for "as low as reasonably achievable." Ultrasound is a form of energy. It undergoes reflection, refraction, and absorption at various tissue interfaces during its propagation among various tissue interfaces. The absorbed ultrasound may get converted into heat energy (thermal side effect) and cause tissue damage. The other biological effect is cavitation. It occurs owing to the fast speed of transmission, which leaves a vacuum behind. This may lead to a collapse of the tissues with resultant damage.

Although the commercially available ultrasound systems are quite safe for human use, sonographers should still follow the ALARA principle.

Basic Operations of the Cervical Duplex Machine

Many companies manufacture cervical duplex machines. An example with various parts of the system is shown in figure 7.

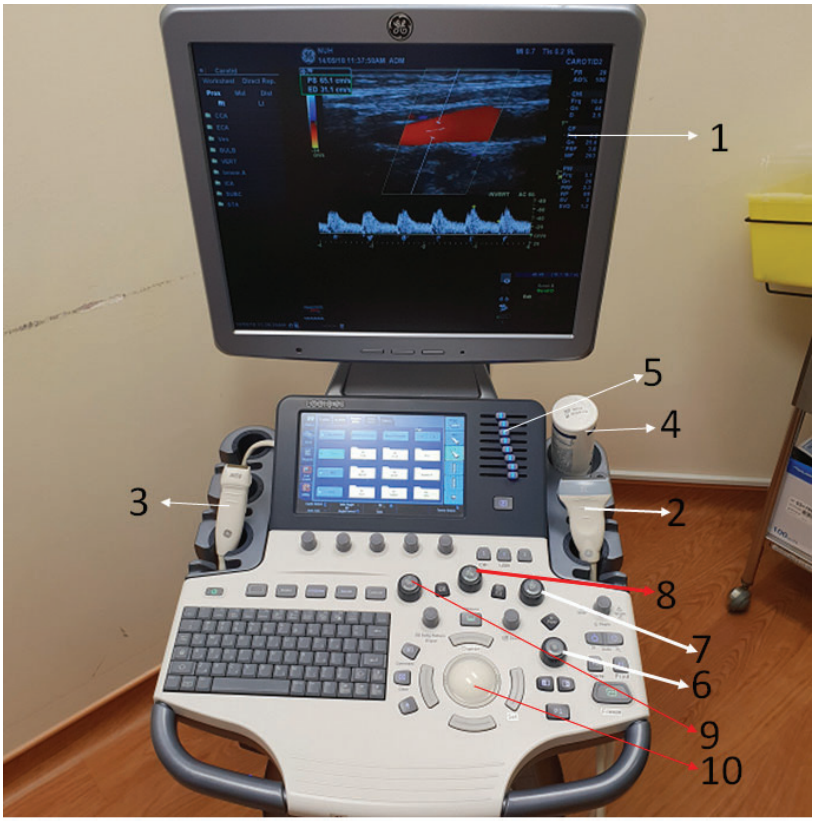


Figure 7. The basic appearance of a cervical duplex machine. Some of the most important components are:

1. Screen. This is just like the screen of modern computers. The method of display of information on the duplex scanner is fast Fourier transform (FFT).
2. Longitudinal ultrasound transducer (frequency 9-MHz) is used for cervical duplex sonography.
3. Spectral ultrasound transducer (frequency 2-MHz). For transcranial color-coded duplex (TCCD) sonography.
4. Ultrasound coupling gel bottle.
5. Time gain compensation (TGC). These knobs move horizontally for adjusting the gain at various depths.
6. Knob for Brightness-mode (B-mode) imaging. B-mode is also called grey-scale imaging. This is the most important mode for imaging of the tissues

under the transducer. This knob is rotated to increase or decrease the gain (brightness).

7. Knob for color-flow imaging (often written as CF). Once the B-mode imaging is completed, this knob is pressed to obtain the flow in a blood vessel. This button can also be rotated to adjust the color gain.
8. PW knob. This is the knob for pulsed-wave Doppler. When pressed, it helps in obtaining the spectral Doppler flow from the area of interest in a blood vessel.
9. M-Mode knob. This initiates the Motion-mode of ultrasound imaging. This mode is not used in cervical duplex imaging. This is of use during echocardiography.
10. Track ball. This ball is similar to those in laptop track-pads. It helps in moving the cursor or image to the desired frame.

Basic Operation of the Transcranial Doppler Machine

Appearance of a commercially available TCD machine is shown in figure 8.

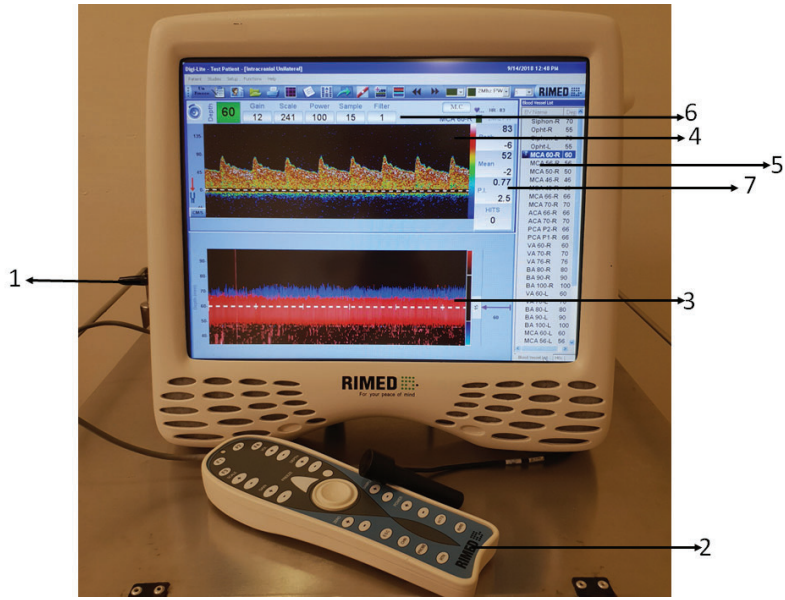


Figure 8. TCD machine. Most of the current machines have touch screens, and various parameters may be adjusted on the screen. The commonly used buttons on the machine are:

1. TCD probe. It emits 2-MHz pulsed wave ultrasound waves.
2. Remote control keyboard. It is provided in all machines since the screen may be far away from the operator to touch the screen for various adjustments. All buttons on the screen are duplicated on the remote control.
3. M-mode on the screen. It shows flow in all arteries that fall in the path of the ultrasound beam. M-mode is color coded in TCD machines where red indicates flow toward the probe while blue denotes flow away from the transducer.
4. Spectral Doppler. It shows the flow spectra from a particular depth of insonation.
5. This window shows the scanning protocol. Names of various intracranial arterial segments with their respective depths appear here.
6. This panel shows the settings of the machine for a particular frame. It shows (from left to right): depth in mm, gain in dB, scale, power, and sample volume (SV) in mm. In this frame, the spectral Doppler represents the strongest signal from a depth range of 60 ± half of SV. This means that the flow spectra shown here are the strongest signal obtained from a depth range of 52.5–67.5 mm.

Various protocols may be stored in the TCD machines. The appropriate protocol may be activated when needed.

TRANSCRANIAL DOPPLER EXAMINATION

ANNABELLE LAO-REYES, JOSE C. NAVARRO AND VIJAY K. SHARMA

Introduction

The circle of Willis at the middle and posterior cranial fossa affords the examination of the proximal blood vessels of the cerebral circulation (figure 1). Several studies have established the utility of the transcranial Doppler (TCD) in diagnosing vascular diseases of the brain. It is portable, reliable, inexpensive, and reproducible, making it a suitable tool for bedside examination. TCD examinations broaden the ability of physicians for rapid determination of pathophysiologic mechanism of cerebral ischemia. It aids in monitoring reperfusion and early arterial re-occlusion in acute ischemic stroke patients. The accuracy of TCD has been compared with angiography (digital subtraction angiography, magnetic resonance angiography as well as computed tomographic angiography), with a reliability rate that ranges from good to excellent.

A. Machine Setup and Steps in Performing TCD Examination

TCD examination relies on the presence of an adequate temporal bone window. However, insonation of some intracranial vessels can be performed adequately through the foraminal and orbital windows. During the routine TCD examination, the machine is set to maximum power of 100 percent (except orbital window where the power is reduced to 10%) and a sample volume of 10mm. Nowadays, modern TCD machines are equipped with an M-mode multi-depth display for faster vessel identification. Additionally, the machine settings are adjusted to increase the gate if no window is detected (figure 2). If the transtemporal insonation at full power yields good echogenic spectra, the gate size and power should be reduced to minimize patients' exposure to ultrasound energy ("as low as reasonably achievable," or ALARA). Low (at 10

percent) power should be used when TCD insonation is performed via the orbital window, burr holes, or the fontanels in children.

Initially, the zero line is placed in the middle of the screen in order to display the bidirectional signals. If the velocities are high, increase the scale to avoid aliasing. This can be achieved by moving the baseline up or down also. To optimize the weaker high velocity signals, increase the gain settings with a slower sweep speed. For weaker signals, check the accuracy of the automated readings with the envelope or waveform follower. Manual cursor measurements should be performed if an erroneous envelope tracing is noted (See also chapter 16, "Pitfalls in Neurosonology").

During the spectral TCD examination, the sonographers should be able to:

1. Follow the course of blood flow for each major branch of the circle of Willis ("go with the flow").
2. Identify, optimize, and store spectral waveforms at two or more key points per artery; MCA signals may also be stored as proximal, mid, and distal; VA signals may be stored at 40–50 and 60–70 mm, and BA signals can be stored at proximal, mid, and distal segments. Note the variability of velocities in these segments.
3. Identify, optimize, and store any abnormal or unusual waveforms or signals.
4. Measure the highest velocity at each key point.

B. Windows for TCD Insonation/Blood Vessels

Transcranial Doppler Windows and Probe Position

Acoustic windows are areas in the skull where the bone is relatively thinner and permits sufficient penetration of the ultrasound. The four commonly employed acoustic windows in adults are: temporal, orbital, sub-occipital or foraminal, and submandibular. Through the transtemporal window, the flow velocities in middle cerebral (MCA), anterior cerebral (ACA), posterior cerebral (PCA), and posterior communicating (PCOM) artery are obtained. The ophthalmic artery (OA) and internal carotid artery (ICA) siphons are insonated through the transorbital window. The natural defect between the occipital bone and atlas vertebra forms the sub-occipital window that allows insonation of the vertebral (VA) and basilar

(BA) arteries. The BA can also be evaluated through the transforaminal approach. Extracranial ICA is insonated at the neck area via the submandibular approach (figures 3A–D).

Transtemporal Insonation

Place the 2-MHz TCD ultrasound transducer over the temporal area just above the zygomatic arch in front of the tragus of the ear and orient the transducer slightly upward and anteriorly (figure 3A). In power motion Doppler (PMD) mode, adjust the probe where the screen is filled with color signals between the depths of 30 and 80 mm. A red signal (toward the probe) between 40 and 65 mm represents the flow in the ipsilateral MCA, while the blue signal between 65 and 80 mm represents the flow from the ipsilateral ACA. In patients with good windows and favorable anatomy, a red signal beyond 80 mm represents flow in the contralateral A1 ACA segment.

The MI MCA stem usually lies at depths of 40–65 mm and is dependent on the size of the patients' head. It bifurcates into two divisions at a depth of 40–45 mm. Flow signals from the two M2 MCA branches are obtained by orientating the probe and optimizing the signal (figure 4A).

The ICA bifurcation is commonly observed at about 65 mm (range is 58–70 mm in adults). A bidirectional signal obtained at 60–70 mm represents ICA bifurcation. The ACA flow is usually away from the probe (blue signal with Doppler spectra below the baseline; see figures 4B and 4C).

After signals from the MCA and ACA have been obtained, the ultrasound probe is slowly orientated posteriorly by 10–30 degrees. Usually, there is a flow gap followed by flow signals from the PCA. Flow signals directed toward the probe represents P1 PCA, while those away from the flow arise from the P2 segment of the PCA (figure 4D); both segments are visualized at depths of 55–70 mm and are dependent on the probe orientation. An absence of the flow gap while moving the transducer posteriorly after the MCA/ACA evaluation usually represents flow signals from the PCOM.

Transorbital Insonation

Although no harmful effects have been reported with diagnostic TCD, the power output is reduced to 10 percent (or less than 17 mW/cm²) when the transorbital acoustic window is employed to evaluate the ophthalmic artery and the ICA siphon. The transducer is placed gently over the eyelid and it is angled slightly medial and upward. Flow signals at a depth of less than 60 mm, with a higher resistance pattern and moving toward the probe, represent the ophthalmic artery (figures 3B and 4G).

The sample volume has to be moved beyond a 60-mm depth to obtain signals from the ICA siphon. As the ICA siphon is a curved artery, the flow signals may be directed toward or away from the probe. Bidirectional signals may be obtained in some cases if the genu of the ICA siphon is insonated (figure 4H).

Suboccipital Insonation

It is convenient to turn the patient to one side and place the transducer just below and medial to the mastoid process. The probe is directed slightly medially and more horizontally toward the bridge of the nose or contralateral eye. This orientation will permit the technician to obtain flow signals from the ipsilateral VA between the depths of 50–75 mm. The flow signals are always moving away from the probe (figures 3C and 4E).

The BA may be insonated through the sub-occipital window by "going with the flow" in the ipsilateral VA by turning the probe slightly upward and medially from the depths of 75–110 mm. The BA flow signals may also be obtained through the transforaminal window by placing the transducer just below the occipital protuberance and orientating it toward the nasal bridge. Similar to vertebral arteries, the flow from the BA moves away from the probe (figures 3C and 4F).

Submandibular Window (Figure 3D)

In monitoring vasospasm after a subarachnoid hemorrhage, information regarding blood flow velocity from the extracranial internal carotid artery (eICA) should be obtained. Insonate the eICA at the neck at the level of the angle of the jaw with probe directed upward toward the head. Waveforms acquired will be moving away from the probe. The recorded

velocity is utilized to compute for the Lindergaard ratio for the diagnosis of a vasospasm.

Normal Spectral Waveform (See also chapter 3)

The normal spectral waveform shows a sharp systolic upstroke and stepwise deceleration with positive end-diastolic flow. The variables noted on typical TCD spectra are:

Peak systolic velocity (PSV in cm per second)

This is the first peak on a TCD waveform from each cardiac cycle. A rapid upstroke represents the absence of a severe stenotic lesion between the insonated intracranial arterial segment and the heart.

End-diastolic velocity (EDV in cm per second)

The end-diastolic flow velocity (EDV) lies between 20 and 50 percent of the peak systolic velocity (PSV) values, indicating a low resistance intracranial arterial flow pattern, which is seen in all major intracranial arteries.

Mean flow velocity (MFV in cm per second)

The mean flow velocity is calculated as EDV plus one-third of the difference between PSV and EDV. The MCA should have the highest MFV among all major intracranial arteries. For normal values of mean flow velocities, see table 1.

Pulsatility index (PI)

Flow resistance is usually assessed by PI, calculated by subtracting EDV from PSV and dividing the difference by MFV. This is the most frequently used TCD parameter to determine the flow resistance. The PI is independent of the angle of insonation, has no unit, and, if the value is more than 1.2, represents high-resistance blood flow.

Resistance index (RI)

The RI is another TCD parameter sometimes used to assess the flow resistance. It represents flow resistance distal to the site of insonation. RI is calculated by subtracting EDV from PSV and dividing the difference by PSV. Any value below 0.75 is normal.

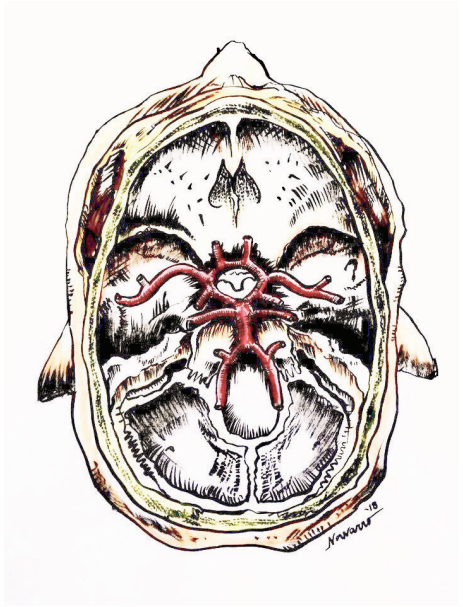


Figure 1. Circle of Willis at the base of the skull

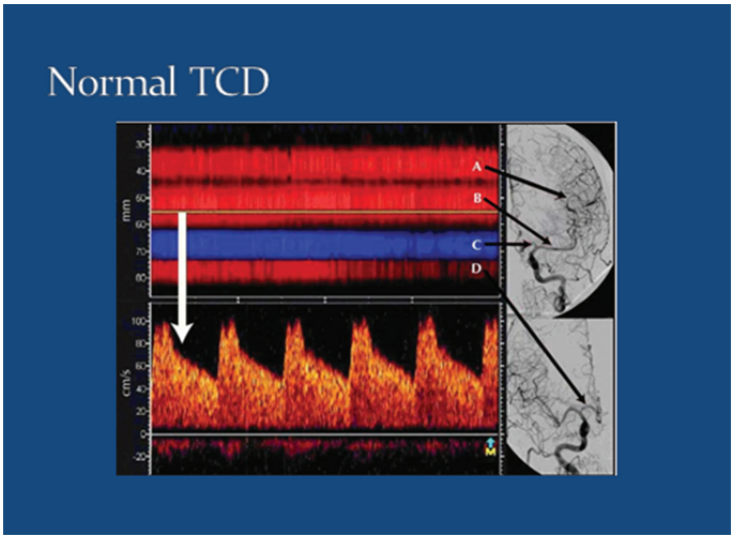


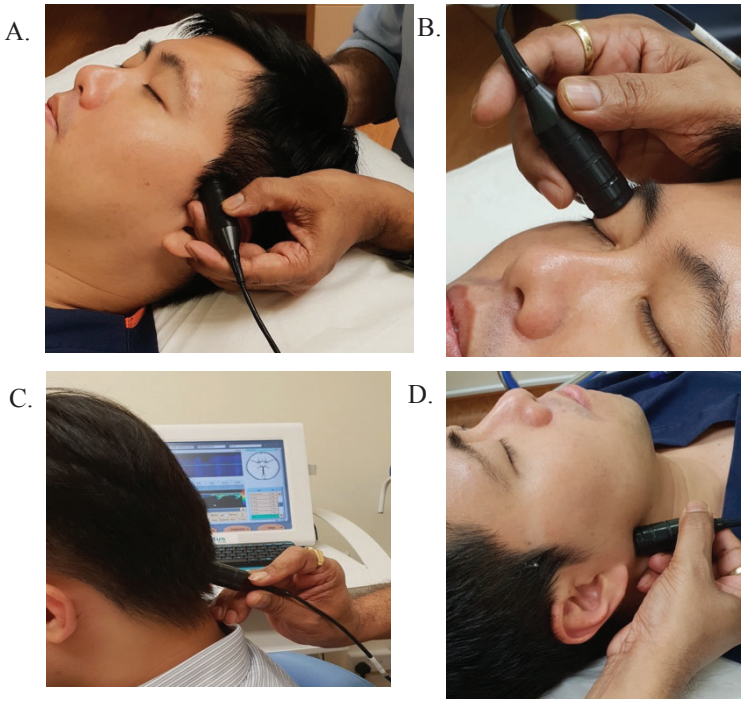
Figure 2. M-mode and normal waveforms

Table 1. Normal Depth, Direction, and Mean Flow Velocities at Assumed Zero-Degree Angle of Insonation of the Arteries of the Circle of Willis.

ARTERY	DEPTH (mm, adults)	DIRECTION	MEAN FLOW VELOCITY (CHILDREN) cm/second	MEAN FLOW VELOCITY (ADULTS) cm/second
M2 MCA	30–45	Bidirectional	<170	<80
M1 MCA	45–65	Toward	<170	<80
A1 ACA	62–75	Away	<150	<80
A2 ACA	45–65	Toward	NA	<80
ICA Siphon	60–65	Bidirectional	<130	<70
OA	40–60	Toward	Variable	Variable
PCA	55–70	Bidirectional	<100	<60
BA	80–100+	Away	<100	<60
VA	40–80	Away	<80	<50

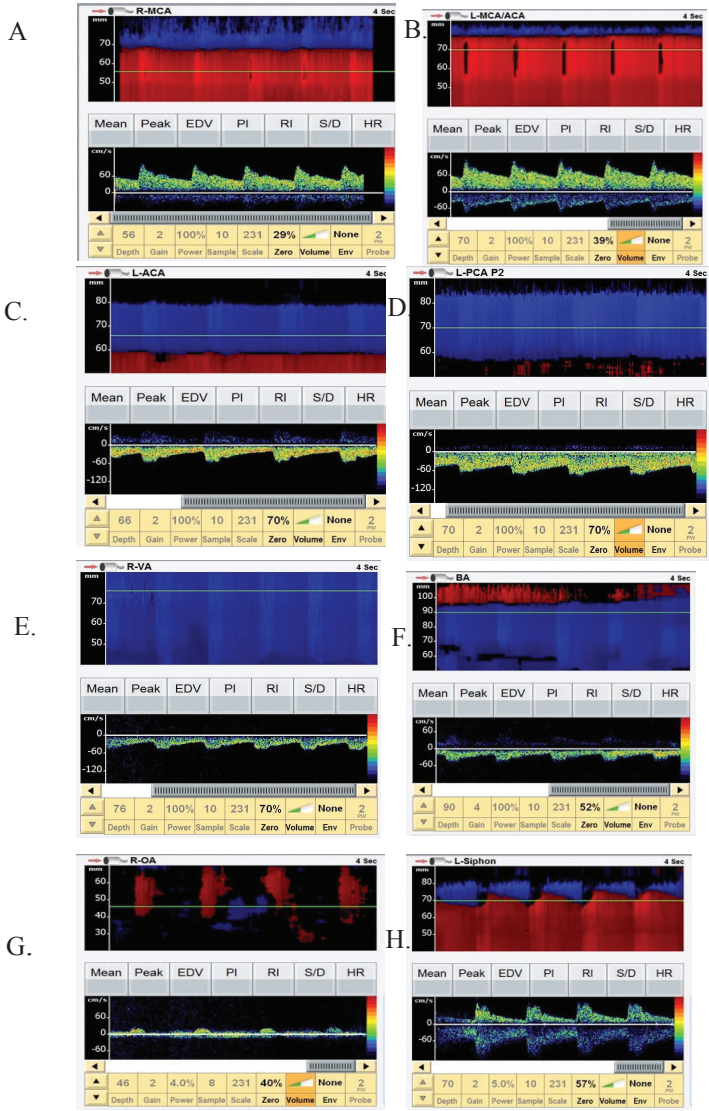
Adapted from table 6.1 of *Diagnostic Criteria for Cerebrovascular Ultrasound*, 2011. Alexandrov A.V. Blackwell Publishing Ltd.

Figures 3 A–D. Windows for insonation and probe position.



A. Transtemporal; B. Transorbital; C. Transforaminal; D. Submandibular

Figure 4 Representative waveform



A. Mid MCA; B. Bifurcation MCA/ACA; C. A1 ACA; D. P2 PCA;
E. Distal VA; F. Mid BA; G. Ophthalmic Artery; H. Carotid Siphon

NORMAL TCD SPECTRAL WAVEFORMS

ARVIND SHARMA,
MAN MOHAN MEHNDIRATTA
AND VIJAY K SHARMA

Transcranial Doppler (TCD) spectra represent the flow characteristics across a certain length of an intracranial artery. Pulsed ultrasound waves with a frequency of 2-MHz, emitted from the transducer (TCD probe), are reflected by the red blood cells (RBCs) flowing in the arterial lumen as rouleaux. Since the blood flow in the major intracranial arteries is laminar and parabolic, returned echoes have variable flow velocities (frequencies), being highest from the center of the lumen and lowest along the arterial wall. These are represented as "dots" of variable intensities (colors) on the screen. The composite of these drops is called the TCD Doppler (or flow) spectra.

An example of the normal TCD Doppler spectra is shown in figure 1.

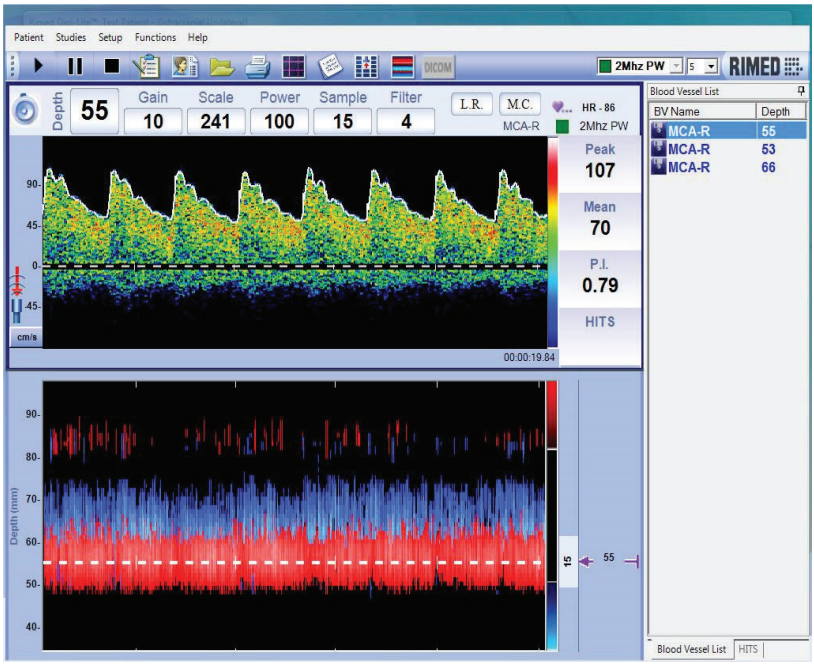


Figure 1. Normal TCD flow spectra obtained from right middle cerebral artery at a depth of 55 mm.

Various components of a typical TCD spectral frame are marked in figure 2.

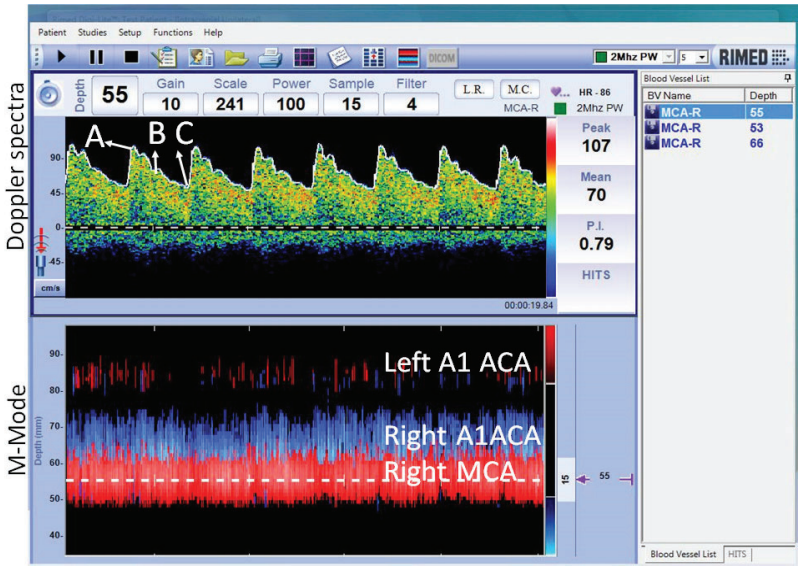


Figure 2. Normal TCD spectra. Doppler spectra on the upper panel represent the flow characteristics from the depth of 55 mm. The lower panel represents the M-mode, which shows the flow signals from all the intracranial arteries that lie between 35 and 95 mm of the ultrasound beam. Red signifies the flow toward the probe, while blue shows the flow away from the probe. The lower broad signal in red between 46 and 64 mm shows the flow in the right middle cerebral artery (MCA). The blue signal between the depths of 64-73 mm is obtained from the ipsilateral A1 segment of ipsilateral anterior cerebral artery (ACA). The faint red signal at the top of the M-mode panel represents flow in the contralateral A1 ACA.

TCD enables the monitoring of blood flow in each cardiac cycle. A normal spectral waveform will show a sharp systolic upstroke and stepwise deceleration with positive end diastolic flow. The variables noted on typical TCD spectra are:

1. *Peak systolic velocity (PSV in centimeter per second; point A in figure 2)*
   This is the first peak on a TCD waveform in each cardiac cycle. A rapid upstroke represents the absence of a severe stenotic lesion between the insonated intracranial arterial segment and the heart.
2. *Dicrotic notch (point B in figure 2)*
   The dicrotic notch is seen as a secondary upstroke in the descending part of a pulse-tracing that corresponds to the transient

increase in aortic pressure upon the closure of the aortic valve. It is seen in young patients and often disappears by about forty-five years of age as well as in hypertensive patients, whose case shows a rounded top in place of the sharp tip of the PSV (see figure 3).

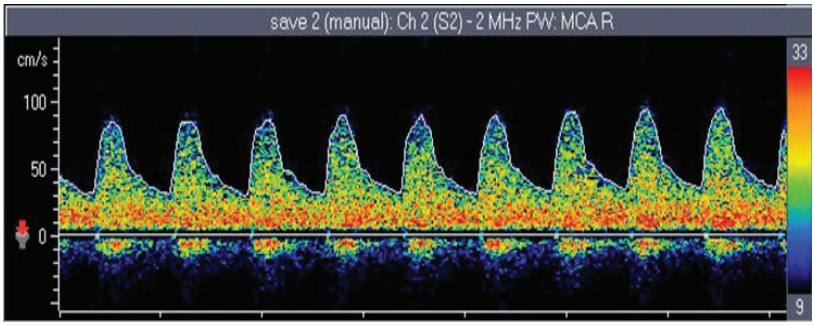


Figure 3. Flow spectra obtained from right MCA show sharp systolic upstroke and a rounded top (compare the sharp systolic top in figure 2). This patient was forty-nine years of age and suffered from hypertension for about five years.

3. *End-diastolic velocity (EDV in centimeter per second; point C in figure 2)*
   The EDV is normally between 30 and 50 percent of the PSV values, indicating a low-resistance intracranial arterial flow pattern, which is seen in all major intracranial arteries. The waveform represents a low resistance when the EDV is more than 50 percent of the PSV (figure 4A). It represents a high resistance when the EDV is less than 30 percent of the PSV (figure 4B).

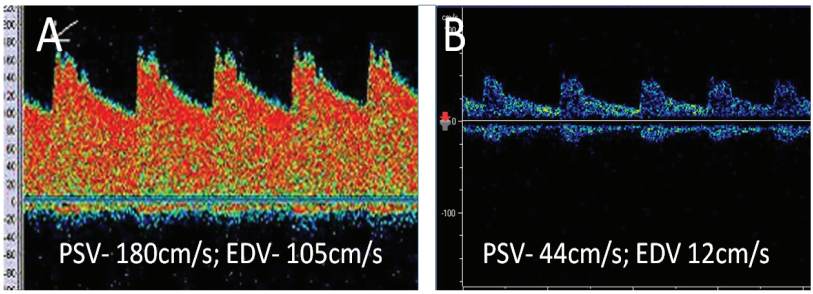


Figure 4. In panel A, the EDV is more than 50 percent of the PSV (low-resistance waveform). In comparison, panel B shows a high-resistance waveform (EDV is less than 30 percent of the PSV).

4. *Mean flow velocity (MFV in centimeter per second)*
   The MFV is calculated as EDV plus one-third of the difference between PSV and EDV. The MCA should have the highest MFV among all major intracranial arteries. Refer to figure 2: the PSV is 107 cm/s, while the EDV is 52 cm/s, giving rise to the MFV of 70 cm/s.

5. *Pulsatility index (PI)*
   Flow resistance is usually assessed by the PI, calculated by subtracting EDV from PSV and dividing the difference by MFV. This is the most frequently used TCD parameter to determine the flow resistance. Being a ratio, the PI is independent of the angle of insonation, has no unit, and has a normal range of 0.6–1.2. A value more than 1.2 represents high-resistance blood flow. Refer to Figure 2: the calculated PI of 0.79 represents a normal resistance flow. In comparison, the flow spectra in figure 4A are low-resistance flow (PI is 0.57), while panel B shows high-resistance flow spectra (PI is 1.41).

6. *Resistance index (RI)*
   The RI is another TCD parameter sometimes used to assess the flow resistance. It was described by Leandre Pourcelot. It is a measure of the resistance to blood flow caused by a microvascular bed distal to the site of measurement. It is used less commonly than PI and calculated by subtracting EDV from PSV and dividing the difference by PSV. The normal range varies between 0.4 and 0.75.

7. *Systolic-diastolic ratio (S/D)*
   This represents the ratio between the PSV and the EDV. It is not routinely used for clinical purposes.

8. *Heart rate (HR)*
   As HR may influence various flow parameters, it is an important variable noted on typical TCD spectra.

CAROTID DUPLEX ULTRASONOGRAPHY

SURYANARAYANA SHARMA P,
VIJAY K SHARMA AND LOKESH BATHALA

Introduction

Large artery disease is a notable stroke subtype and causes significant morbidity and mortality. Internal carotid artery (ICA) stenosis is seen in about 20 percent of patients with ischemic stroke. Carotid revascularization is an established treatment of patients with recently symptomatic and significant carotid artery stenosis.

Various imaging tools are available for an evaluation of the carotid arteries. Cervical duplex ultrasound (CDU) is an excellent bedside and noninvasive imaging tool that provides real-time information on the vessel wall, lumen, plaques, thrombus burden, and the degree of stenosis. Diagnosis of an ICA stenosis of 50-99 percent by CDU has sensitivity and specificity between 90 and 95 percent. When performed with transcranial Doppler (TCD), it provides vital information on the effect of carotid stenosis on cerebrovascular hemodynamics and helps the clinician in identifying patients who are at high risk for stroke recurrence. In this chapter, we will describe the techniques of performing cervical duplex sonography along with various diagnostic criteria for carotid stenosis, defining plaque morphology, measurement of intima–media thickness (IMT) and various pitfalls of the procedure.

Indications for CDU

- Patients with acute stroke or transient ischemic attack (TIA)- for the diagnosis of a relevant and significant carotid stenosis.
- Patients with symptomatic or asymptomatic carotid stenosis- for the assessment of the degree of stenosis, plaque morphology, lumen status, and need for revascularization.

- As a pre-operative evaluation of candidates for a major surgery, especially for coronary artery bypass grafting (CABG) and aortic root surgery- to assess the risk of perioperative stroke.

The Ultrasound Equipment

Current CDU systems use the pulse-echo technique. Pulses of ultrasound waves, produced by the piezoelectric crystal in the ultrasound transducer, travel into various tissues including the carotid arteries. The ultrasound pulses are reflected back and sensed by the transducer, in proportion to the flow direction, velocities, and flow characteristics, and they are represented on the screen as fast Fourier transform (FFT).

Various diagnostic modes used in the modern imaging systems are B-mode (brightness mode or grey-scale), color mode, the pulse wave Doppler, and power Doppler mode. Doppler spectral waveforms reflect the flow characteristics and speed in various layers within the gated sample volume.

Prerequisites for the test

- Test is performed in a well-equipped neurosonology laboratory by an experienced technologist under the supervision of either a neurologist or radiologist.
- Vital parameters such as the pulse rate, blood pressure and oxygen saturation are recorded.
- Level of consciousness of the patient is evaluated. Defer the test to a later date in uncooperative or hostile patients.
- A crash cart and emergency medicines are available in a close vicinity.
- The patient and attendants are updated about the test result.
- Relevant medical records (CT-scan/MRI/angiogram images) are available to the interpreting neurologist, if needed. Clinical indications for the test are clearly mentioned on the request form.

The Technique

- The patient should be in the supine position on the examination couch. Use a small pillow for the comfort of the patient. Assure the patient that CDU is a non-invasive test and that he can indicate to stop the test at any time in case of a discomfort. The examiner sits

at the head end of the patient to avoid any obstruction in the clinical management.

- Identify the head end of the transducer by a gentle tap on the head-end of the transducer. Observe the signals on the extreme upper-left edge of the image. This end of the transducer would be kept either "medial," i.e., in transverse plane imaging, or "up," i.e., cephalad in longitudinal imaging. This would enable the sonographer to maintain the anatomical orientation. The examination should start by identifying the jugular vein, thyroid gland, and common carotid artery (CCA). The internal jugular vein (IJV) can be easily compressed with probe pressure. A quick upward swipe in the transverse plane must be done from the proximal CCA to the carotid bifurcation. It will give valuable information about the arterial wall, plaque, patency of the vessel, and the level of bifurcation and guides the examiner in anticipating various findings and customizing the test accordingly. The technique is shown in figure 1.
- Once the transverse plane imaging is completed, it is essential to visualize the arterial lumen in the longitudinal plane. The examiner should scan from the most proximal CCA and move cephalad. Locate the bifurcation, flow-divider, carotid bulb, ICA, and external carotid artery (ECA). The ICA should be visualized as distally as possible in the neck. Longitudinal plane imaging can be done in either an anterior or a lateral approach. Compared with the anterior, the lateral approach is better because a longer segment of distal ICA can be evaluated. The CCA bifurcation as a typical Y is not seen commonly since the ICA and ECA overlap each other. The technique is shown in figure 2. The ICA and ECA are evaluated by the "rocking" movement of the transducer. Bilateral vertebral artery imaging is performed at proximal and distal segments. During B-mode imaging, atheromatous plaques and IMT are also evaluated. Representative images are stored for further analysis.
- B-mode findings are confirmed by the color mode. Color-gain settings should be adjusted to optimize the filling of the arterial lumen. Color mode imaging might help in the detection of hypoechoic/isoechoic plaques, which are often difficult to visualize on B-mode. Fresh blood clots are usually anechoic, appear dark on B-mode, and can be detected only on color mode as filling defects. Aliasing, represented as a mixture of colors is seen at the exit of stenosis, helps in locating the site of highest flow velocity. An area

of flow reversal in the carotid bulb is a normal finding, occurring due to the opening of a CCA (smaller lumen) into the bulb (larger lumen).

- After the B-mode and color-flow imaging, Doppler spectra are obtained from a small sample volume, placed in the center of the lumen by maintaining a steering angle parallel to the arterial wall. It shows the flow characteristics and velocities. Measurements of flow velocities should be avoided at the bends in tortuous arterial segments to prevent overestimation (remember that the flow appears to be always faster at the bends). Doppler spectra and velocities are obtained from proximal and distal CCA, carotid bulb, proximal and distal ICA, and ECA. An additional sampling is performed if more stenotic segments are seen.
- Doppler spectra reflect peak systolic velocity (PSV) and the end diastolic velocity (EDV). Because cerebral circulation is characterized by a significant blood flow during systole and diastole, ICA Doppler spectra demonstrate a "low resistance" pattern (EDV between 30 and 50 percent of PSV). The ECA supplies the muscular structures of the scalp and, hence, shows blood flow predominantly in systole, with a high resistance pattern (with EDV of less than 30 percent of the PSV). In the event of an uncertainty, the ECA may be differentiated from the ICA by "temporal tapping" maneuver. Doppler spectra obtained from CCA are usually a combination of the spectra of ECA and ICA.
- The IMT is measured on the far wall of distal CCA. It is measured as the distance between lumen/intima interface and the media/adventitia interface.
- Carotid plaque imaging is the most important aspect of the CDU in patients with suspected carotid stenosis**.** An atheromatous carotid plaque is described as a focal increase in the IMT (by more than 1.2 mm). Carotid plaques are described by their composition (echogenicity) and surface. Owing to their different compositions, plaques differ in their echogenicity. An anechoic region in a plaque represents either a necrotic or a hemorrhagic lesion, while the echogenic plaques predominantly contain fibrotic material.

  Qualitatively, carotid plaques are classified as:

  Type 1: Hypoechoic or echolucent, with uniform distribution of echoes;

  Type 2: Hypoechoic or echolucent, with heterogeneous distribution of echoes (≥50 percent of the plaque is hypoechoic);

Type 3: Hyperechoic or echodense, with heterogeneous distribution of echoes (≥50 percent of the plaque is hyperechoic);
Type 4: Hyperechoic or echodense, with homogeneous distribution of bright echoes; and
Type 5: Calcified plaque with bright echoes and acoustic shadowing.

The echogenecity of a plaque should be standardized against three reference structures: flowing blood for anechogenecity, sternocleidomastoid muscle for isoechogenecity and transverse vertebral apophysis for hyperechogenecity.

- **Plaque Surface:** The plaque is usually covered with a thin hyperintense rim of fibrous tissue, while "ulceration" corresponds to an irregularity or break on its surface. A significant ulceration recess (crater) must be at least 2 mm deep and 2 mm long. The plaque surface irregularity or ulceration exposes the thrombogenic layers, leading to thrombus formation and embolization.

- **Grading of ICA Stenosis:** The PSV is the most important parameter to gauge the severity of carotid stenosis. Additional criteria include EDV, spectral configuration, and carotid index. Degree of carotid stenosis is the most important predictor of cerebrovascular events. Duplex imaging is considered as the primary diagnostic modality for carotid stenosis. The criteria for quantifying carotid stenosis are determined through comparisons of ultrasonographic findings with digital subtraction angiography. Current diagnosis and grading of carotid stenosis on the CDU are based on the consensus criteria as shown in table 1. The CDU can be used to monitor the progression of carotid stenosis in asymptomatic patients. Furthermore, it can also be used for monitoring the results of carotid revascularization procedures.
- The relationship between carotid stenosis and flow velocities is not linear. A short arterial stenosis produces focal velocity increase. However, when the stenosis becomes severe, the flow velocities may show paradoxical reduction. Similar findings may be seen in patients with long segments or multiple contiguous stenosis in a single artery. This relationship is often represented as the Spencer's curve of cerebral hemodynamics.

Precautions while doing the test

- The B-mode, color Doppler, and power Doppler are complementary to one another in interpreting the results, as each modality addresses different aspects of carotid stenosis imaging.
- The position of the sample volume box in a normal artery should be in the center of the lumen and parallel to the vessel walls, whereas in a diseased vessel (with plaque), it should be aligned parallel to the direction of blood flow, which the velocities may be under- or overestimated.
- The PSV should not be measured on the sharp curves of a tortuous artery, as this may result in a false high-velocity reading. It may show changes in the color flow and even aliasing at the bends in the arteries. Do not measure velocities at the bends.
- Proximal CCA spectral waveforms show significant changes in patients with cardiac valvular regurgitant disease.
- Temporal tapping may differentiate the ECA from ICA. However, it should not be used as the sole diagnostic criterion as the tapping may be transmitted even into the ICA.
- In ICA occlusions, the ECA becomes a collateral pathway for intracranial circulation and might show a low-resistance flow pattern (internalization) and appear like the ICA, leading to a serious error.
- All the components of an ICA stenosis, such as the PSV, EDV, plaque surface characteristics, and the ICA-CCA ratio, should be interpreted together and not in isolation
- In patients with acute thrombus, B-mode may be normal, as thrombus is isoechoic. A color Doppler will show a filling defect. In difficult cases, these findings may be confirmed with high-resolution magnetic resonance imaging or digital subtraction angiography (DSA)/CT angiography (CTA).
- As the CDU is a real-time monitoring test, supervising clinician must act without delay in ordering further tests, optimizing treatment strategy in patients in which acute conditions such as acute thrombus in carotid artery or carotid dissections are diagnosed.
- The carotid web is a congenital shelf-like or web-like structure in the distal CCA/bulb and can be erroneously read as stenosis. Such errors should be avoided.

Clinical Implications of the Test

The CDU is an excellent bedside imaging tool in identifying high-risk lesions in patients with carotid artery stenosis and in guiding clinicians in therapeutic decisions. Getting above the plaque in carotid stenosis implies that the disease is largely limited to extracranial carotid arteries and revascularization procedures may be planned. If the sonographer is unable to determine the distal end of the plaque on the CDU, it suggests that the affected artery may not be amenable to CEA, as the plaque may be crushed and embolized during cross-clamping. After an analysis of the degree of stenosis, other tests like the TCD, micro-embolic signal monitoring, and vasomotor response testing helps the clinician in assessing the cerebrovascular hemodynamics and prognosticating the clinical outcome in patients with CAS. In patients with asymptomatic carotid disease and steal phenomenon, early revascularization may be considered. It is of great clinical significance in patients undergoing CABG or aortic root surgery, as plaques in the proximal CCA increase the risk of perioperative embolic stroke.

CDU has some limitations in the clinical practice. It is operator-dependent, and thorough knowledge and understanding of the anatomy and clinico-radiological correlation help in minimizing the diagnostic errors. In patients with short neck and increased neck circumference as in obesity, insonation may be suboptimal. It should be correlated with other standard imaging tools such as CTA/DSA in inconclusive cases.

Case Report

A fifty-eight-year-old male, known hypertensive and chronic smoker, presented with a transient episode of left-sided weakness, which improved in thirty minutes. No acute stroke was noted on the CT scan. About two weeks ago, he was investigated for possible coronary artery disease. Coronary angiogram revealed triple vessel disease, and CABG was planned. As part of the workup for TIA, extracranial CDU revealed large heterogenous plaque at proximal ICA resulting in severe stenosis across the lesion (>90 percent) with blunted flow signals in distal ICA (figures 3 and 4). TCD revealed blunted flow in right middle cerebral artery (MCA). Vasomotor reactivity (VMR) testing with hypercapnoeic challenge showed exhausted vasodilatory reserve in right MCA (breath holding index is 0.3), suggestive of a high risk for perioperative stroke during the planned CABG. Therefore, we decided for a simultaneous CABG and CEA. Surgery was uneventful. He has remained well for the past year.

Suggested Reading

- Grant EG, Benson CB, Moneta GL, Alexandrov AV, Baker JD, Bluth EI, et al. "Carotid artery stenosis: Gray-scale and Doppler US diagnosis–Society of Radiologists in Ultrasound Consensus Conference." *Radiology* 229 (2003): 340–46.
- Bathala L, Mehndiratta MM, Sharma VK. Cerebrovascular ultrasonography: Technique and common pitfalls. *Annals of Indian Academy of Neurology* 16 (2013): 121–127.

FIGURES:

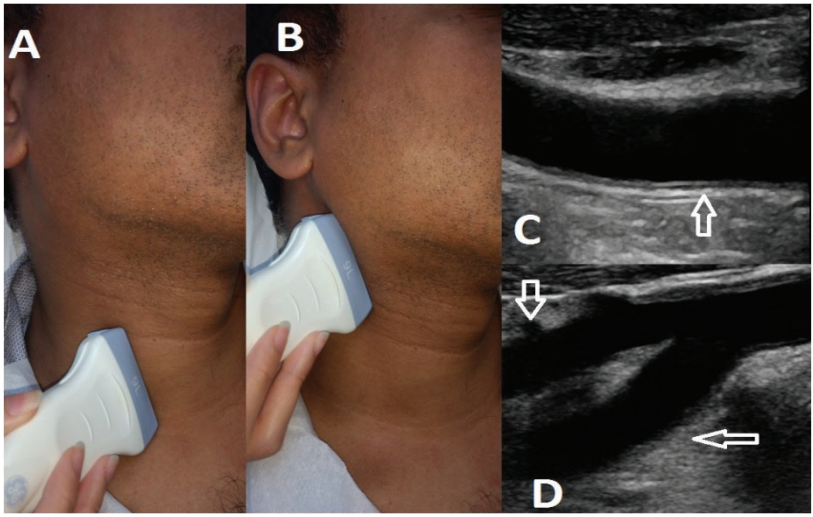


Figure 1: CDU B mode in transverse plane. A and B: patient neck position and probe position at proximal CCA and carotid bifurcation respectively. C: B-mode imaging in transverse plane showing proximal CCA (right sided arrow). D: transverse plane at carotid bifurcation showing ICA (left sided arrow) and ECA (upward arrow).

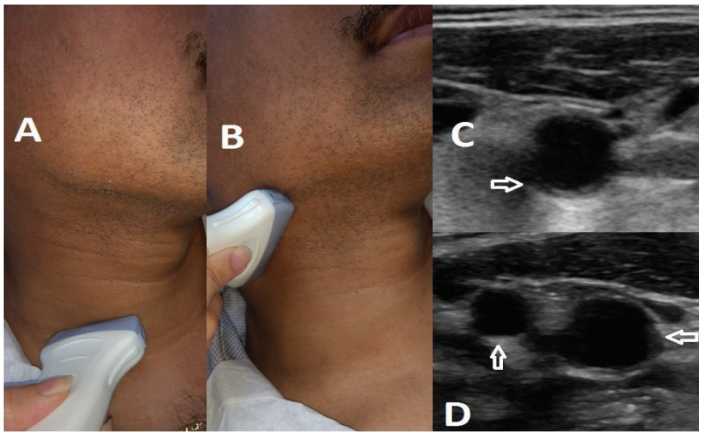


Figure 2: CDU on B-mode in longitudinal plane. A and B: patient neck position and probe position at proximal CCA and carotid bifurcation, respectively. C: B-mode imaging in longitudinal plane showing proximal CCA (upward arrow). D: longitudinal plane at carotid bifurcation showing ICA (left sided arrow) and ECA (downward arrow).

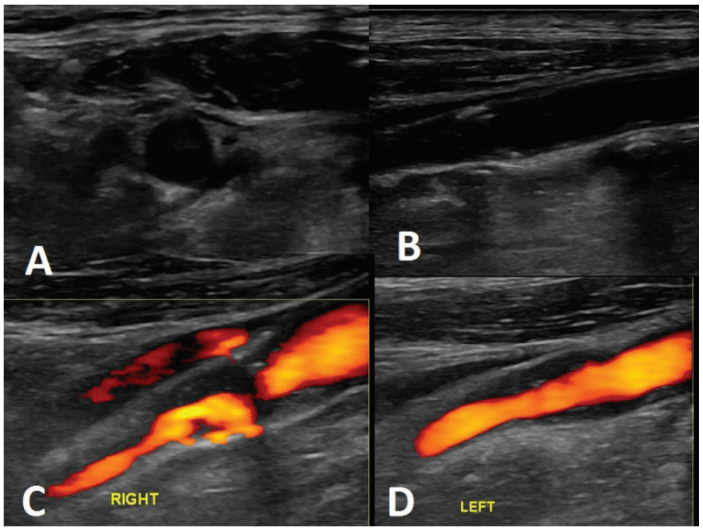


Figure 3: CDU on B-mode Doppler demonstrating irregular heterogenous plaque at ICA causing significant narrowing of the lumen as seen in transverse (A) and longitudinal plane (B). Power Doppler showing reduced flow in right ICA (C) and normal flow across left ICA (D).

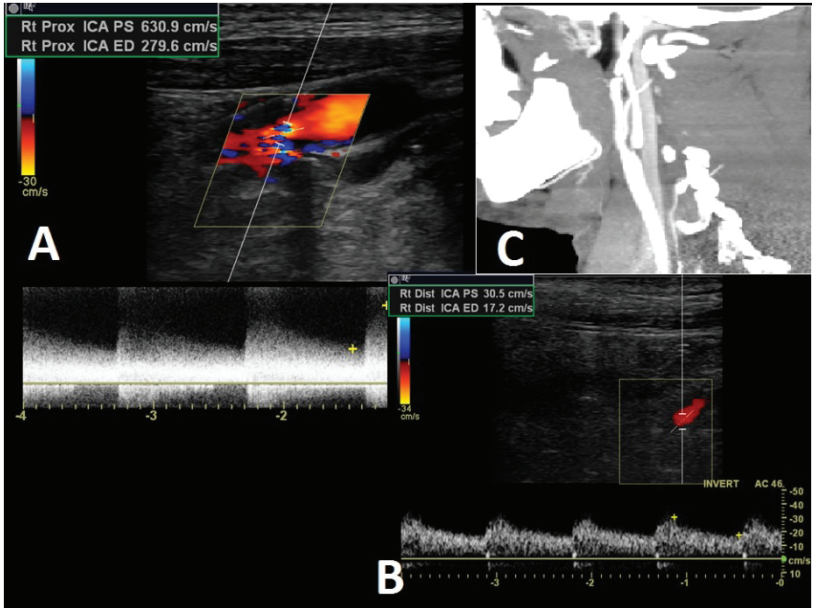


Figure 4: Color and pulsed-wave Doppler across the stenosis shows high PSV/EDV (630.9/279.6cm/s) (A). Distal ICA Doppler shows blunting of waveforms (30.5/17.2 cm/s) suggesting severity of stenosis (B). CT Angiogram correlates of the ICA stenosis (C).

Table 1: Assessment of severity of Carotid stenosis

Degree of stenosis (%)	ICA PSV (cm/sec)	ICA/CCA PSV ratio	ICA EDV (cm/sec)	Plaque
50-69%	125-230	2.0-4.0	40-100	>50% diameter reduction
70-99%	>230	>4.0	>100	>50% diameter reduction
Near Occlusion	Low/ undetectable	Variable	Variable	Near total occlusion of the lumen

TRANSCRANIAL COLOR-CODED DUPLEX SONOGRAPHY

VIJAY K SHARMA AND JOSE C. NAVARRO

The transcranial Doppler (TCD) is often criticized for being a blind procedure, which makes it highly operator-dependent. The introduction of the power-motion mode Doppler (PMD) into the current, commercial TCD systems has removed several problems faced by the original single-gated TCD machines. Since the PMD-TCD permits simultaneous evaluation of multiple contiguous branches of the circle of Willis, the TCD is no longer considered a blind technique and operator-dependent as it had been often criticized of being. Of course, the individual intracranial arteries are not visualized, and inference is drawn from the depth, flow, and spectral characteristics.

Modern duplex machines provide a 2-3 MHz spectral array probe (figure 1), which may be used for direct visualization of the intracranial arteries. Once a relevant parenchymal field is seen, the color mode is activated to depict the intracranial arteries. Then the pulsed Doppler ultrasound is applied on the arterial segment of interest to obtain the flow spectra. This technique of cerebrovascular ultrasonography is called the transcranial color-coded duplex (TCCD).

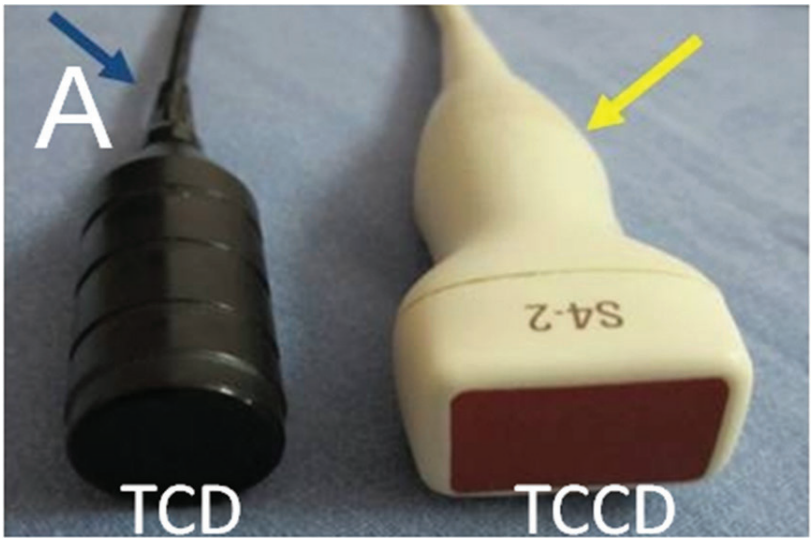


Figure 1. Difference between the TCD and TCCD probes

Test Performance

The TCCD procedure is explained to the patient. The patient is requested to lie on a comfortable bed. Various intracranial arteries are insonated via the acoustic windows on the skull: temporal, orbital, and foraminal. With as much importance as the TCD, the TCCD is performed following a standard protocol, and temptation should be avoided to jump to the artery or side of the intracranial vascular pathology. Best B-mode, color mode, and Doppler spectral signals from various intracranial arteries are saved for final reporting.

In preparing for a TCCD report, the sonographer is advised to record the following:

1. The correct name, age, and gender of the patient. The hospital-record number or the number allocated to the patient on the TCCD machine, or both, should be recorded on the report form. Verify or confirm whether the requested procedure is appropriate for the patient's presentation or suspected diagnosis.
2. Relevant history and the indication for the TCCD examination.
3. Current medications (especially if the patient is on beta blockers and vasodilators).

4. Some TCCD laboratories record information on various vascular risk factors like diabetes mellitus, hypertension, dyslipidaemia, ischemic heart disease and patient's smoking habits.
5. Blood pressure (BP), preferably on both arms.

Examination Procedure

The sequence of various acoustic windows employed for performing the TCCD is similar to the TCD.

The transtemporal window (see figure 2) is considered most informative because a large number of intracranial arteries (MCA, ACAs, PCAs, and the top-of-basilar) can be evaluated.

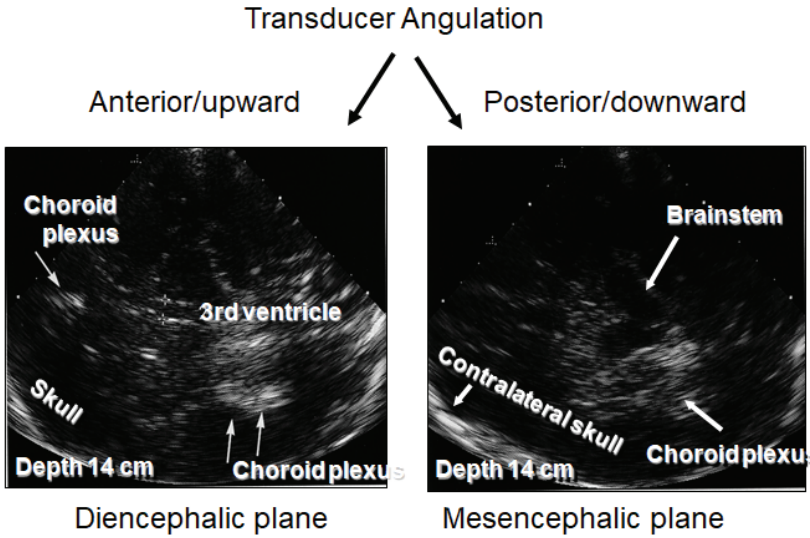


Figure 2. Transtemporal window for the TCCD.

The TCCD probe can be moved in a cephalo-caudal direction to obtain axial sections, while the antero-posterior movement provides coronal views of the circle of Willis. Often, the sonographer uses intermediate coronal and axial sections for best visualization of an individual artery or many intracranial arteries.

The sonographer's first aim during the transtemporal TCCD is to obtain a section in the diencephalic view that shows two parallel bright lines in the center (at a depth of about 75 mm), which represent the third

ventricle. This landmark ensures that color-Doppler imaging will show the major branches of the circle of Willis (see figure 3).

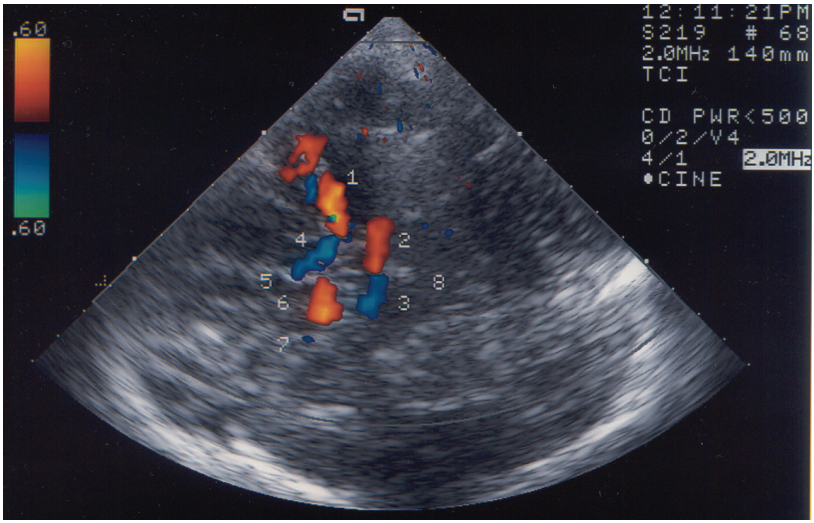


Figure 3. Normal appearance of various branches of the circle of Willis on the TCCD via right transtemporal insonation. Various arteries seen on this frame are: (1) Right MCA; (2) right P1 PCA; (3) left P1 PCA; (4) right A1 ACA; (5) A2 ACA; (6) left A1 ACA; and (7) left MCA.

An example of MCA stenosis on the TCCD is shown in figure 4.

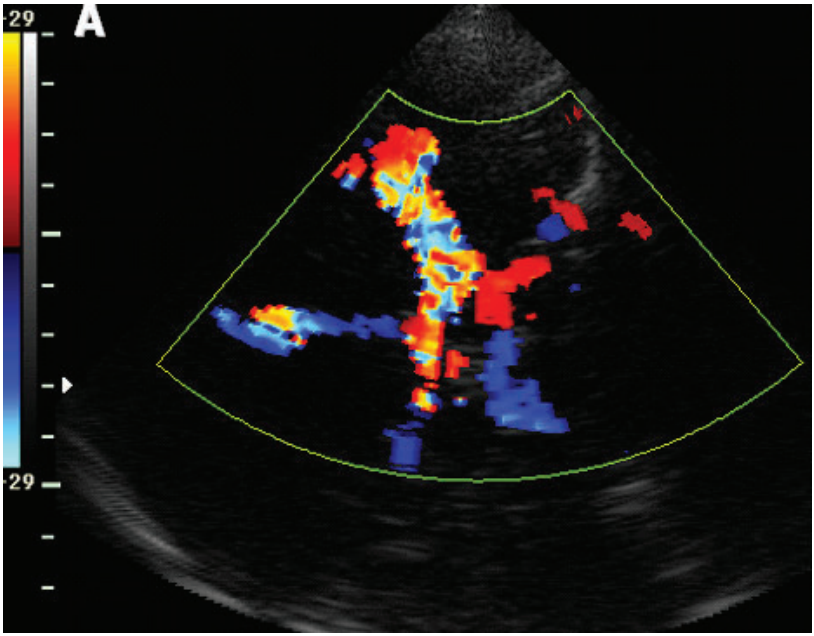


Figure 4. Aliasing (turbulence) is noted in the right MCA, suggestive of an underlying stenosis.

Similar frames can be obtained for the vertebrobasilar system via the transforaminal window (see figure 5).

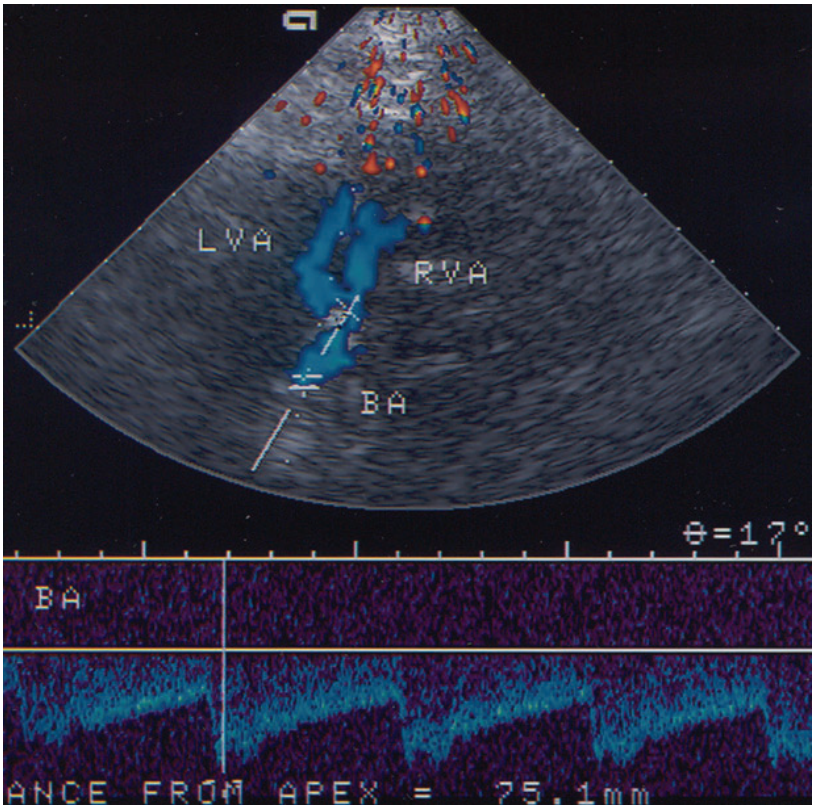


Figure 5. TCCD frame obtained from the vertebral and basilar arteries via the transforaminal acoustic window. Doppler spectra are obtained from the proximal basilar artery.

After completing the diagnostic TCCD examination, the sonographer obtains various hemodynamic parameters from individual arterial segments. The number of arterial segments for individual intracranial arteries should be determined by individual laboratories.

In addition to the hemodynamic parameters from various intracranial arteries, the sonographer should mention the following in the report:

a. Bruit in an artery.
b. Irregular cardiac rhythm (remember that the TCD examination may serve as the only test to demonstrate paroxysmal atrial fibrillation).
c. Any abnormal flow, especially if a blunted signal is obtained.
d. Whether the patient was uncomfortable during the test.

Every neurovascular laboratory is encouraged to develop its own TCCD report form. Whether the TCCD report contains the frames for individual flow spectra from various intracranial arteries should be left for the individual laboratories to decide.

TCCD reporting

A TCCD result is often reported by neurologists who are credentialed in neurosonology. The reporting of the TCCD follows the same principles as that for the TCD. A TCCD report may be either descriptive or just conclusive. Descriptive TCD reports are often considered better, since they may be easily understood by other clinicians involved in patient-care as well as neurologists who are not well-trained in neurosonology.

Is TCCD better than the TCD?

Although the TCCD visualizes an intracranial arterial segment from where the Doppler spectra are obtained, it has several limitations that lead to the authors' laboratories not performing TCCD routinely. Some of these limitations are:

1. Temporal acoustic windows permit only partial penetration of the ultrasound waves. The windows become clinically suboptimal in most of the patients of Asian ancestry, especially middle-aged females. We feel that failure rates for the TCCD are much higher than the TCD.
2. The intracranial arteries are very small in diameter, with the MCA being the largest artery with an average diameter of 3–5 mm in adults. Current TCCD systems, especially when a lower frequency ultrasound is used, lack the resolution to demonstrate the real anatomical narrowing. Instead, the diagnosis is made on turbulent flow or disturbed Doppler spectra, both of which are better observed on the TCD. Perhaps future TCCD machines will provide better screen resolution to overcome this limitation.
3. There are no validated criteria for the diagnosis of stenosis on the TCCD. Just to match the TCD diagnostic criteria, sonographers should not adjust the Doppler angle, which almost defeats the advantage of the TCCD.
4. There are no head-frames that can be used to hold the TCCD transducers for prolonged monitoring. Thus, the TCCD cannot be used for emboli monitoring, detection of a right-to-left shunt,

perioperative monitoring, or any other functional studies of cerebral vasomotor reactivity.

TCD Microembolic Signal Detection

Hui Meng Chang and Kay Sin Tan

Introduction

Microembolic signal (MES) monitoring with standard transcranial Doppler (TCD) equipment gives additional information in a variety of clinical settings. An MES is defined, on consensus, as a short-lasting, unidirectional intensity increase (> 3 dB) within the Doppler frequency spectrum. There have been attempts to classify embolic signals according to the content (whether gaseous or solid), but this has not been found to be clinically useful. The detection of MES has implications in the clinical situations described below.

Definition

MES diagnostic criteria according to the International Consensus Committee in 1995 are as follows:

1. It is transient, usually lasting less than 300 milliseconds.
2. The intensity of a Doppler MES is usually at least 3 dB higher than that of the background blood flow signal and depends on the characteristics of the individual microembolus.
3. The signal is unidirectional within the Doppler velocity spectrum.
4. An MES is accompanied by a "snap," "chirp," or "moan" on the audible output.

Indications for MES Monitoring

Monitoring for MES may be clinically useful in the following situations:

1. Acute stroke and transient ischemic attack with existing intracranial or extracranial stenosis

The presence of MES in symptomatic and asymptomatic middle cerebral artery stenosis predicts further ischemic stroke or transient ischemic attack

(TIA) (adjusted odds ratio, 8.45; 95% CI, 1.69 to 42.22; P = 0.01). In a meta-analysis, the presence of MES in the middle cerebral artery of patients with acute stroke predicted a higher risk of recurrent ischemic event (OR, 2.44; 95% CI, 1.17–5.08; P = 0.02). MES were more likely to be found in symptomatic extracranial carotid artery stenosis and ulcerated plaques, validating these known markers of increased risk.

2. Evaluation of therapy

MES monitoring can be used as a surrogate marker for the efficacy of antithrombotic therapies, if selected patients have MES present at the baseline. In a case study, MES positivity in symptomatic intracranial arteries (intracranial internal carotid artery or middle cerebral artery) was used to assess efficacy of dual antiplatelet (aspirin and clopidogrel) therapy, as compared to monotherapy with aspirin. On day-2, there was a relative risk-reduction of over 40 percent in the number of patients who were MES positive, in favor of dual antiplatelet therapy.

Another study included MES positive patients, with recently symptomatic extracranial carotid stenosis ($\geqslant$ 50 percent). Similarly, dual antiplatelet therapy with clopidogrel and aspirin was more effective at reducing MES than monotherapy with aspirin. There was a 40 percent relative risk reduction in the MES count on day seven, in favor of dual antiplatelet therapy.

3. Asymptomatic extracranial carotid artery stenosis

The presence of MES in asymptomatic severe carotid stenosis identifies a subgroup who are at higher-risk for subsequent ischemic events. The annual risk of ipsilateral stroke or TIA was 7.13 percent in patients with MES and 3.04 percent in those without.

4. Identification of structural abnormalities

Presence of right-to-left shunting (RLS). A patent foramen ovale (PFO) is present in about 25 percent of the population. However, in young patients with cryptogenic ischemic strokes, the prevalence of a PFO increases to 40 percent. Paradoxical embolism is believed to be the mechanism of stroke. RLS may occur at rest or with provocative maneuvers that increase the intrathoracic pressures, such as the Valsalva maneuver or coughing. TCD studies using agitated saline injected intravenously can help detect the presence of microemboli in the

intracranial arteries. The presence of these microemboli can support the existence of RLS.

TCD is a reliable diagnostic tool for detecting RLS. Bivariate meta-analysis of twenty-seven studies found high sensitivity (97 percent) and specificity (93 percent) for TCD, with transesophageal echocardiogram (TEE) as the gold standard. Up to 40 percent of the studies used agitated saline for detection of RLS (on TCD and TEE), while the remaining studies used contrast agents. TCD may actually be more sensitive than TEE for the detection of RLS, as sedation used for TEE could contribute to a sub-optimal Valsalva maneuver.

Intracranial blood-flow monitoring during procedures and in traumatic vascular injuries and peri-operative monitoring in carotid endarterectomy. TCD monitoring during carotid endarterectomy has been used since the 1980s. Both MES and changes in blood-flow velocities may be detected, predicting perioperative stroke with a specificity of 72.7 percent and sensitivity of 56.1 percent, according to a recent meta-analysis. MES are also detected during arterial dissection, arterial manipulation, and insertion or release of arterial clamps.

Blunt cerebrovascular injury. Blunt trauma to the internal carotid artery is associated with a 5-10 percent risk of stroke. TCD-MES monitoring in the affected internal carotid artery may identify the affected vessels with higher risk of stroke. A retrospective analysis at a level-one trauma center showed that the presence, number, and persistence of MES detection, after isolated internal carotid artery injuries, predicted significantly higher risk of stroke as compared to those without MES.

Procedure for MES TCD monitoring

MES monitoring is performed with the traditional single-channel TCD or the power M-mode Doppler (PMD). Both modalities are available in most of the modern TCD machines. The single gate TCD uses the Doppler frequency shift. The PMD-TCD records from numerous gates simultaneously along the path of the ultrasound beam. Data are displayed with multiple depths on the vertical axis and time on the horizontal axis. The power of the Doppler shift signal is displayed as color intensity, with red for blood flowing toward the probe and blue away from the probe. MES on PMD are displayed bright as sloping tracks and evidence that the MES travel through the arteries with time.

The standard procedures employed for MES monitoring include the following:

1. Perform a routine anterior circulation TCD examination and establish that the temporal window is adequate.
2. Set the headframe in a routine manner using the monitoring probe.
3. Fixing the headframe involves the central knob on the headgear and the smaller screws to hold the monitoring probe firmly.
4. Most TCD devices have single and multi-gated display.
5. A depth of 56 mm is ideal for the middle cerebral artery and should be used bilaterally.
6. Select lowest gain to allow easier visualization of MES, especially those with lower intensity.
7. Duration of monitoring should be standardized (preferably for at least 30 minutes).

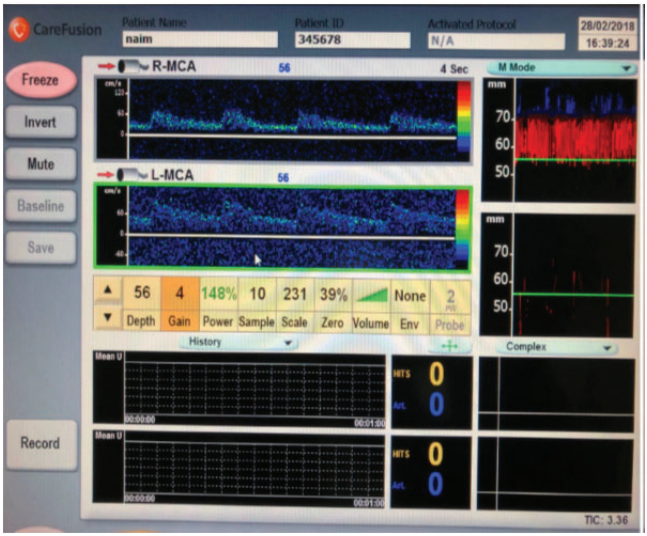


Figure 1. TCD monitoring interface and suggested parameters.

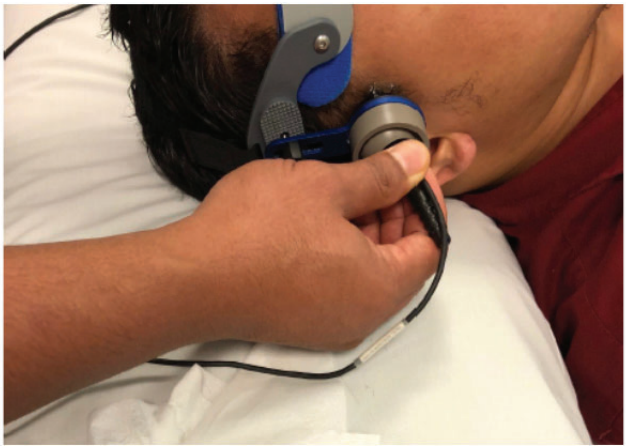


Figure 2. Use of attached TCD probe to improve insonation angle.

Figure 3. Secure fixation of TCD probes with central knob.

What does a positive study mean?

The presence of one microembolic signal, in the earlier described settings, constitutes a positive test. The clinical inferences are described in accordance with the clinical scenario. Follow-up monitoring may be required.

Grading scales used for MES detection in an RLS are the International Consensus Criteria (ICC) and the Spencer Grade. The grades, in ascending order, are proportional to the size (functional) of the shunt (see table 1).

Table 1.

ICC Grade	No of MES	**Spencer Grade**	No of MES
0	0	0	0
1	1–10	1	1–10
2	> 10, but no curtain	2	11–30
3	Curtain	3	31–100
		4	101–300
		5	>300

All practitioners should recognize that MES detection is influenced by numerous factors, which include body position, presence of cardiac prosthetic valves (especially if they are metallic), time from symptomatic event, co-existing antiplatelet and anticoagulation therapy, and procedures involving cardiac and cerebrovascular structures.

Future aspects

In the future, TCD software may incorporate artificial intelligence to optimize vessel insonation in conditions of poor ultrasound windows. It can also aid in the interpretation of TCD waveforms and microembolic signals. This will enhance the utility of TCD for research and clinical purposes.

Acknowledgment

The authors would like to thank their respective senior technologists, Mohd Azly Yahya and Gan Moi Pin for their contributions in this chapter.

Suggested Readings

1. Basic identification criteria of Doppler microembolic signals. Consensus Committee of the Ninth International Cerebral Hemodynamic Symposium. *Stroke* 26 (1995):1123.
2. King A, Markus HS. Doppler Embolic Signals in Cerebrovascular Disease and Prediction of Stroke Risk. A Systematic Review and Meta-Analysis. *Stroke* 40 (2009):3711-17.
3. Katsanos AH, Psaltopoulou T, Sergentanis TN et al. Transcranial Doppler versus transthoracic echocardiography for the detection of patent foramen ovale in patients with cryptogenic cerebral ischemia: A systematic review and diagnostic test accuracy meta-analysis. *Ann Neurol* 79 (2016): 625-35.
4. Moehring MA, Spencer MP. Power M-Mode Doppler (PMD) for observing cerebral blood flow and tracking emboli. *Ultrasound in Med & Biol* 28 (2002): 49.
5. Bonow RH, Witt CE, Mosher BP et al. Transcranial Doppler microemboli monitoring for stroke risk stratification in blunt cerebrovascular injury. *Critical Care Medicine* 45 (2017): e1011-17.
6. Lao AY, Sharma VK, Tsivgoulis G et al. Effect of body positioning during transcranial Doppler detection of right-to-left shunts. *Eur J Neurol* 14 (2007):1035-39.
7. Sliwka U, Lingnau A, Stohlmann WD et al. Prevalence and time course of microembolic signals in patients with acute stroke. A prospective study. *Stroke* 28 (1997): 358-63.
8. Stygall J, Kong R, Walker JM et al. Cerebral microembolism detected by transcranial Doppler during cardiac procedures. *Stroke* 31 (2000): 2508-10.
9. M Seera, C P Lim, KS Tan, and WS Liew. Classification of transcranial Doppler signals using individual and ensemble recurrent neural networks. *Neurocomputing* (2017): 1–8.

BUBBLE TEST IN RIGHT-TO-LEFT SHUNT

JOSE C. NAVARRO
AND ANNABELLE LAO-REYES

Introduction

The diagnosis of ischemic stroke secondary to right-to-left shunt (RLS) due to a patent foramen ovale (PFO) should be suspected only when the following conditions have been established:

a. the source of venous thrombosis,
b. demonstration of RLS from a PFO,
c. an arterial stroke.

The demonstration of an RLS can be easily and reliably demonstrated by performing a bubble test. Unlike the transesophageal echocardiography (TEE) where the patient is partially sedated, the bubble test with transcranial Doppler (TCD) can be performed on an awake patient, making it superior to the TEE. Furthermore, a more effective Valsalva maneuver can be performed to detect even a small RLS. The sensitivity and specificity of TCD for detecting a PFO are 90 percent and 92 percent, respectively.

Procedure

RLS testing is usually performed in both supine and sitting positions. The following materials should be readily available:

a. IV cannula, preferably a large-bore and inserted into the antecubital vein;
b. Three-way stopcock;
c. 2 vials of sterile normal saline solution (10 ml);
d. Two 10-cc syringes with a Luer lock; and
e. TCD machine with 2 MHz pulse-wave Doppler diagnostic hand-held probe, monitoring probes and an adjustable headframe.

1. Perform a routine TCD exam to establish the presence of adequate temporal window. For the single-gated TCD machine, set the insonation depth at 55 mm. Most of the current TCD machines are equipped with the power motion-mode Doppler (PMD), which enables simultaneous multi-gated sampling from multiple depths. Keep the gain lower to identify even the microebolic signals (MES) with lower intensities.
2. Set the headframe for adequate and steady fixed position.
3. Insert a large bore IV canula into the antecubital vein.
4. Aspirate 9 cc of normal saline and add 1 cc of air.
5. Through the three-way stopcock, connect the syringe with the normal saline and air to one port and another syringe to the other port. The third port is connected to the IV line. Obtain 9 cc of normal saline, 1 cc of air, and 0.5 cc of blood mixture (adding a few drops of the patient's blood to this solution is preferred since it increases the life span of microbubbles) by opening the three-way stopcock between the two syringes and mixing vigorously between the two syringes 15–20 times. Once the mixture is ready, open the port connected to the IV line and inject the saline-air-blood contrast mixture. Wait for about six seconds for the microbubbles to reach the right atrium. Also, observe for the appearance of MES during this period.
6. Ask the patient to perform a strong Valsalva maneuver for 4–6 seconds and observe the TCD for MES for the next twenty-two seconds.
7. The test may be performed in both supine or sitting upright positions.
8. Count the number of MES for a functional grading of the RLS.

What is a positive test?

The appearance of MES constitutes a positive test. The number of MES observed on the TCD during the entire recording indicates the functional grading of the RLS and is often considered proportional to the size of the shunt. Currently, there are two grading systems to quantify the RLS.

International consensus criteria (ICC)

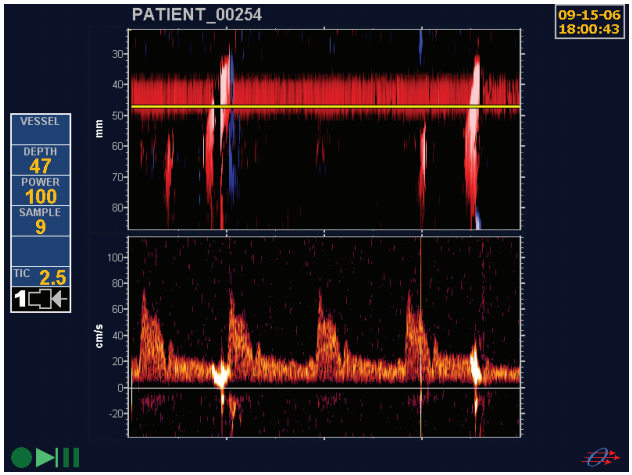
Grade 0: no MES
Grade 1: MES count of 1–10
Grade 2: MES count of 11–30

Grade 3: more than 30 with shower or curtain appearance

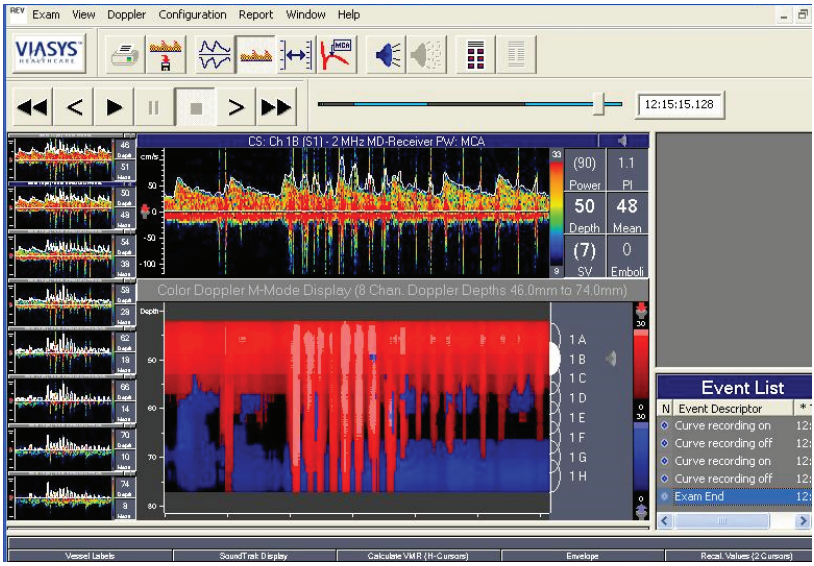
Spencer's logarithmic scale
Grade 0: no MES
Grade 1: MES count of 1–10
Grade 2: MES count of 11–30
Grade 3: MES count of 31–100
Grade 4: MES count of 100–300
Grade 5: MES count of more than 300

Criteria	**Sensitivity (%)**	**Specificity (%)**	**PPV (%)**	**NPV (%)**	**Accuracy (%)**
International Consensus (any positive MES count)	100%	72.4%	32.1%	100%	64.1%
Spencer Logarithmic Scale Grade III+ (> 30 MES)	100%	91.3%	60%	100%	85.5%

Figure 1. MES should be random occurrences in the cardiac cycle, for a brief durations (<0.1 s) and with high intensity (>3 dB over compared to the background Doppler spectra). It should also be unidirectional. The characteristic sound of MES is a whistle, chirp, or a pop.



A small grade shunt (about five MES) noted on this TCD screen.



A large grade right-to-left shunt noted on this TCD screen (about twenty MES are noted on one screen).

EVALUATION OF INTRACRANIAL STENOSIS

NIJASRI C SUWANWELA, JOSE C NAVARRO AND VIJAY K SHARMA

Introduction

Intracranial stenosis leads to cerebrovascular events in at least 10 percent of all ischemic strokes in North America. However, it is one of the most common causes of stroke worldwide, especially among patients of Asian, African, or Hispanic ancestry. Importantly, intracranial stenosis is associated with an increased risk (10–15 percent a year) of recurrent stroke.

Digital subtraction angiography (DSA) remains the gold standard for the diagnosis of intracranial stenosis. Computerized tomographic angiography (CTA) is a sensitive and specific tool for the assessment of intracranial stenosis. Magnetic resonance angiography (MRA) can also detect intracranial stenosis with reasonable accuracy and does not require intravenous contrast injection.

The transcranial Doppler (TCD) is a reliable, widely available, and portable technique for the diagnosis of intracranial stenosis. TCD detects stenosis by identifying various characteristic alterations in the Doppler signals, which include focal increases of velocity, local spectral turbulence, pre- and/or post-stenotic changes, and various collateral flow patterns. Sensitivity, specificity, and positive predictive value and negative predictive value of TCD are generally higher in anterior circulation than in the vertebrobasilar circulation because of the lesser tortuosity and congenital asymmetry of the former.

TCD in focal intracranial disease

The basic aim of TCD in patients with cerebrovascular ischemia is the detection of a moderate-to-severe ($\geq$ 50 percent) stenosis in a relevant intracranial arterial segment. Normally, flow velocities obtained from various intracranial arteries follow the normal hierarchy: MCA $\geq$ ACA $\geq$ Siphon $\geq$ PCA $\geq$ BA $\geq$ VA. The absolute velocities can be equal in

between these arterial segments, sometimes even in excess of 5–10 cm/sec. For example, ACA > MCA or BA > ICA may be noted, mostly due to the angle of insonation or common anatomic variations.

In general, a 50-percent focal stenosis doubles the velocity while a 70-percent stenosis triples the velocity across a focal lesion and/or compared to the contralateral side. Various diagnostic criteria have been proposed to diagnose a ≥ 50-percent stenosis in different intracranial arteries (figure 1). However, TCD laboratories are advised to validate their results against contrast angiography (either a CTA or DSA) and develop their own diagnostic criteria. For the routine TCD procedure of individual cerebral artery, see chapter 2. The diagnostic criteria for individual arteries are presented below.

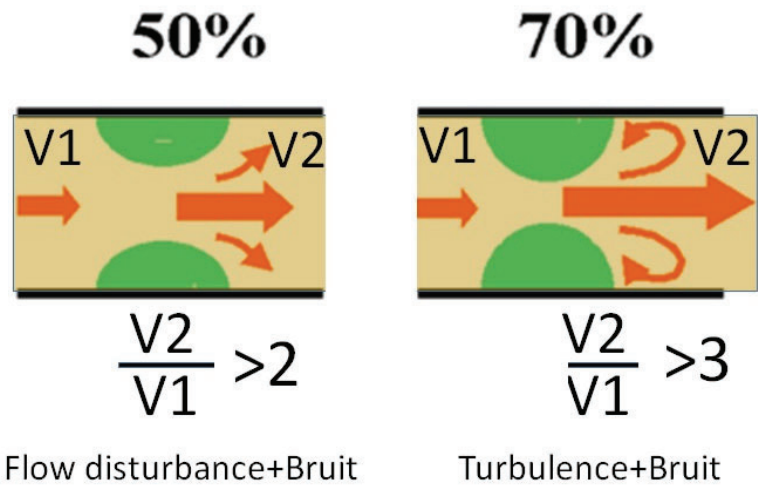


Figure 1. Crude estimation of focal stenosis according to the change in flow velocities across the stenotic lesion.

Middle Cerebral Artery (MCA) Stenosis

Various TCD parameters proposed for the diagnosis of a moderate stenosis (figure 2) of the MCA are:

a. A focal increase of mean flow velocity (MFV) of ≥80-100 cm/sec. New laboratories may start using the MFV of more than 80 cm/sec as the initial diagnostic criterion to screen patients for intracranial

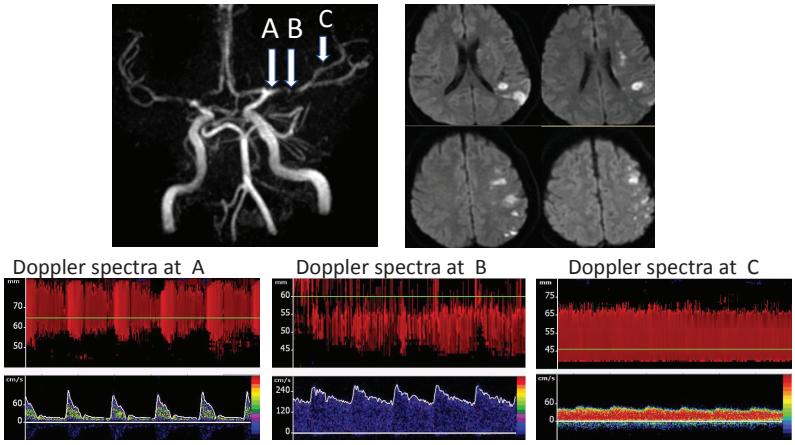
stenosis. After some experience, they should validate their findings against CTA and revise their diagnostic criteria.

b. A focal peak systolic velocity (PSV) increase of ≥140 cm/sec.
c. An inter-hemispheric MFV difference of ≥30 percent.
d. A stenosis-to-pre-stenosis ratio (SPR) of >2.

Absolute flow velocities may be generally higher in patients with hyperdynamic circulatory states like anemia and hyperthyroidism.

Based on the TCD findings, MCA stenosis may also be suspected in the presence of one of the following:

1. Bruit or disturbed flow distal to the stenosis, suggesting turbulence.
2. An increased MFV in the ipsilateral ACA, which represents flow diversion.
3. A harsh bruit represents turbulent flow. Note that the bruit becomes musical when the stenosis becomes severe.
4. Spontaneous microembolic signals (MES) in the distal MCA. One needs to be careful about MES since they may originate from more proximal sources like the heart, aorta or the cervical carotid arteries.
5. Very low flow velocities with slow systolic acceleration, especially in tight, elongated MCA stenosis.



TCD findings in a patient with symptomatic left MCA stenosis. Doppler spectra just before the stenosis at a depth of 64mm (A) show high resistance pattern. Marked focal flow acceleration is noted at the site/exit of the stenosis (B) at a depth of 58mm. Doppler spectra obtained from distal MCA showed blunted flow pattern with low resistance due to compensatory vasodilatation (C).

Figure 2. MCA stenosis

Anterior Cerebral Artery (ACA)

Primary findings in an ACA stenosis include:

1. Focal significant ACA MFV increase (ACA > MCA);
2. ACA MFV of ≥80 cm/sec;
3. A ≥30-percent difference between the proximal and distal ACA segments; and/or
4. A ≥30-percent difference compared to the contralateral ACA.

The collateral flow via the ACOM can be differentiated by a normal contralateral ACA flow direction and the absence of stenotic signals at 75 mm. Sometimes, very low velocities are noted in the A1 ACA due to a suboptimal angle of TCD insonation.

Terminal ICA and ICA Siphon

Supraclinoid ICA segments are difficult to insonate properly. Transorbital insonation may reveal stenotic flow directed toward or away from the probe at 58–65 mm in adults. The sonographer should avoid deeper insonation to avoid the strong ACA signals in some cases.

From the transtemporal window, terminal ICA bifurcation is located at 60–75 mm. A terminal ICA/siphon stenosis (figure 3) is suspected if the following findings are observed:

1. Focal significant MFV increase with ICA > MCA;
2. ICA MFV of ≥70 cm/sec;
3. A ≥30 percent difference between arterial segments; and/or
4. Very low velocities, especially in patients with long segments of severe stenosis.

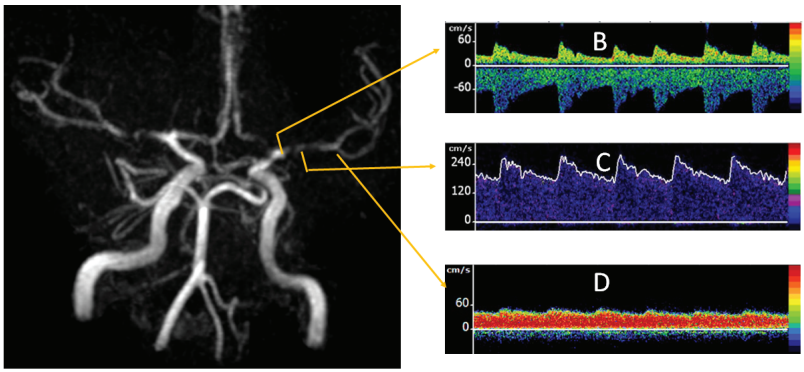


Figure 3. TCD findings in left terminal ICA stenosis (A). Panel B shows MCA flow velocities much lower (with higher resistance) than the ipsilateral ACA (flow diversion). Panel C shows markedly elevated focal flow acceleration at the site of proximal MCA stenosis. Panel D shows blunted flow signals in distal MCA.

Posterior Cerebral Artery (PCA)

A PCA stenosis is suspected if the following conditions are met:

1. Focal significant flow velocity increase: MFV of PCA > ACA or ICA; and/or
2. PCA MFV of $\geq$50 cm/sec.

Basilar Artery (BA)

Primary findings in BA stenosis include:

1. Focal significant velocity increase: MFV of BA > MCA or ACA or ICA;
2. MFV BA $\geq$60 cm/sec in adults;
3. Difference of $\geq$ 30 percent between arterial segments (proximal to the site of stenosis).

For adult skulls, we suggest to use the following depths for BA: proximal BA at 75–90 mm; the mid-BA segment at 90–100 mm; and the distal BA at >100 mm.

Additional findings may include:

1. Signs of turbulence and disturbed signals distal to the stenosis;
2. High resistance flow (PI > 1.2) proximal to the stenosis;

3. Compensatory flow increase in VAs and PICAs, indicating cerebellar collateralization; and
4. Collateral supply via PComA to PCA and reversed distal basilar artery.

Common sources of error include a tortuous basilar ("not found" does not always mean obstructed), elongated BA obstruction, and distal BA lesions that were not reached by the TCD insonation.

Vertebral Artery (VA)

Intracranial VA is usually insonated at 40–75 mm from the sub-occipital window. Findings of an intracranial VA stenosis include the following:

1. Focal significant velocity increase where MFV of VA > BA;
2. MFV VA $\geq$50 cm/sec;
3. A difference of $\geq$30 percent between VAs or its segments; and/or
4. VA stenosis may also cause a high resistance (PI $\geq$ 1.2) flow in its proximal segment, and/or a blunted or minimal flow signal at or distal to the stenosis segment.

One should be careful when comparing the two VAs since most of the individuals have considerable asymmetry. Additional findings may include:

1. Signs of turbulence or disturbed flow signal distal to the stenosis;
2. A compensatory flow increase in the contralateral vertebral artery or its branches (cerebellar collaterals);
3. Low BA flow velocities (hemodynamically significant lesion, hypoplastic contralateral VA) and a low resistance flow distal to the stenosis (compensatory vasodilation); and
4. High resistance flow proximal to the stenosis.

Diffuse Intracranial Disease

A short segment of arterial stenosis produces focal velocity increases on the upslope of the so-called Spencer's curve of cerebral hemodynamics (figure 4). However, the relationship between flow velocity and diameter reduction is also affected by the length of the stenosis or the presence of multiple distal lesions (figure 5). TCD also provides information about impedance to the flow by calculating the pulsatility index (PI), which was originally described by Gosling and King (PI calculated as PSV–EDV/MFV, where PSV is peak systolic velocity, EDV is end-diastolic velocity, and MFV is mean flow velocity).

With the progression of an intracranial stenosis, long and severely narrowed vessels can reduce blood flow velocities on the down-slope of the Spencer's curve (figure 4). PI can differentiate the velocity reduction due to reduced cardiac output (low PI) from increased distal resistance (high PI). We have previously reported many combinations of MFV and PI, which represent various distinct pathological states (figures 6).

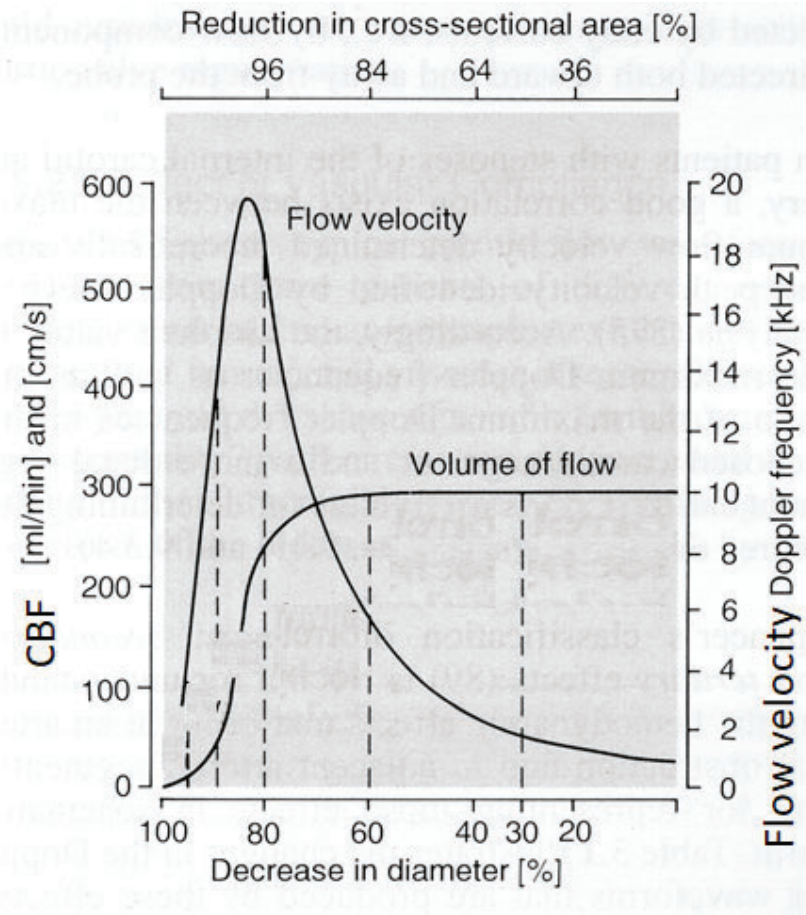


Figure 4. Spence's curve of cerebral hemodynamics.

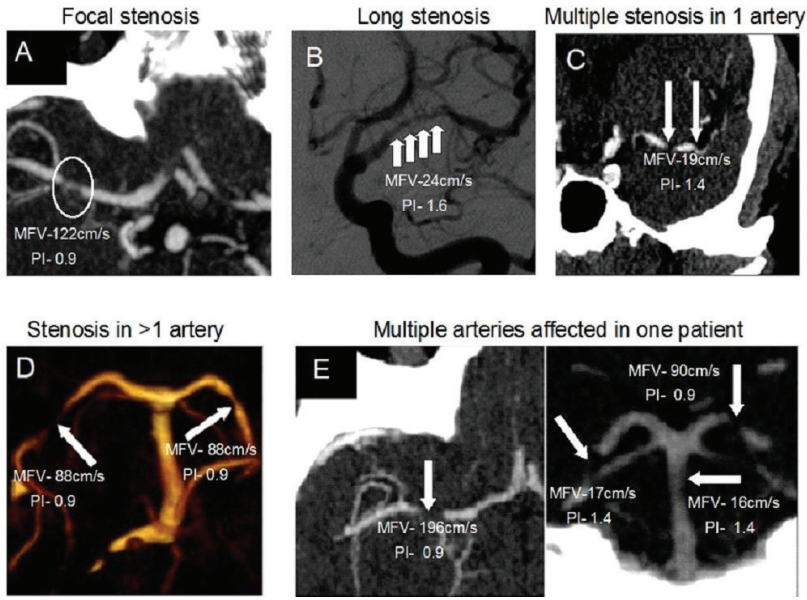


Figure 5. Patterns of stenosis noted on CT angiography.

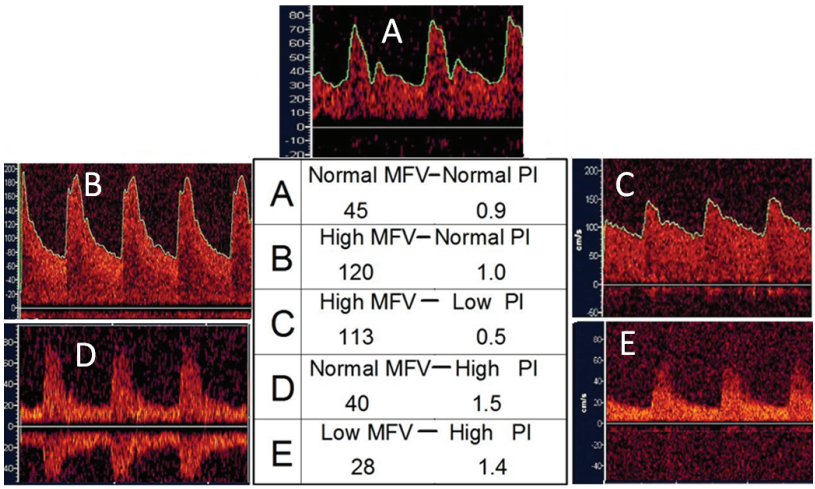


Figure 6. Various patterns of TCD flow spectra.

In conclusion, TCD has an established clinical value in the diagnosis of intracranial stenosis. Well-established and validated criteria exist for the diagnosis of a focal intracranial stenosis. One needs to be careful with very low flow velocities (especially when accompanied by a high PI), which may suggest a severe intracranial disease.

Suggested Readings

1. Sharma VK, Tsivgoulis G, Lao AY, Malkoff MD, Alexandrov AV. Noninvasive detection of diffuse intracranial disease. Stroke. 2007;38: 3175-81.
2. Sharma VK, Wong KS, Alexandrov AV. "Intracranial Stenosis" In Cerebrovascular Ultrasound in Stroke Prevention and Treatment 2nd Edition 2011. Edited by Andrei V Alexandrov. Wiley Blackwell publishers.

FAST-TRACK TCD PROTOCOL: TCD IN ACUTE ISCHEMIC STROKE

REZA BAVARSAD SHAHRIPOUR, THANG NGUYEN AND JOSE C. NAVARRO

Introduction

In this era of emergent recanalization after an acute occlusion of cerebral vessels, an urgent demonstration of a steno-occlusive lesion is mandatory. Vascular imaging of the cerebral vessels may not be ideal in some settings because of urgency. Hence, a reliable method should be available. Although claimed to be operator-dependent and despite the fact that some patients may have poor insonation window, transcranial Doppler (TCD) is still a wise choice in this case.

In the fast-track TCD (FTT), it is necessary to have enough information about the extracranial, anterior, and posterior circulation (both carotid and vertebral arteries) before preforming the diagnostic test.

Some tips before beginning the fast track ultrasound

1. Take a good and reliable history and perform a neurological examination to identify the clinically relevant artery affected.
2. Set the power of the machine to 100 percent.
3. For the single-gated TCD, use a large sample volume (>10 mm). However, for power motion mode (PMD)-TCD, a sample volume of 3–9 mm is often used.
4. Try to go with the flow and visualize vessels that are expected to be found in that depth and direction. (Using the PMD-TCD is very helpful to correct the probe direction in the limited time.)
5. In fast-track TCD evaluations, a complete study may not be necessary. It is important to be organized when performing the test and to find enough data to justify the patient's symptoms, affected territory, and the collateral status.
6. For middle cerebral artery (MCA) interrogation, the probe should

be placed in the mid-temporal window and directed slightly upward and anteriorly. If the MCA signals are not obtained, it would be necessary to differentiate between a poor window and no flow in the acute setting of an MCA steno-occlusive lesion. Insonate the posterior cerebral artery (PCA) by turning the probe posteriorly over the mid-temporal window. A good flow in the PCA is indicative of a possible occlusion in the MCA.

7. In patients with resolved symptoms (pure motor, pure sensory, non-localizable symptoms), try the anterior circulation to save time, and then expand the examination to the posterior circulation.
8. During the study and as long as you are following vessels in different territories, try to save any abnormal signal, segmental change in the MFV, or an embolic signal in different territories.
9. After finding the affected segment, save the data and try to track the proximal and distal segment. You need to approximate the depth of the occlusion. If the patient deteriorates after an initial improvement, you can double check that depth and rule out either a thrombosis relocation or thrombosis expansion.

Procedure

Referring to the clinical diagnosis of the patient, the treating clinician should complete and interpret the fast-track TCD in real-time.

1. Determine the presumed arterial territory affected by ischemia according to the clinical information. For example, if the neurological deficits are compatible with MCA territory, start the insonation of the MCA on the non-affected side to confirm the presence of a temporal window and to assess the normal MCA wave form and velocity range.
2. If there is enough time, evaluation of the circle of Willis on the non-affected side should also provide the depth and velocity range for the Ml and M2 MCA segments and the internal carotid artery (ICA) bifurcation. This step of the study, however, is not necessary if time is limited.
3. Evaluate the MCA on the affected side by insonating the mid-MCA at depth range (56–58 mm). It is important to compare the waveform, systolic flow acceleration, and pulsatility index (PI) to the non-affected side. If the mid MCA has a normal wave and parameters, insonate the distal segment of MCA (M2) in the range of 40–50 mm, followed by the proximal MCA (60–65mm) and an ICA bifurcation assessment (range of 60–70 mm). If the MCA

shows a "blunted" waveform or if no signal is detected at the mid MCA (56–58 mm), to save time, there is no need to proceed to the proximal MCA.

4. If the temporal window on the affected side is not sufficient or the signal is not optimal despite an adequate window on the normal side, the opposite MCA and anterior cerebral artery (ACA) can be interrogated, utilizing this window, at the depth range of 80-100 mm. Have in mind that the MCA, at this point, is moving away from the probe, while the ACA is moving toward it. Hence, the Doppler spectra will be reversed.
5. To evaluate the ophthalmic artery (OA) via the transorbital window on the affected side, focus on the artery's direction and its PI. This is done by insonating at the depth of 52-58 mm with the lowest power (10 percent or lower). Track the carotid siphon through this window as well, at the depth of 60-64 mm. Evaluation of the OA is very useful in patients with possible proximal carotid occlusion or near occlusion when we cannot find any signal in the affected MCA side. You may see the reverse flow in the OA on the affected side with a strong diastolic component (low PI), which could be a significant finding in proximal carotid occlusions. The OA signal (direction and PI) can be followed up during the subsequent TCD examinations to evaluate reperfusion or re-canalization of the proximal carotid artery.
6. The insonation of the basilar artery (BA) is helpful to show the evidence of compensatory flow velocity through the posterior circulation or detecting any asymptomatic stenosis.

While performing the fast-track TCD, insonation of the vertebral arteries (VAs), OA and ICA on the non-affected side, and PCAs is not mandatory and can be performed at a later time

For patients with a clinical picture that is compatible with a posterior circulation ischemia, the following steps may be carried out:

1. First step is to find the VA junction (at about 75 mm) through the sub-occipital window. This depth is helpful to evaluate both VAs and proximal BA at the same time. Focus on the PI, waveform, direction of the flow, and diastolic component. After detecting the strong signal, you need to be sure that you are in the best direction. Utilizing TCD with the power-motion Doppler (PMD), the wide blue band at the depth of 45-70 mm is the vertebral artery at

different depths, indicating that the probe is in the best direction.

2. The next step is to increase the depth and follow the BA at the depth of 80-100 mm. While tracking the BA, one should note the importance of looking for any significant change in the flow velocities, PI, or bruits.
3. If an abnormal signal is found at the depth between 75 and 100 mm, locate the terminal VA on the unaffected side (based on your clinical judgment) at the depth of 40-80 mm and compare that to the VA on the affected side (at the same depth and direction). Take note that the VA may not be seen terminating at the BA. In this case, it may have terminated in the posterior inferior cerebellar artery (PICA). Additionally, the vertebral dominancy, which may show prominence in some patients, should also be considered (with increased flow velocities, normal PI and different waveform compared to the hypoplastic side).
4. In case of any stenosis or near occlusion of the VA (especially on the dominant side), following the BA proximal to distal end to rule out reversible flow in the BA. **Sometimes, a sluggish antegrade flow in the BA, which is distal to the stenosis, and a high resistance flow (increased PI) in the proximal VA (affected side) confirm the suspected stenosis in the vertebral segment.**
5. The next step is to evaluate the PCA on the affected side and to identify the posterior communicating artery (PCOM). To find the PCA, insonate at the temporal window in a posterior direction at the depth of 55-75mm. The PCOM should be evaluated on both sides, and it may indicate anterior circulation compensatory activity. The direction of flow in the PCOM would be from the anterior to the posterior (in this case).
6. If you have enough time, you can evaluate both MCAs and anterior cerebral arteries (ACAs) at the depth of 60-75mm. They may show compensatory increase in the flow velocities, which is another confirmatory sign of vertebro-basilar obstruction.

Fast-track TCD findings can help to detect occlusion, intracranial clot dissolution, distal embolization, re-occlusion, and stenosis. In addition, the location of arterial occlusion on the TCD (specific segments such as M2 or Ml MCA, terminal ICA, and proximal versus distal BA) can help you explain the distribution of the neurologic deficit, since TCD also shows major collateral vessels that compensate for the occlusion.

The most important role of TCD in acute stroke treatment is the determination of the presence and location of arterial occlusion and

possible residual flow signals around the clot. One of the most worthwhile roles of TCD in acute phase is the recanalization after intravenous thrombolysis with tissue plasminogen activator (IV-tPA) through the TIBI flow grades. The TIBI (or thrombolysis in brain ischemia) classification grades blood-flow into six groups. Grade 0 is absent, grade 1 minimal, grade 2 blunted, grade 3 dampened, grade 4 stenotic, and grade 5 as a normal waveform. The TIBI flow grade can be measured in the region at or just distal to the presumed depth of arterial occlusion.

SONOTHROMBOLYSIS

GIRIANTO TJANDRAWIDJA,
CYRUS ESCABILLAS, JOSE C. NAVARRO
AND VIJAY K. SHARMA

Sonothrombolysis is described as the clot lysis assisted by continuous ultrasound exposure during the intravenous thrombolysis for acute ischemic stroke. Sonothrombolysis has shown promise for becoming a rapidly available, non-invasive, and portable tool in the armamentarium of the stroke neurologists.

The ultrasound is a mechanical and pressure wave. In the blood clots that are continuously exposed to the ultrasound waves, transient and repeated thinning of the fibrin threads is observed. This action potentially separates the strands of fibrin and creates microstreaming of the blood flow through the clot, thereby enhancing better and faster clot lysis. Other plausible mechanisms for sonothrombolysis are microcavity formation in the shallow layers of thrombus, superficial vasodilation, and promotion of nitric oxide release. These mechanisms increase the penetration of tissue plasminogen activator (tPA) into the clot, leading to clot lysis.

In-vitro and animal models demonstrated a significant effect of ultrasound waves in the range of MHz–KHz frequency on the therapeutic efficacy of the tPA. Previous trials with low frequency ultrasound demonstrated unacceptable risks of intracranial hemorrhage. However, the use of the routine diagnostic 2-MHz ultrasound showed better recanalization rates when the acutely occluded intracranial artery was insonated during intravenous thrombolysis. The thrombolytic effect of sonothrombolysis may be enhanced further with the use of ultrasound contrast agents. However, more clinical trials are needed to establish the dose and efficacy of these ultrasound contrast agents.

We follow a systematic step-by-step fast-track neurovascular ultrasound evaluation in an emergency acute care setting:

1. To avoid a major delay between the administration of intravenous thrombolysis and continuous TCD monitoring, set the machine as follows: use a 2-MHz probe, power at 100 percent, and gate setting of 6-10mm protocol to immediately locate the acoustic window.
2. Secure the ultrasound monitoring probe with the headframe and fix it properly to avoid movement-artifacts during monitoring. Identify the affected segment based on the Thrombolysis in Brain Ischemia (TIBI) flow grade.
3. If the patient has undergone a CT angiography, which shows an arterial occlusion, the reader can measure the distance between the site of occlusion and the skull bone. This depth may help the sonographer for a faster setup of the TCD machine.
4. If there is no CT angiogram, then a fast-track TCD is performed to locate the site of arterial occlusion with worst TIBI flow. When time permits, the sonographer should do a fast exploration of other intracranial arteries also.
5. Set the depth of the TCD machine to the occluded vessel where the worst TIBI grade is detected. There should be a firm contact between the skin and monitoring probe to have an optimized transmission of the ultrasound waves to the site of vessel occlusion.
6. TCD monitoring is performed for two hours and final TIBI signals are noted. One main reason for keeping TCD monitoring for two hours is to detect and prevent arterial re-occlusion during the first hour after completing the tPA infusion. Furthermore, the half-life of tPA (about 6-8 minutes) ensures the presence of tPA in the cerebral circulation even after the tPA infusion has been completed. TCD signals may be recorded intermittently or as a continuous movie file. However, it is advised to save fixed traces at every thirty minutes (or when the spectra show a significant change) to identify any worsening or improvement of TIBI flow grades.

A follow-up TCD examination can be performed on day two to evaluate the status of arterial patency.

The usual sonothrombolysis protocol employed at our centers is described below:

Step 1

A. For anterior circulation

If time is short

1. Use the clinical information to determine the side of the intracranial occlusion.
2. Start the insonation of the MCA at the depth of 55 mm. If no signal is received, go to the proximal segment at 62 mm. Once the arterial flow signal is obtained, start reducing the depth until the worst TIBI signal is found.
3. Search for a possible flow diversion to the ACA, PCA, and M2 MCA (if another M2 MCA branch is occluded, evaluate it).
4. Check also the flow spectra of the ophthalmic artery and ICA siphon on the affected side.

If time permits

1. Examine the whole MCA (from the distal to proximal end at 40–65 mm) of the unaffected side.
2. Look for the shapes of the TCD waveforms and flow velocities and compare them to the affected side.

B. For posterior circulation

If time is short

1. Access the BA from the transforaminal window, starting at the depth of 75-80 mm, and continue until the distal segment at 100-110 mm.
2. If there is any abnormal signal at a certain depth of the BA, examine the VA on the affected side, and then compare the flow velocity and signal of the terminal VA with the terminal VA of the unaffected side.
3. Examine the right and left PCA from the respective transtemporal windows.

If time permits, examine the flow velocity of the anterior circulation, right and left, for possible compensatory accelerations.

Step 2

1. To ensure constant and steady insonation, place the head frame to secure the probe aiming to the affected blood vessel.
2. If the affected vessel is in the posterior circulation, a handheld monitoring via the transforaminal window should be done.
3. Monitor for two hours.
4. Watch for signs of recanalization with the TIBI grading system as the guide.

Limitations of Sonothrombolysis

Sonothrombolysis influences the thrombolytic activity of the tPA. However, the optimal frequency of therapeutic TCD remains debatable. The temporal bone attenuates almost 86 percent of the currently used 2-MHz ultrasound energy. An insufficient temporal acoustic window leads to an inability to monitor the intracranial arteries in a fair proportion of patients, especially among Asians in whom temporal bone window may not be optimal in 8-29 percent of the cases. Hyperostosis is a possible reason for this observation, especially in elderly females, observed in 6-12 percent of adult women of all ages and in >50 percent of those above sixty years of age (compared with only 1 percent in men).

TIBI grading for the assessment of residual flow in an intracranial artery. The grading system is shown in figure 1.

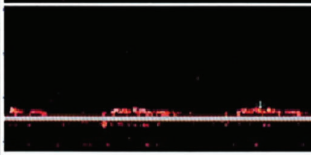
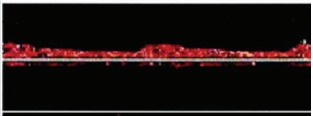
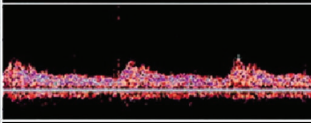
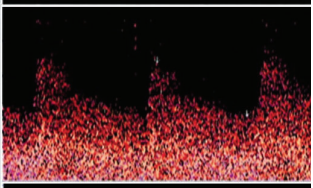
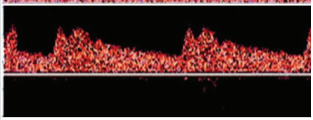
Grade 0: **Absent**	- absent flow signals are defined by the lack of regular pulsatile flow signals despite varying degrees of background noise.	
Grade 1: **Minimal**	- systolic spikes of variable velocity and duration - absent diastolic flow during all cardiac cycles based on a visual interpretation of periods of no flow during end diastoli. Reverberating flow is a type of minimal flow	
Grade 2: **Blunted**	- flattened systolic flow acceleration of variable duration compared to control. - positive end diastolic velocity and pulsatility index < 1.2.	
Grade 3: **Dampened**	- normal systolic flow acceleration - positive end diastolic velocity - decreased mean flow velocities (MFV) by >30% compared to control	
Grade 4: **Stenotic**	- MFV of >80 cm/s AND velocity difference of >30% compared to the control side or - if both affected and comparison sides have MFV <80 cm/s due to low end-diastolic velocities, MFV >30% compared to the control side AND signs of turbulence.	
Grade 5: **Normal**	- <30% mean velocity difference compared to control - similar waveform shapes compared to control	

Figure 1.

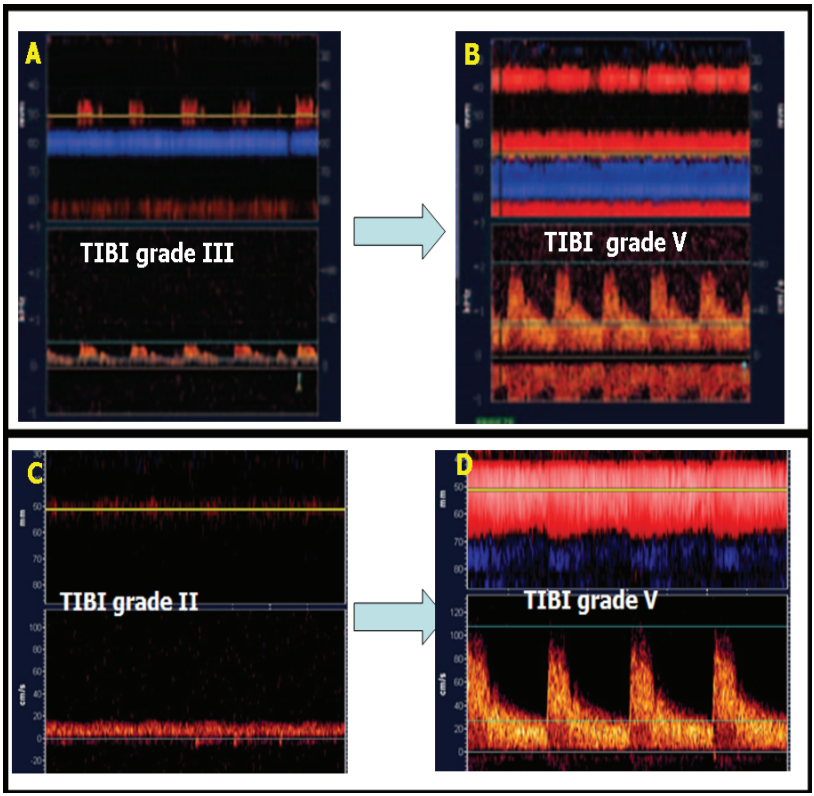


Figure 2. Real-time monitoring of complete recanalization in two patients with proximal MCA occlusion treated with sonothrombolysis.

Patient I. Baseline TIBI: Grade III (A); TIBI at the end of the two-hour TCD monitoring: Grade V (B).

Patient II. Baseline TIBI: Grade II (C); TIBI at the end of the two-hour TCD monitoring: Grade V (D).

Transcranial Doppler in Vasospasm Monitoring after a Subarachnoid Hemorrhage

K J Lara, Prakash Paliwal

Symptomatic cerebral vasospasm (VSP) is a serious but treatable complication of a subarachnoid hemorrhage (SAH). About 25 percent of the patients who suffer from an SAH may develop delayed ischemic deficits due to VSP. It develops between four and seventeen days after an acute episode, but the maximum risk is between the fourth and ninth day after the initial bleed, and the morbidity and mortality are considered to increase significantly to almost 20 percent.

Transcranial Doppler (TCD) ultrasonography is a noninvasive, repeatable, and inexpensive bedside tool for diagnosing and monitoring VSP. It identifies cerebral hemodynamic changes in diagnosing VSP before the appearance of clinical neurologic deficits and can suggest earlier interventions. Contrast angiography is considered the gold standard in VSP detection following an SAH. However, it is an invasive technique and not useful for dynamic monitoring.

TCD monitoring for VSP is an established indication. TCD has the capacity to diagnose vasospasm of the middle cerebral artery (MCA) and basilar artery (BA) with good sensitivity and specificity. A systematic review of twenty-six studies comparing the TCD with angiography shows that MCA with a mean flow velocity (MFV) of >120 cm/sec on TCD, carries 99% specificity and 67% sensitivity to identify angiographic VSP of ≥50%. Moreover, MCA with an MFV > 200 cm/sec carries 98% specificity and 27% sensitivity. A BA MFV/VA MFV ratio over 3.0 with BA velocities higher than 85 cm/sec was 92% sensitive and 97% specific for BA narrowing of more than 50%, and this specificity can be brought up to 100% with MVF >95 cm/sec.

TCD Techniques in Diagnosing Vasospasm

1. Using the low frequency 2-MHz probe, perform on day one a baseline TCD that involves all intracranial vessels, as well as the extracranial ICA (eICA). Determination of the Lindegaard index (LI), which is calculated as the ratio of MFV MCA/MFV of the ipsilateral eICA, will allow monitoring for VSP.
2. The transtemporal windows are utilized for determining the MCA MFV. The probe is directed slightly anterior-superior manner, with the flow velocity measured by adjusting the depth between 40 and 65 mm. The MCA Doppler spectra are characterized by a low resistance (vessel) blood flow, with the flow signals toward the direction of the probe.
3. For the measurement of eICA MFV, insonate at the submandibular acoustic window. The probe is directed in a superior-medial manner, with the depth placed at 45-50 mm. The eICA is identified as a low-resistance vessel with the waves pointing away from the probe.
4. Daily measurements of LI are recorded until day fourteen, and every other day thereafter if needed.

Interpretation of LI results

MCA VSP (%)	**MFV (cm/sec)**	**LI**
Mild (< 25)	120–149	3–6
Moderate (25–50)	150–199	3–6
Severe (> 50)	> 200	> 6

In addition, hyperemia can also be associated with the SAH, and this should be differentiated from a true VSP. A simplified interpretation of the TCD for an evaluation of VSP versus hyperemia are as follows:

MFV in MCA	Lindegaard Ratio	Interpretation
< 120	< 3	Normal
> 120	< 3	Hyperemia
120-180	3-6	Spasm + hyperemia
> 180	> 6	Spasm (severe)

5. For patients with poor temporal windows, measurement of the basilar artery, in lieu of MCA, can be used for determining LI. This is known as the *modified LI*, and this is computed by determining

the ratio of BA MFV/left or right extracranial vertebral artery (eVA) MFV.

The BA is insonated at the suboccipital window, with the probe angled superiorly toward the root of the nose. The flow velocities are recorded by adjusting the depths from 80 to 120 mm. The BA is identified as a low-resistance vessel, with the waves pointing away from the probe.

Interpretation of results using the modified LI:

BA VSP (%)	**MFV (cm/s)**	**Modified LI**
Mild (< 25)	70–85	2 to 2.49
Moderate (25–50)	> 85	2.5 to 2.99
Severe (> 50)	> 85	> 3

Therefore, an increase in the MFV on TCD monitoring is highly predictive of VSP of the intracranial vessels following SAH. Day-to-day changes in MFV of about 50 cm/sec, or a daily increase in MFV > 65 cm/sec from days three to seven, indicate a high risk for delayed ischemic changes due to VSP. However, if this increase in MFV is accompanied by an LI < 3, hyperemia may likely be the cause of elevated flow velocities.

ASSESSING INTRACRANIAL PRESSURE WITH TRANSCRANIAL DOPPLER ULTRASONOGRAPHY

BENJAMIN R WAKERLEY

Introduction

Identification of raised intracranial pressure (ICP) remains important in the management of several neurological conditions (e.g., traumatic brain injury and meningitis), which, if left untreated, may lead to a permanent neurological deficit or even death.

Standard methods to measure the ICP (e.g., intraventricular drain, pressure bolt, and lumbar puncture) are invasive and in some circumstances (e.g., coagulopathy) are contraindicated. The development of non-invasive techniques, which are reliable and do not require technical expertise, is therefore important.

Although TCD is primarily used to assess blood flow in major intracranial vessels, there is mounting evidence to suggest that it can also be used to indirectly measure ICP.

ICP is determined by four physiological factors:

1. arterial volume and flow,
2. venous outflow,
3. cerebrospinal volume, and
4. brain volume.

If we assume that there is a close relationship among these four components, then it is conceivable that an analysis of arterial flow dynamics can be used to assess the ICP. Major intracranial vessels (e.g., middle cerebral artery) are subjected to external pressure by surrounding brain tissue and internal pressure by arterial blood flow. Shifts in pressure across the compliant vessel walls determine the flow velocity waveform (Figure 1a). Characteristically, peaked waveforms are associated with a raised ICP.

Procedure

Provided that there is a sufficient temporal window, a standard 2-MHz diagnostic TCD can be used to evaluate Doppler spectra from either of the middle cerebral arteries.

a. Certain precautions should be taken during a TCD evaluation to ensure better reliability. The patient should be supine and comfortable.
b. As intracranial hemodynamics changes significantly with changes in the arterial PaCO2, it is important that the patient breathes steadily and avoids holding his breath or hyperventilating.
c. The operator should aim to visualize a clearly defined waveform, which does not fluctuate significantly over time. Often, the inbuilt computer software will calculate the pulsatility index (PI) in real time, but sometimes, especially if the waveform is poorly defined, it is better to acquire the raw video data and calculate the PI.
d. By placing the probe into a headframe, the operator can record continuous TCD spectra. This allows continuous monitoring.

Analysis of Waveforms

A more quantitative assessment can be made by measuring the TCD-derived PI, which in several studies has been shown to positively correlate with raised ICP. The PI is a measure of the variability of blood velocity and can be calculated by measuring TCD-derived blood flow velocities in a given vessel (figure 1b). In our experience, a PI >1.26 can reliably predict intracranial pressure ≥20 cm H2O (15.4 mm Hg).

Rather than accurately predicting an ICP value with the PI, the operator should be aware that changes in the flow velocity waveform or PI, in an otherwise hemodynamically stable patient, may represent contemporaneous changes in the ICP.

Limitations

A number of physiological factors are known to affect cerebral blood flow and, therefore, the PI. These include changes in the PaCO2, arterial blood pressure, cerebral atherosclerosis, and cardiac regurgitant lesions. Fluctuations in the PaCO2 are particularly important as it has a reciprocal relationship with the PI. Hyperventilation is therefore associated with an elevated PI, whereas breath-holding depresses it.

Example

In one example, we used the TCD to monitor the intracranial pressure of a patient with a cerebral venous sinus thrombosis. At onset, while the patient was complaining of "high pressure" headaches and noted to have papilloedema, TCD-derived PI was elevated (day five PI = 1.93). With the relief of his headache and papilledema over four weeks, the PI value decreased (day thirty-one PI = 0.8).

Further Reading

Wakerley B, Yohana K, Luen Teoh H, Tan CW, Chan BP, Sharma VK. Non-invasive intracranial pressure monitoring with transcranial Doppler in a patient with progressive cerebral venous sinus thrombosis. *J Neuroimaging* 24 (2014): 302–04.

Wakerley BR, Kusuma Y, Yeo LL, Liang S, Kumar K, Sharma AK, Sharma VK. Usefulness of transcranial Doppler-derived cerebral hemodynamic parameters in the noninvasive assessment of intracranial pressure. *J Neuroimaging* 25 (2015): 111–06.

Cardim D, Robba C, Bohdanowicz M, Donnelly J, Cabella B, Liu X, Cabeleira M, Smielewski P, Schmidt B, Czosnyka M. "Non-invasive Monitoring of Intracranial Pressure Using Transcranial Doppler Ultrasonography: Is It Possible?" *Neurocrit Care* 25 (2016): 473–91.

Rasulo FA, Bertuetti R, Robba C, Lusenti F, Cantoni A, Bernini M, Girardini A, Calza S, Piva S, Fagoni N, Latronico N. "The accuracy of transcranial Doppler in excluding intracranial hypertension following acute brain injury: a multicenter prospective pilot study." *Crit Care* 21 (2017): 44.

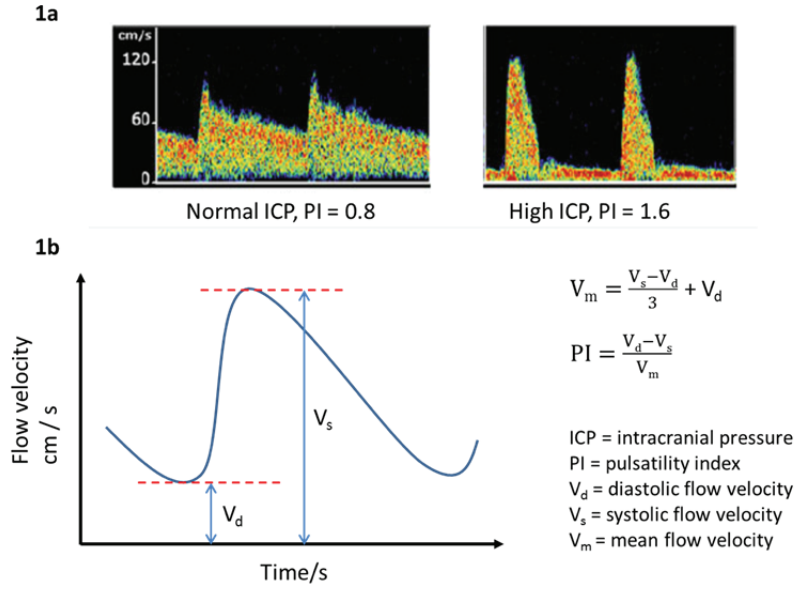


Figure 1. The transcranial Doppler (TCD) was used to acquire hemodynamic flow spectra from the right middle cerebral artery. A peaked waveform (right) indicates the presence of raised intracranial pressure (1a). By measuring flow velocities during systole and diastole, the operator can calculate pulsatilty index (PI) (1b). Peaked waveforms are associated with an elevated PI, and this correlates with raised intracranial pressure.

PERIOPERATIVE TCD MONITORING

YOHANNA KUSUMA, JOSE C. NAVARRO
AND VIJAY K SHARMA

An important, extended application of the transcranial Doppler (TCD) is its use in the operation theater. It is well known that TCD monitoring during a carotid and aortic surgery improves the clinical outcomes.

During a carotid and aortic surgery, the patient carries a higher risk of perioperative ischemic stroke because of embolization or hypoperfusion, or a combination of the two.

Embolization. When an atherosclerotic plaque in the carotid artery or arch of aorta is manipulated or crushed, small particles may embolize into the cerebral circulation and cause ischemic strokes.

The TCD is the only diagnostic modality that can detect and quantify microembolic signals (MES) in real-time. The sonographer can guide the surgeon in taking necessary actions to reduce the MES load.

Hypoperfusion. During a coronary artery bypass graft (CABG) and a major aortic surgery, blood pressure often drops, reducing cerebral perfusion. In carotid endarterectomy (CEA), the artery is clamped both distal and proximal to the stenosis. During this period, perfusion in the ipsilateral middle cerebral artery (MCA) may drop to critical levels. If perioperative TCD monitoring is performed, the sonographer may warn the surgeon in real-time to take corrective actions.

It is believed that, most of the time, the combination of hypoperfusion and embolization is responsible for the ischemic insult during the surgery. The state of slow flow facilitates the survival as well as prolonged stay of MES in cerebral circulation.

TCD monitoring during CEA

This is an important role played by the neurosonologist in the assessment of the upper limit of the carotid plaque. It is important because a plaque extended high in the carotid artery may be erroneously crushed during clamping.

Before the surgery is started, the sonographer fixes the head frame, and the TCD transducers are fixed (preferably bilateral) and secured when stable signals are obtained from the proximal-to-mid MCAs.

If the TCD shows numerous embolic signals before a dissection of the carotid artery, the sonographer should consider the following causes:

1. Spontaneous MES from the active carotid plaque.
2. Spontaneous MES from artificial heart valves, heart chambers, or aortic arch atheroma. This possibility may be resolved if MES are noted on the contralateral MCA also.
3. MES due to surgical maneuvers, like vigorous skin preparation, skin dissection, and mechanical compression of very soft plaques. In this case, nurses and surgeons may consider being gentler in skin and wound preparation and a modification of the surgical technique.

TCD signals during cross-clamping

The appearance of numerous MES during cross-clamping indicates a crushed carotid plaque. The surgeon should be notified. The clamp may be released and put at a higher level in the carotid artery.

The flow velocities in the ipsilateral MCA may drop immediately after cross-clamping. A decrease of TCD velocity to below 30 percent of the pre-clamp value may be dangerous if it persists for over two minutes. The velocity drop may indicate:

1. Lack of collateral blood supply.
2. Significant decrease in cerebral blood flow due to reduced blood pressure (BP). The sonographer may check if the transducer has not moved from its place.

Patients with these findings may be considered for selective shunting.

TCD signals after shunt placement

A few MES may be noted immediately after the shunt placement. However, the shunt should immediately improve the flow in the ipsilateral MCA. The sonographer should know the following:

1. Harmless air MES may occur during a shunt placement.
2. Particulate MES may come from plaque fragments or microthrombi that may form at the shunt edges.
3. Spontaneous embolization from a proximal source.

4. Shunt carries a large amount of blood flow and may form froths, which may appear as MES, at the distal end.
5. In some cases, the blood flow in the ipsilateral MCA may drop after an initial rise. The sonographer should alert the surgeon to a possible kinking of shunt as well as shunt thrombosis.

TCD signals after shunt and cross-clamp removal

Flow velocities in the ipsilateral MCA often increase more immediately than the pre-clamp and on-shunt values. However, these velocities gradually decrease during the next few minutes by around 50 percent and finally stabilize at values within a 30 percent difference from the contralateral MCA.

TCD waveforms improve, and the pre-operative blunted flows are no longer seen. However, a few MES may be noted within the first two minutes of cross-clamp release.

Abnormal flow after clamp release

A TCD may show a transient increase in the flow velocities upon clamp release (hyperemic response). However, if the velocity increases by more than 1.5 times of the pre-clamp values, it may indicate hyperperfusion. If elevated flow velocities persist, the differential diagnosis includes:

1. A benign finding, if the overall mean flow velocity does not exceed 80 cm/sec or 130 percent of the contralateral MCA.
2. BP increases due to the disturbed autoregulation.
3. Cerebral hyperperfusion syndrome may occur in 19 percent of patients undergoing CEA. This is mostly benign and self-limiting. However, it may lead to a significant cerebral edema or even a devastating parenchymal haemorrhage.

Potential measures that can prevent hyperperfusion early after a cross-clamp release include:

1. hyperventilation or increased oxygenation;
2. blood pressure reduction; and
3. partial cross-clamp and slow release.

The TCD may show a few MES early after cross-clamp release. However, if MES persist longer than they should, one may suspect early thrombus formation at the site of CEA. If this is accompanied with a

reduction in the flow velocities, it may be advisable to assess the carotid patency, and revision of CEA may also be needed.

A case example is shown below:

A seventy-one-year-old Chinese male presented with a non-disabling left MCA subcortical stroke three days ago. His risk factors included hypertension, dyslipidaemia, and smoking.

A CT angiography showed a focal and severe stenosis of proximal left ICA (A). Pre-operative TCD (B) showed blunted flow signals in the left MCA (see figure 1).

He underwent CEA on day five.

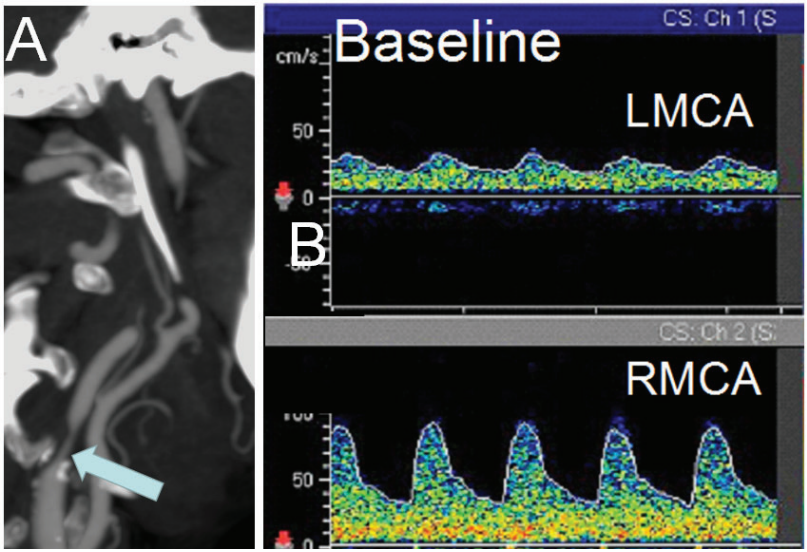


Figure 1. Preoperative CT angiogram and TCD findings.

During the shunt placement, a single MES was noted in the left MCA (see figure 2).

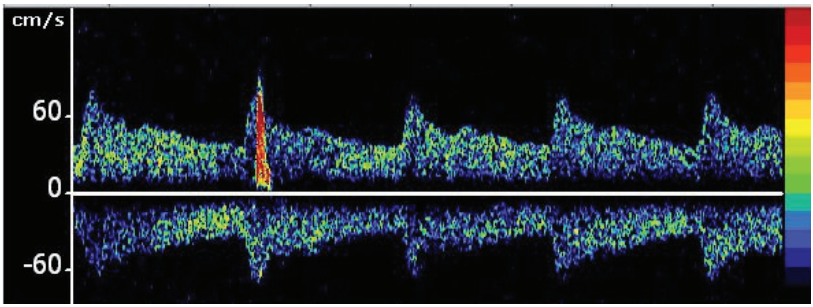


Figure 2. A single loud MES was noted at the time of shunt placement.

However, upon extubation, the patient was restless, with less movements on the right side. BP was 160/100 mmHg. TCD showed markedly elevated flow velocities in the left MCA (see figure 3).

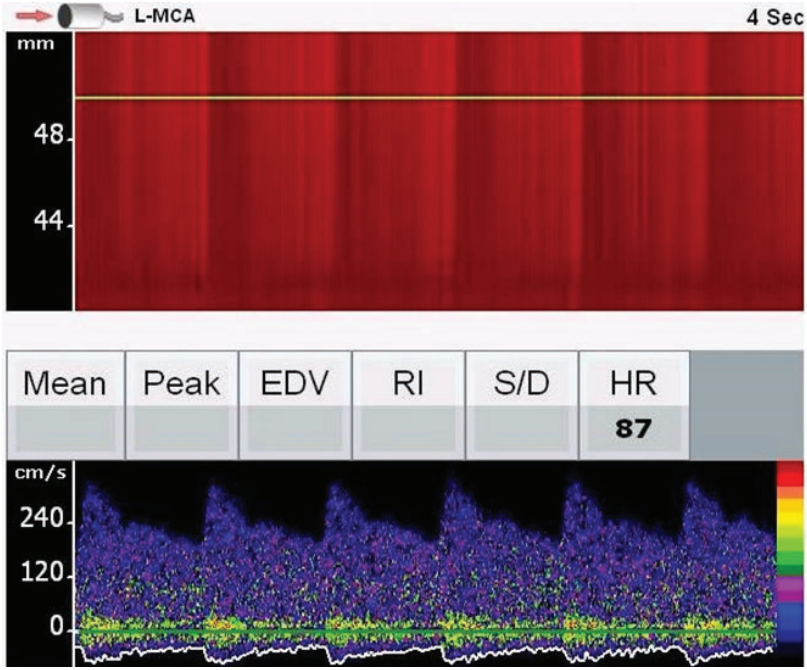


Figure 3. TCD shows markedly elevated flow velocities with vasodilated flow, suggestive of cerebral hyperperfusion syndrome.

CT perfusion confirmed left MCA hyperprfusion (see figure 4).

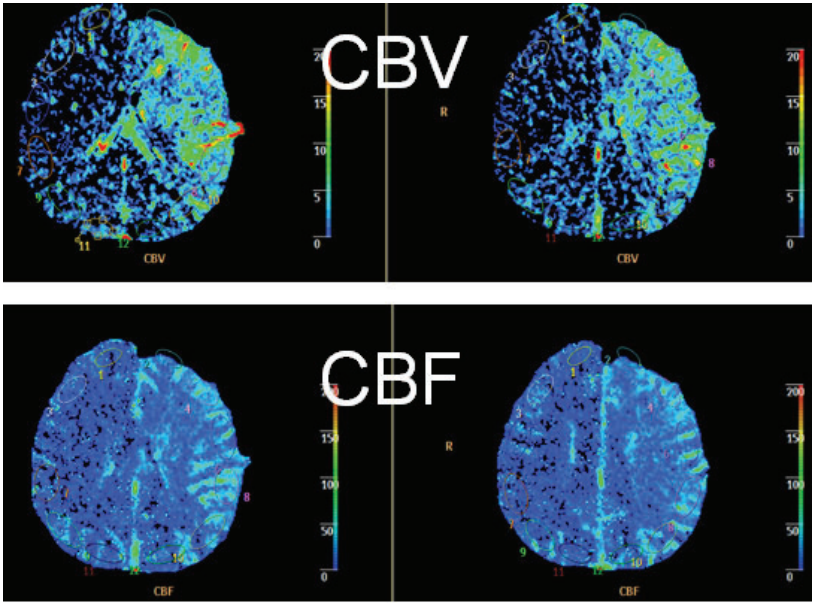


Figure 4. CT perfusion shows increased cerebral blood volume (CBV) as well as cerebral blood flow (CBF) in the left MCA territory.

His blood pressure was pharmacologically reduced and maintained at 110–120 mmHg (systolic). He showed rapid clinical improvement in the next three days and achieved functional independence within two weeks.

In conclusion, the TCD is an important tool in the armamentarium of stroke neurologists. TCD monitoring during CABG and carotid and aortic surgeries plays an important role in making the surgery safer by providing real-time information about cerebral embolization as well as hypoperfusion. All neurovascular laboratories should make efforts to provide this supportive care to vascular and cardiothoracic surgeon, if sufficient manpower is available.

CEREBRAL AUTOREGULATION

SURYANARAYANA SHARMA,
JOSE C NAVARRO AND VIJAY K SHARMA

Introduction

Cerebral vasculature has an actively intrinsic ability to maintain constant cerebral blood flow (CBF) to the tissues within a certain range of systemic blood pressure. This ability is called cerebral autoregulation and, together with the structure of the circle of Willis (CoW), guarantees maintaining CBF even in a partial occlusion of supplying arteries. However, there are some situations in which combinations of these two mechanisms are unable to compensate and may even permit "stealing" of the blood from an affected artery during a hypercapnic challenge. This condition is called the reversed Robin Hood syndrome (RRHS). Andrei V. Alexandrov et al. described this phenomenon of paradoxical intracranial stealing due to poor cerebral blood reserve as an analogy: "Robbing the poor to feed the rich."

RRHS is believed to be responsible for various clinical phenomena. For example, in patients with a moderately large stroke and persistent arterial occlusion, drowsiness may cause hypoventilation and hypercapnia, leading to vasodilatation of the unaffected artery, which in turn steals the blood from an affected artery. Similar mechanisms are implicated in the pathogenesis of early morning headaches: lethargy and change in the diurnal rhythm in some patients with flow-limiting stenoses of large cervicocranial arteries.

Although various structures in the circulation play a role in cerebral autoregulation, a major contribution comes from arteriolar sphincters, which are under direct control of the autonomic nervous system. In general, the parasympathetic nervous system (PNS) causes vasodilatation and increases blood flow, while activation of the sympathetic nervous system (SNS) results in vasoconstriction and reduction of blood flow. Carbon dioxide is the strongest stimulus of the autonomic nervous system. Accordingly, hypercapnia stimulates PNS, and hypocapnia stimulates the SNS. Although the relationship is not linear, each change by 1 mmHg in

carbon dioxide concentration results in about 10 percent of change in blood flow.

Two terms that have been loosely used to describe cerebral autoregulation are vasomotor reactivity (VMR) and cerebral vasodilatory reserve (CVR). It should be clearly understood that VMR estimates the effect of both PNS and SNS on the blood flow, while CVR tests only the PNS. Various diagnostic techniques have been employed for the estimation of VMR, such as computed tomographic perfusion (CTP) with acetazolamide challenge, single-photon emission CT (SPECT) with acetazolamide challenge, Xenon-CT, oxygen extraction fraction by photon emission tomography (PET), and transcranial Doppler (TCD). Of these, the TCD is a simple, non-invasive, and bedside imaging tool with a good reproducibility to assess cerebrovascular hemodynamics and VMR. The techniques of examination, advantages, pitfalls, and an illustrative case are discussed in this review.

Vasomotor Reactivity (VMR)

VMR assesses the response of the cerebral vasculature to various vasomotor stimuli for maintaining a near-constant CBF. Vasomotor changes in response to various stimuli can be studied in real time by the TCD. Carbon dioxide (CO_2) is the strongest stimulator to both the PNS and SNS. Increased levels of carbon dioxide cause vasodilatation in normal individuals, resulting in increased flow velocities on the TCD, while an opposite response is noted in response to hypocapnia.

Indications

1. Patients with intracranial stenosis with acute stroke/transient ischemic attack (TIA).
2. Carotid stenosis in symptomatic and asymptomatic patients, to assess the cerebral vascular reserve and need for revascularization.
3. In patients with obstructive sleep apnea, to evaluate the effect of hypercapnia on cerebral circulation and risk assessment of stroke.
4. As a pre-operative test in patients undergoing coronary artery bypass grafting (CABG) and aortic root surgeries, to assess the risk of perioperative hypoperfusion stroke.

Contraindications

1. Patients with hemodynamic instability, those who are critically ill, or those on vasopressors.
2. Patients with an upper airway obstruction due to any cause.
3. As this is a provocative test, patients with a serious medical illness in the ICU may have to be stabilized first and the procedure is done in an elective time.

Prerequisites for the Test

1. The test should be done in a well-equipped neurosonology lab by an experienced technologist under the supervision of a neurologist.
2. Vital parameters like pulse, blood pressure, and oxygen saturation have to be checked.
3. Consciousness has to be evaluated. If the patient is uncooperative or hostile, the test may be deferred to a later date.
4. A crash cart and emergency medicines have to be available in the vicinity.
5. The patient and attendants have to be updated about the test.
6. Relevant medical records, CT/MRI, angiogram images, baseline extracranial Doppler, and TCD reports shall be reviewed by the neurologist before ordering the test.

The Techniques

1. The patient should be in a lying-down position on the examination cot and, with a pillow, on mild head rise. Assure the patient that the test is non-invasive. Instruct him to raise an index finger to indicate any discomfort and that the test be stopped.
2. A TCD 2-MHz probe is used to insonate at the transtemporal window. Bilateral simultaneous monitoring is strongly recommended for optimal results. In case of difficulties in insonation, a unilateral probe may be used.
3. After explaining the procedure to the patient, gel is applied and the middle cerebral arteries (MCA) are insonated bilaterally at the transtemporal window. A headband should be applied and the probes shall be fixed. The depth of insonation is usually 50–60 mm. Try to insonate the most distal segment of the MCA for a better response. The sample volume may be kept at 10 cm.

4. Once bilateral MCA waveforms are clearly recordable on the monitor, instruct the patient to breathe normally before starting breath-holding. He should not take a deep inhalation or sigh.
5. Start recording the waveforms and ask the patient to hold his breath for thirty seconds. The patient should breathe normally after thirty seconds, and the recording may be stopped five seconds after the breath-holding time.
6. Analyze the graph and repeat after five minutes if the recordings are suboptimal.
7. If the patient cannot hold his breath for thirty seconds for any reason, the rebreathing-bag technique may be used. A validated rebreathing paper/plastic bag may be applied over the nose and mouth like a silhouette for thirty seconds, and the patient should breathe normally into the bag for thirty seconds. Response is recorded, and take precaution to avoid air leaks.
8. The procedure should be stopped immediately in cases of a drop in saturation or hemodynamic instability and fluctuation in the patient's level of consciousness. The patient should be stabilized.
9. Allow the patient to breathe normally for a few minutes until he achieves his baseline flow velocities in the MCA.
10. Ask him to start hyperventilating (similar to the one performed during an EEG). The patient should breathe deep and fast for about a minute.

Difficulties Encountered during the Test

1. In bilateral simultaneous monitoring, any head movement can cause a change in the insonation angle and produce erroneous waveforms, thereby skewing the results.
2. Factors such as inadequate breath-holding, air leaks during rebreathing into a bag, and deep breathing before starting the test may lead to suboptimal results.
3. Patient cooperation is very crucial during the test. Inappropriate vigorous head movement and inability to understand the instructions due to the patient's low education status or language barrier are the common practical difficulties encountered by the technologists during the procedure.
4. Hyperventilation may lead to dizziness in normal individuals and seizures in patients known to have epilepsy. Close monitoring is essential during the test.

Interpretation of Results

1. Record the mean flow velocities at the baseline, end of breath holding, and at the end of hyperventilation.
2. Calculate VMR by the formula: VMR = (MFV at the end of breath-holding – MFV at the end of hyperventilation / MFV at baseline) x 100

   Normal VMR: > 66 percent

   Borderline Impaired: 33–65 percent

   Exhausted: < 33 percent

Cerebral Vasodilatory Reserve (CVR)

Normally, there are no conditions in which the SNS attains a dominant role in decreasing the CBF. On the other hand, by inducing vasodilatation, the PNS often plays an important role in maintaining the regional CBF distal to a stenosed artery. Therefore, it appears reasonable to test the cerebral vasodilatory reserve (only the PNS). This test involves the increase in the mean flow velocities in response to hypercapnia.

TCD Techniques

1. The patient should be in a lying-down position on the examination cot and, with a pillow, on mild head rise. Assure the patient that the test is non-invasive and instruct him to raise an index finger to indicate any discomfort and that the test be stopped.
2. A TCD 2-MHz probe is used to insonate at the transtemporal window. Bilateral simultaneous monitoring is strongly recommended for optimal results. In case of difficulties in insonation, a unilateral probe may be used.
3. After explaining the procedure to the patient, gel is applied, and the middle cerebral arteries (MCA) are insonated bilaterally at the transtemporal window. A headband should be applied and probes shall be fixed. The depth of insonation is usually 50–60 mm. Try to insonate the most distal segment of the MCA for a better response. The sample volume may be kept at 10 cm.

4. Once bilateral MCA waveforms are clearly recordable on the monitor, instruct the patient to breathe normally before starting breath-holding. He should not take a deep inhalation or sigh.
5. Start recording the waveforms and ask the patient to hold his breath for thirty seconds. The patient should breathe normally after thirty seconds, and the recording may be stopped five seconds after the breath-holding time.
6. Analyze the graph and repeat after five minutes if the recordings are suboptimal.
7. If the patient cannot hold his breath for thirty seconds for any reason, the rebreathing-bag technique may be used. A validated rebreathing paper/plastic bag may be applied over the nose and mouth like a silhouette for thirty seconds, and the patient should breathe normally into the bag for thirty seconds. Response is recorded, and take precaution to avoid air leaks.
8. Procedure should be stopped immediately in cases of a drop in the saturation or hemodynamic instability and fluctuation in the patient's level of consciousness. The patient should be stabilized.

Breath Holding Index (BHI) can be calculated as per the following formula:

BHI = **MFV (end) – MFV (baseline) x 100**

MFV (baseline) x seconds of breath holding (thirty seconds)

Normal: > 0.69

Impaired: 0–0.68

Exhausted (intracranial steal): < 0

An impaired VMR and the steal phenomenon are seen in acute strokes with proximal vessel occlusion with poor collaterals. They are seen in patients with tandem lesions with possible hemodynamic compromise.

Clinical Implications of the Test

This procedure is used to assess the cerebrovascular reserve and to prognosticate the clinical outcome of patients with acute ischemic stroke. It is an important cause for deterioration after an improvement and is seen in up to 15 percent of patients with an acute ischemic stroke, especially when associated with disturbed breathing patterns. Adequate hydration

with head-low position and optimal BP control may help in some patients, while other cases with disturbed breathing (like obstructive sleep apnea or central hypoventilation) may benefit from short-term assisted non-invasive ventilation. In patients with asymptomatic carotid disease and steal phenomenon, early revascularization may be advised, especially when they are being subjected to major vascular surgeries like a coronary artery bypass or aortic root repair.

Case Report

A sixty-four-year-old hypertensive male presented with giddiness and left-sided, sudden-onset weakness, from which he completely recovered within two hours. Neurological examination was unremarkable. CT brain did not reveal any hemorrhage. Extracranial cervical duplex ultrasound revealed bilateral carotid stenoses (> 70 percent in the right and 50–69 percent in the left). A TCD revealed antegrade flow in both ophthalmic arteries. Blunted waveforms were seen in the right MCA, consistent with a high-grade carotid stenosis (see table 1). VMR assessment showed exhausted reserve (with RRHS) in the right MCA (see table 2). He underwent an uneventful right carotid endarterectomy. He remained well and a VMR, which was performed on the third month, showed normal response in both MCAs. This case depicts the importance of VMR testing to ascertain the functional status of intracranial vessels and to prioritize intervention in affected vessels first for optimal therapeutic benefits.

Table 1 Neurovascular findings in extracranial and intracranial vessels

	RIGHT	LEFT	DEPTH
ECD – ICA	296/125	137/53	
TCD			
OA	Ante	Ante	
ICA SIPHON	161/99	128/56	
MCA M1	64/30 blunted	209/88	66
MCA M2	34/20 blunted	101/48	40
ACA A1	126/49	128/75	66
PCA P1	58/25	58/23	66
PCA- P2	73/36	59/24	66
VA PROX	85/42	105/49	60
VA DIST	97/46	96/45	70
BA	100/47	95/47	90

Table 2 Vasomotor Test Report

Temporal window	AT REST	After hypercapneic challenge	BHI
Right MCA MFV cm/sec (mean)	47	44	exhausted
Left MCA MFV cm/sec (mean)	53	73	1.24

How to Create a TCD Report

Amit Kulkarni, Jose C. Navarro
and Vijay K Sharma

The transcranial Doppler (TCD) measures real-time flow in the intracranial arteries. It is aptly described as the stethoscope of the brain and an extension of the clinical examination.

After being explained the TCD procedure, the patient is requested to lie on a comfortable bed. Various intracranial arteries are insonated via the acoustic windows at the skull's temporal, orbital, foraminal, and submandibular areas. It is very important that the TCD is performed in a standard protocol and temptation should be avoided to jump to the artery or side of the intracranial vascular pathology. Best spectral signals from various intracranial arteries are saved for reporting. (See also chapter 2 for techniques of a TCD examination).

In preparing the TCD report, the sonographer is advised to record the following:

1. Correct name, age, and gender of the patient. Hospital record number and/or the number allocated to the patient on the TCD machine should be recorded on the TCD form. Verify/confirm whether the requested procedure is appropriate for the patient's presentation or suspected diagnosis.
2. Relevant history and the indication of TCD examination.
3. Document current medications (especially if the patient is on beta blockers or vasodilators).
4. Some TCD laboratories record the information about various vascular risk factors (such as diabetes mellitus, hypertension, dyslipidaemia, ischemic heart disease, smoking, etc.).
5. The BP should be recorded, preferably from both arms.

Examination Records

After completing the diagnostic TCD examination, the sonographer obtains various hemodynamic parameters from individual arterial

segments. The number of arterial segments for individual intracranial arteries should be determined by separate laboratories.

Our laboratories report TCD hemodynamic parameters from the following arteries:

1. M1 MCA: one or two depths.
2. M2 MCA: one or two depths.
3. A1 ACA: one depth (report the direction especially if reversed).
4. A2 ACA: one depth.
5. P1 PCA: one depth.
6. P2 PCA: one depth.
7. Vertebral artery: one proximal and one deeper depth.
8. Basilar artery: proximal, mid, and distal end.
9. Ophthalmic artery: direction is the most important component.
10. ICA siphons: one or two depths.
11. Anterior and posterior communicating arteries are reported only when they are noted as a predominant finding with a clinical relevance.
12. Submandibular ICA is reported only when TCD is performed for vasospasm study.

In addition to the hemodynamic parameters from various intracranial arteries, the sonographer should mention the following on the report:

1. Bruit in an artery.
2. Presence of spontaneous microembolic signals.
3. Irregular cardiac rhythm (remember that the TCD examination may serve as the only test to demonstrate paroxysmal atrial fibrillation).
4. Any abnormal flow, especially if a blunted signal is obtained.
5. Whether the patient was uncomfortable during the test

Every neurovascular laboratory is encouraged to develop its own TCD-report form. Whether the TCD report should contain individual flow spectra from various intracranial arteries should be left to individual laboratories to decide.

Sonographers can use the following schematic diagram of the circle of Willis and mark their findings on individual arterial segments (see figure 1).

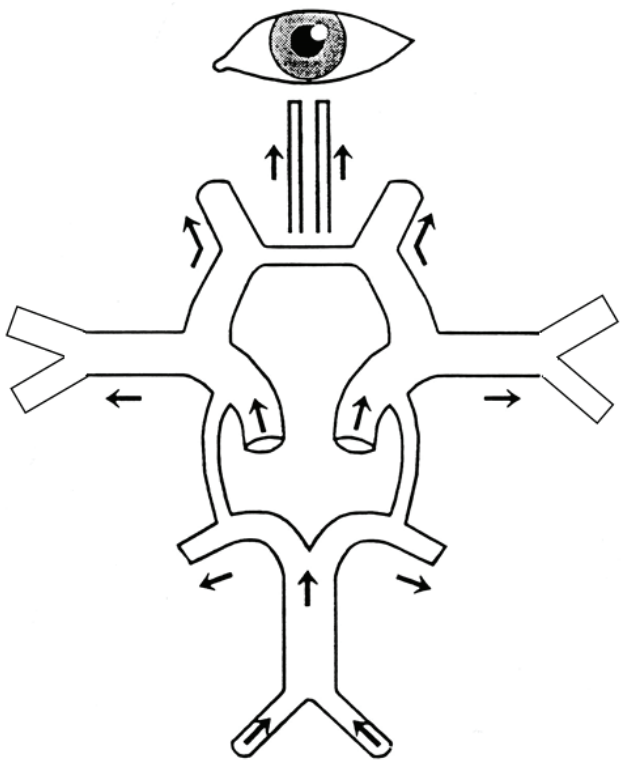
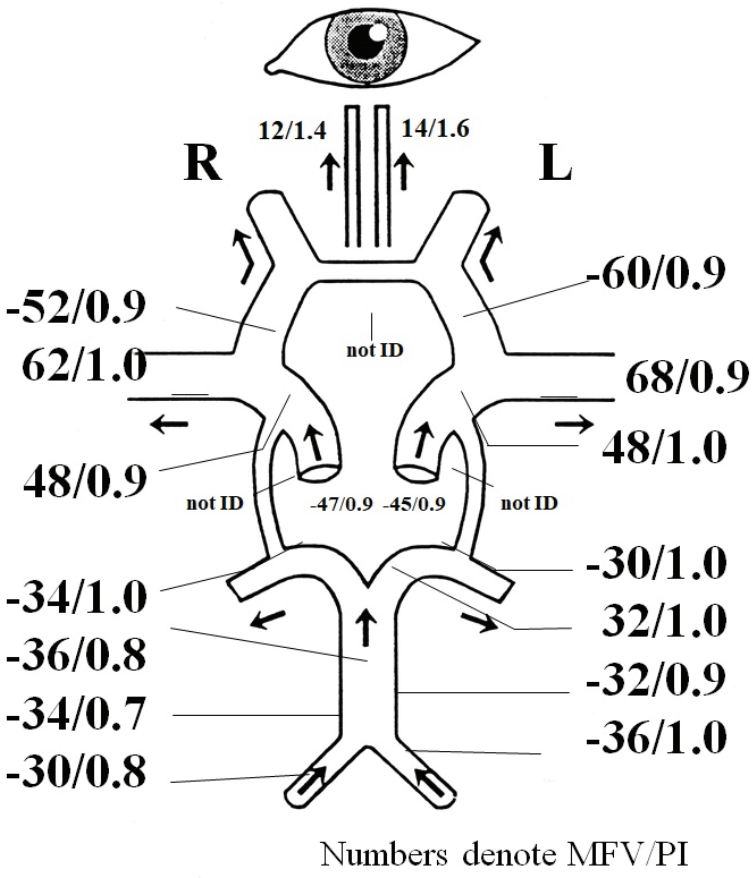


Figure 1. Schematic diagram of the circle of Willis.

We present two sample TCD forms below. Both forms convey almost similar information to the treating neurologists.

Sample TCD Report Form 1

This format contains the various branches of the circle of Willis, and the sonographer writes the mean flow velocity (MFV) and pulsatility index (PI) on various arterial segments. Adding a minus (-) sign before the flow velocity indicates the direction of blood flow. One such report form is shown in figure 1.



Sample TCD Report Form 2

The form appended below (figure 2) is another form used at some centers to report TCD findings. In this form, instead of the MFV and PI, only peak systolic (PSV) and end-diastolic velocities (EDV) are recorded.

-	Right	Left	Depth
ORBITAL	-	-	
OA	28/07 Ante	33/13 Ante	50
ICA SIPHON	72/20	86/27	64/74
-			
TEMPORAL			
MCA M1	56/32 Blunted	150/74	60
MCA M2	24/11	49/14	36/40
ACA A1	304/167 (MFV=212)	84/40	70/65
PCA P1	107/47	55/17	65
PCA P2	117/60	111/64	70
TICA		173/85 (MFV=111)	
OCCIPITAL			
VA PROX	112/50	93/51	60
VA DIST	111/59	92/46	76
BA PROX	102/55	100/50	80
BA MID	124/59	125/65	90
BA DIST	141/70	136/73	100

- The numbers denoted PSV/EDV in cm/s. The depth of insonation is in mm.

Additional Features

Some collateral flows are important and need to be reported. Anterior cross-filling occurs in the presence of severe occlusions of the extracranial carotid artery or in its intracranial segment before the bifurcation into the MCA and ACA. In such cases, the anterior communicating artery may be recruited as an important collateral and perfuses the MCA via reversed flow in the ipsilateral A1 ACA. Since a large amount of blood has to flow through a small ACOM, it would produce a high velocity jet at the midline (around 75mm from the transtemporal acoustic window). Similarly, posterior cross-filling occurs when the posterior circulation feeds the anterior circulation via the posterior communicating artery (PCOM).

Some flow diversions occur in acute intracranial occlusions. Accordingly, when the MCA is occluded (as represented by an absent or minimal flow signal in the MCA), flow may be diverted into the ipsilateral ACA, which is detected by a high velocity in the ipsilateral ACA. This hemodynamic alteration feeds the MCA territory via the trans-cortical collaterals.

In patients with an ICA occlusive disease, compensatory flow velocity increase may be noted on the contralateral side (the MCA and ACA). This should be considered at the time of reporting, and an erroneous diagnosis of stenotic flow should not be made.

In some patients, especially those of Asian ancestry, reliable flow signals may not be obtained from the temporal windows. This technical

difficulty may be overcome, at least to some extent, by using the commercially available ultrasound diagnostic contrast agents. In such cases, the sonographer should mention that the contrast agents were used for facilitating the insufficient temporal acoustic windows.

TCD Reporting

TCD is often performed by neurologists who are credentialed in neurosonologic performance and interpretation. A TCD report may be descriptive or just conclusive. Descriptive TCD reports are often considered better since they may be easily understood by other clinicians involved in patient-care as well as the neurologists who are not well-trained in neurosonology.

While reporting, the neurosonologist should take into account the normal findings. Although normative data exist, neurovascular laboratories are advised to generate such data on their population.

Examples of TCD reporting

Example 1 (Refer to figure 1): This TCD form shows normal flow velocities and the pulsatility index in various intracranial arterial segments. This may be reported as "Normal TCD study" or as "No hemodynamically significant stenosis is noted in major intracranial arteries."

Example 2 (Refer to figure 2): The report is written as:

> Blunted flow in the right MCA is suggestive of an underlying severe steno-occlusive disease. Elevated flow velocities in the ipsilateral A1 ACA represent flow diversion.
>
> Elevated flow velocities are noted in the right terminal ICA also, suggestive of an underlying moderate stenosis.
>
> Flow velocities are generally elevated in the vertebrobasilar system, which are most probably compensatory in nature from the bilateral significant stenosis in the anterior circulation.

PITFALLS OF THE EXTRACRANIAL AND TRANCRANIAL ULTRASOUND

N VENKETASUBRAMANIAN RAMANI

Introduction

A pitfall is an unexpected or hidden hazard. Pitfalls may affect the performance of an ultrasound study and may lead to incompleteness, inaccurate study results, incorrect interpretation, and, consequently, a misdiagnosis, which may lead to an inappropriate treatment of the patient. Being aware of potential hazards and taking preventive and corrective actions whenever possible allows for a more complete and accurate study that benefits the patient.

Challenging Patient Factors

There are a number of patient-related factors that may affect the performance of the study. Instead of sending the patient away, there may be some possible solutions that will still allow the study to be performed.

1. Short, thick neck. This makes it difficult to fully study the extracranial arteries. Possible solutions that would "open up" the area for insonation include turning the head in the opposite direction, using a more lateral approach, and angulating the probe more steeply.
2. High carotid bifurcation. The bifurcation may be close to or hidden by the angle of the jaw. Possible solutions include using a more lateral approach and angling the probe more steeply under the angle of the jaw.
3. Deeply seated extracranial vertebral artery. The often-used anterolateral approach may not be sufficient. Possible solutions include the lateral approach, extending the neck, placing the probe more vertically parallel to the trachea and pointing it dorsally.
4. Scars at the site of insonation. The irregular skin surface may make it difficult to apply the probe flat onto the skin. Possible solutions

include applying more ultrasound gel to provide a better coupling between the probe and the skin and placing a soft gel pack between the skin and the probe as it may better fit the skin contour.

5. Dressings at the site of insonation. The air trapped within the dressing and between the dressing and skin may not allow sufficient penetration of the ultrasound beam. Possible solutions include placing the probe close to the dressing, and then angling it toward the arteries to be insonated, applying a sterile plastic dressing over open dressings, and then placing the probe over it. The study may need to be repeated after the dressing has been made smaller or removed completely.
6. Snoring patients. The artifacts from snoring affect the Doppler signals especially during an extracranial ultrasound. Possible solutions include gently tapping the patient to induce a less deep stage of sleep.
7. Restless or breathless patients. If the patient cannot be adequately calmed down, a possible solution is to perform a very rapid study using a B-mode and color-coded Doppler to screen for significant diseases. But make a clear note in the case records that the results are preliminary and that a full study is needed. The patient can be recalled when he is calmer and more cooperative for the full study. A patient who is breathless when lying down in the usual supine position may be able to tolerate a study in a propped-up position.

Incorrect Gain Settings

1. Too low a B-mode gain would result in dark images, making it difficult to see structures clearly. Important abnormalities such as plaque may be missed. Too high a gain would result in overly bright images, which may result in over-diagnosing lesions (e.g., thrombus filling the lumen) or assessing plaque echogenicity, which is usually compared to the stenomastoid muscle, or even the intima-medial layer, as all tissues will seem bright. The correct B-mode gain setting is when the vascular lumen is dark, but other tissues are just bright enough to be easily distinguished.
2. Too low a Doppler or color-Doppler gain would make detecting the flow difficult. Too high a Doppler gain would make the waveforms and background equally bright and, thus, indistinguishable. Too high a color gain would result in “bleeding” of the color-coded flow signal into the B-mode image of the vascular walls. The right Doppler gain will be when the flow signal is clearly seen against a

dark background; the right color-Doppler gain is when the color-coded signal just fills the vascular lumen without spilling over.

Mistaking the Jugular Vein (JV) for the Common Carotid Artery (CCA)

The CCA can be distinguished from the JV:

1. CCA is more deeply situated than the more superficially lying JV.
2. CCA is largely non-compressible with the probe, while the JV is easily compressible.
3. CCA has a bifurcation, which can usually be found mid-way in the neck, while the JV has no bifurcation.
4. CCA has no valves, while the JV may have valves seen proximally at the base of the neck.
5. CCA has flow toward the brain, while the JV has flows away from the brain.
6. CCA flow is pulsatile, rhythmic, and synchronous with the heartbeat, while JV flow is more irregular.
7. CCA flow is not affected by the Valsalva maneuver, while JV flow usually stops.
8. A gentle compression by a finger at the base of the neck will not affect CCA flow, while JV flow may stop.

Mistaking the External Carotid Artery (ECA) for the Internal Carotid Artery (ICA)

The ICA can be distinguished from the ECA:

1. ICA is generally located more posterolaterally while the ECA is more anteromedial.
2. ICA diameter is larger than the ECA.
3. ICA has a dilatation (bulb) at the origin, while the ECA does not.
4. ICA has no branches while the ECA has many.
5. ICA flow is normally low resistant while ECA flow is usually high resistant.
6. ICA flow is not affected by tapping the superficial temporal artery, while ECA flow is disturbed by temporal tapping.

Missing Carotid Plaques

Carotid plaques may be variable in number, scattered, eccentric, and hypoechoic, and they may be missed by a rapid B-mode scan. Possible solutions are to perform the B-mode scan slowly, scan the entire artery from the base of the neck to the angle of the jaw, use both longitudinal and cross-sectional views (especially for eccentric plaques), and use of the color-coded Doppler as it may outline hypoechoic plaques.

Missing a Stenosis Hidden below a Calcified Carotid Plaque

Heavily calcified plaques located on the near wall may deeply cast an acoustic shadow because sound beams are unable to penetrate the plaque. Possible solutions include insonating the vessel from various angles (e.g., more medially or laterally) or from the more distal segment while steering the Doppler beam proximally to the vessel lumen.

Missing a Site of Significant Stenosis

A hastily performed study may miss a site of significant stenosis. Possible solutions are performing a careful study, using a color-coded Doppler to locate the site of maximal turbulence, placing the Doppler sample volume at that site, and interrogating for highest velocities.

Over- and Underestimating Stenosis

Using B-mode imaging only to estimate the stenosis may be inaccurate as flow is volumetric and, thus, should not be based on cross-sectional diameters or area estimations. Hypoechoic plaques may also be missed. Possible solutions are mentioned above. Validated Doppler criteria should be used to estimate the degree of stenosis.

In critical stenosis, pseudonormalization of velocities may occur. Possible solutions include looking at the waveform and at the flow characteristics in adjacent vessels.

A critically stenosed artery may have Doppler features suggestive of occlusion. Possible solutions include using the power Doppler to detect slow flow that would otherwise be missed.

Absent Temporal Window during Transcranial Ultrasound

Temporal acoustic windows may be difficult to find especially in elderly females or those of Asian or African descent. Possible solutions include carefully and slowly searching the various parts of the temporal bony window, using higher power, employing the contralateral temporal window, and using an echo contrast.

Misidentifying Vessels during a Blind Transcranial Doppler (TCD)

As the TCD is a blind technique, there is a potential misidentification of vessels. Possible solutions are the careful attention to the technique of performing the study, consideration of the vessels not insonated ~~insonable~~ via that window, direction of probe, insonation depth, blood-flow direction, and detection of bi-directional flow at the middle-anterior cerebral artery originating at the terminal internal carotid artery.

Missing Significant Extracranial Stenosis by Relying on a "Normal" Transcranial Study

The intracranial flow may appear to be normal ("pseudonormal"), for example, in the middle cerebral artery despite the stenosis of a significant internal carotid artery, intra- or extracranially. This may be due to excellent collaterals in that patient. Possible solutions include doing a complete trans- and extracranial study even if only a transcranial study is requested.

Overdiagnosing "Vasospasm" Based Solely on Intracranial Velocities during a Transcranial Ultrasound

High intracranial velocities among patients who have subarachnoid hemorrhage and are receiving hypertensive, hypervolemic, and hemodilution (triple-H) therapy have many causes. The use of the Lindegaard ratio (of the intracranial middle cerebral and extracranial internal carotid artery velocity) would help distinguish high velocities due to focal intracranial stenosis, or "vasospasm," from the generalized high velocities due to HHH therapy or other systemic causes.

Mistaking an Artifact for an Embolus during Embolus Detection Monitoring by a TCD

High intensity transient signals (HITS) may be due to emboli or artifacts. There are a number of criteria that need to be satisfied before the HITS can be classified as an embolus:

1. transient (usually < 300 ms);
2. amplitude > 3 dB or higher than that of the background blood flow;
3. unidirectional; and
4. accompanied by a "snap," "chirp," or "moan" on the audible output.

Software that can also help make the differentiation is commercially available.

Failure to Detect Emboli by a TCD

Embolism occurs unpredictably and may be missed by a single, short period of monitoring that is performed a long time after the symptoms have occurred. Possible solutions include performing the monitoring for at least thirty minutes each time and on more than one occasion. It should also be done as soon as possible after the clinical event.

Failure to Detect Right-to-Left Shunting by a TCD

An inadequate number of microbubbles arriving at the right-sided circulation may reduce the number available to cross the shunt. Possible solutions include using adequate volumes of air and saline solution, vigorous air-saline mixing, adding a small amount of the patient's blood before the mixture process, rapid injection of the air-saline mixture, post-injection milking of the injected veins toward the heart, asking the patient to perform a strong Valsalva maneuver shortly after injection, and repeating the study in various positions, such as a sitting-up position.

Misevaluation of the Cerebrovascular Reserve by Breath-Holding Technique during a TCD

Failure to adequately hold the breath or holding the breath for too short a time may lead to an incorrect assessment of the patient's cerebrovascular reserve. Possible solutions include using a timer to ensure that breath-

holding is done for at least thirty seconds to allow carbon dioxide to build up. Older patients or those with facial weakness may not be able to hold their breath for long. Possible solutions include adequately training the patient and asking the patient to re-breathe from a bag. Carbogen inhalation, especially at various concentrations, or the use of acetazolamide, may be employed instead.

Conclusions

A pitfall is a hidden hazard that interferes with the accuracy of the study. A high index of suspicion and of great care is needed while performing the study to avoid these pitfalls. Various solutions are available. Adequate training and practice are key to avoid falling victim to these pitfalls.

VASOSPASM AFTER A SUBARACHNOID HEMORRHAGE

KATHREEN JANE LARA
AND PRAKASH PALIWAL

This is about the case of a twenty-five-year-old female Chinese who was admitted with subarachnoid hemorrhage secondary to a ruptured aneurysm. The CT imaging, as seen in figures 2A and 2B, showed hyperdensities over the basal cisterns, subarachnoid space, and left frontal cortex (Fisher Grade IV). A CT angiogram (CTA) showed a left MCA aneurysm, hence a follow-up digital subtraction angiography (DSA) was done (see figures 1C and 1D). Clipping of the said aneurysm (figure 2) was done on day one of hospitalization.

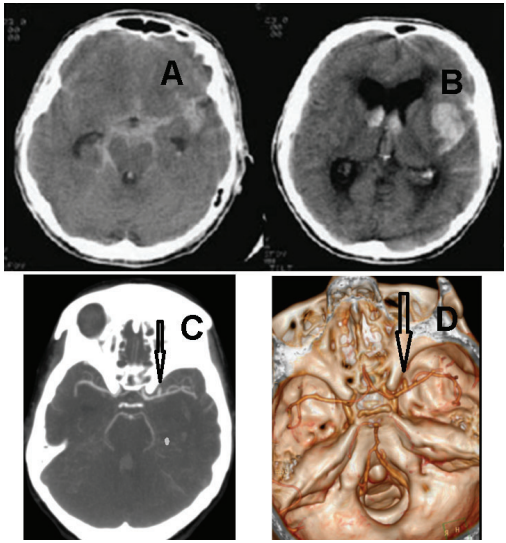


Figure 1: (A and B) Hyperdensities over the basal cisterns, subarachnoid space, and left frontal cortex. (C) DSA showing visualization of the left MCA aneurysm.

(D) CTA showing left MCA aneurysm

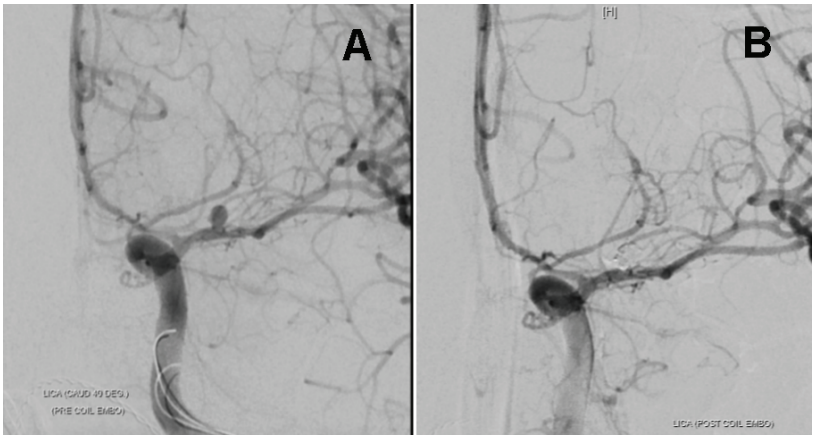


Figure 2: Coiling of the left MCA aneurysm.

A daily transcranial Doppler ultrasound was done to monitor for vasospasm. On day one, the TCD showed normal results. On the fourth day of hospitalization, the patient was noted to be lethargic, and the TCD monitoring showed severe VSP at the left MCA and moderate VSP over the right terminal ICA, as shown in figures 3A and 3B.

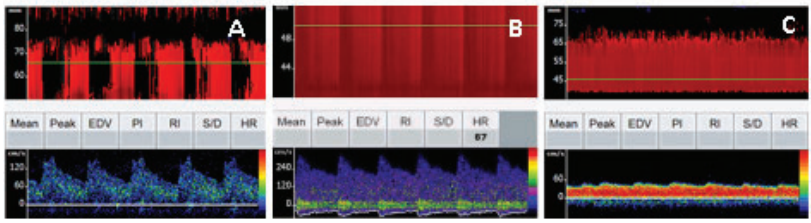


Figure 3: TCD findings at different stages of the follow-up (left MCA waveforms).

The MFV of the left proximal MCA was 226, with a Lindegaard ratio of 6.6. On day five of hospitalization, TCD monitoring showed blunting of the left MCA waveforms (figure 3C), suggestive of a worsening VSP.

A CT perfusion was done, and it showed reduced blood flow and prolonged mean transit time (MTT) along the left MCA distribution

(figures 4A and 4B). The patient was then given nimodipine, which was administered intravenously, and was also managed with aggressive triple-H therapy. An improvement in the patient's neurologic deficits was noted. Subsequent TCD monitoring showed an improvement and eventual resolution of the previously noted VSP over the left MCA. This was also seen in the succeeding CT perfusion (figures 4C and 4D). Having improved, the patient was eventually discharged, and on the third-month of follow-up, was noted to have modified Rankin Scale 1.

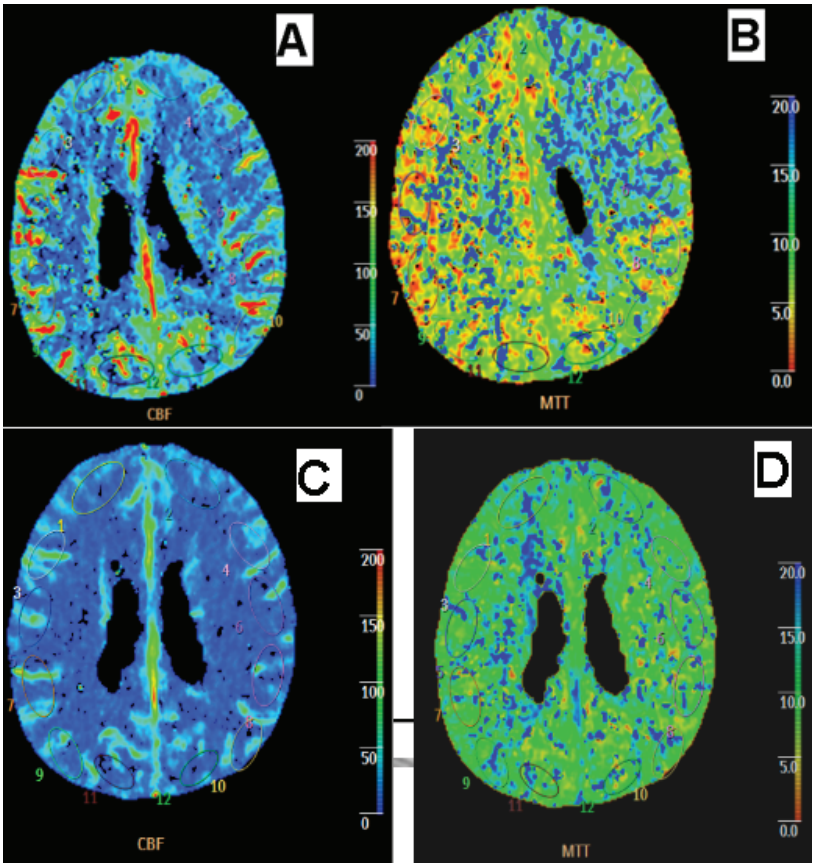


Figure 4: CT perfusion showing hypo perfusion in left MCA distribution (A and B) and resolution on follow up after procedure (C and D).

BRAIN DEATH

CYRUS G. ESCABILLAS, JOSE C. NAVARRO AND VIJAY K. SHARMA

Introduction

In modern societies, confirmation of brain death is crucial in the healthcare system—and for legal purposes, too. The early determination of brain death has assumed greater importance, especially in places with active organ donations for transplantation. According to the consensus report in 1995, the diagnosis of brain death can be made only through a set of clinical criteria, and all laboratory tests are considered supplementary or optional. Clinical tests such as the vital signs, brainstem reflexes, and the apnea challenge test form the basis for confirming brain death.

Supplementary tests for brain death largely involve the evaluation of neuronal function and cerebral blood flow. Tests that assess neuronal function are the electroencephalogram, brainstem evoked potentials, and somatosensory evoked potentials. On the other hand, tests that assess cerebral blood flow are the digital subtraction angiography, magnetic resonance angiography, computed tomography angiography, cerebral radionuclides angiography, single-photon emission tomography, and transcranial Doppler ultrasonography.

The transcranial Doppler (TCD) ultrasound is a rapid, reliable, and repeatable test that can be performed at the bedside in an intensive care setting to delineate various spectral patterns suggestive of a cerebral circulatory arrest (CCA), which is a marker of brain death.

Usefulness of the TCD in Demonstrating Cerebral Circulatory Arrest

The TCD is a non-invasive ancillary test that demonstrates characteristic cerebral blood-flow patterns of a CCA. It has a sensitivity of 90 percent and a specificity of 98 percent, as compared to the cerebral angiography, the latter being considered as the gold standard for diagnosing CCA.

However, TCDs may yield false-positive results because of some residual parenchymal flow, even at a very late stage in some cases.

TCD Methods for the Diagnosis of CCA

A scanning protocol for suspected cerebral circulatory arrest has been developed:

1. Document arterial blood pressure at the time of TCD examination.
2. Assess both MCAs (starting depth: 50 mm) and the BA (80 mm).
3. If positive MCA or BA end-diastolic flow is found = no cerebral circulatory arrest.
4. Absent end-diastolic flow = uncertain cerebral circulatory arrest (either too early or too late).
5. Reversed minimal end-diastolic flow = possible cerebral circulatory arrest (continue monitoring and document diastolic BP $\geq$ 50 mmHg).
6. Reverberating flow = probable cerebral circulatory arrest (confirm in both MCAs at 50–60 mm and BA at 80–90 mm, then monitor arrest for thirty minutes if the TCD is used as a sole confirmatory test).

Case Presentation

A forty-three-year-old man was brought to the emergency room eighteen hours after a sudden loss of consciousness. He was stuporous, and decerebrate posturing was noted. His pupils were unequal (the left pupil was bigger than the right one) with sluggish reaction, and plantar responses were upgoing. The patient was intubated for airway protection. A non-contrast head CT scan revealed massive hemorrhage on the left capsuloganglionic area (approximately 67 cc in volume) with intraventricular extension and midline shift. He was deemed to be a poor surgical candidate. On day three, he required cardiopulmonary resuscitation for cardiac arrest. Neurological examination on day six was consistent with brain death.

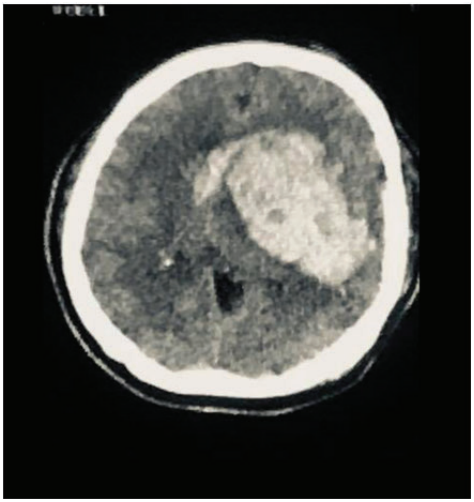


Figure 1. Non-Contrast Head CT of patient showed massive hemorrhage in the left capsuloganglionic area with mass effect

TCD Findings

A TCD examination was performed on the second day of admission by insonating the middle cerebral arteries (MCA) through the temporal window, which showed tall systolic spike, decreased diastolic flow, and increased pulsatility index (see figure 1). A repeat TCD examination on day four showed a biphasic flow (see figure 2). Such flow spectra occur when the cerebral perfusion reaches almost zero. From days four to six, the ICP was approximately equal to the systolic blood pressure, producing the biphasic flow pattern. To and fro movement of blood flow represents a zero net flow in the intracranial circulation. He was terminally extubated on day six.

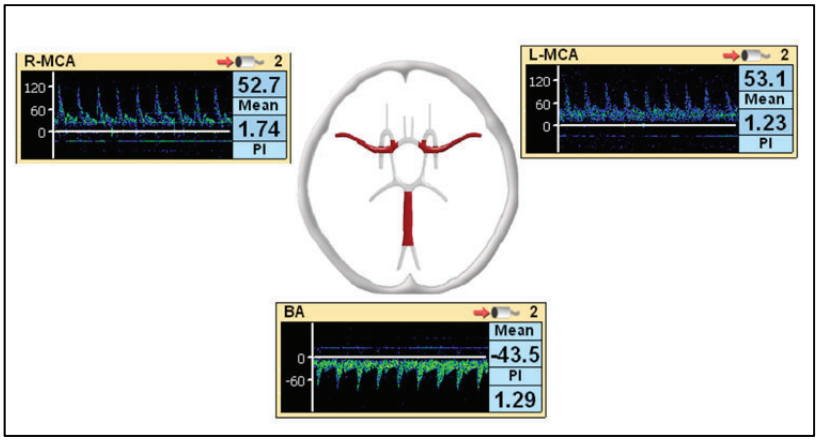


Figure 2. Day 2 Baseline TCD of patient showed tall systolic spikes with reduced diastolic wave. The increased pulsatility index correlates with increased intracranial pressure.

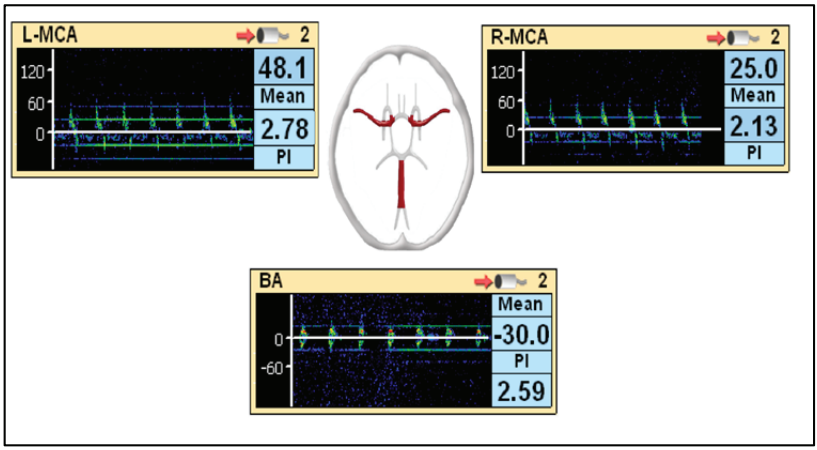


Figure 3. Repeat TCD done on day 6 showed reverberating waveform pattern.

Conclusion

The TCD is a reliable, reproducible, and portable tool for the diagnosis of cerebral circulatory arrest. This is considered as a supplemental test. Caution is advised during an interpretation of the TCD findings, if ICP

remains unknown or very high. Serial TCD evaluations increase the reliability in such cases. Findings on TCD should be interpreted in the context of clinical findings and ICP values.

SUBCLAVIAN STEAL SYNDROME

MEI-LING SHARON TAI

Introduction

Subclavian steal syndrome is a phenomenon of flow reversal in a branch of the subclavian artery, which is the result of an ipsilateral hemodynamically significant lesion of the proximal subclavian artery. When the severity of the subclavian stenosis worsens, the pressure distal to the stenosis will be reduced to the value that is less than the pressure transmitted by the contralateral normal vertebral artery via the basilar artery.

If the subclavian stenosis is severe and the affected arm is exerted, this pressure drop can cause the blood flow in the ipsilateral vertebral artery to reverse, stealing the blood from the normal subclavian artery via the contralateral vertebral artery. This will ensure enough blood supply to the affected arm.

The prevalence of this syndrome is 0.6–6.4 percent. A diagnosis of subclavian steal syndrome is suspected if there is a pulse deficit or a systolic blood pressure difference of more than 20 mmHg between the arms.

Duplex ultrasonography and transcranial Doppler (TCD) are more sensitive investigation modalities, as compared to conventional angiography for the evaluation of flow reversal. Moreover, Duplex ultrasonography and transcranial Doppler (TCD) are non-invasive, inexpensive, readily available, and can be performed rapidly.

Procedure

The patient is placed in the supine position. An examination of the common carotid artery, internal carotid, and external artery will be initially performed. The probe will then be tilted and angled posteriorly in order for the insonation of the vertebral artery and the subclavian artery to be carried out.

The insonation can be started either at the distal vertebral artery or at the subclavian artery first. The subclavian is located proximally to the proximal vertebral artery. The examination begins with the grey-scale (B-mode). During the grey-scale examination, the vertebral artery can be identified as being located between the transverse processes. This is followed by the color Doppler. The presence of subclavian steal syndrome is suspected when the Doppler spectra of one vertebral artery shows alternation or reversed flow.

Hyperemia ischemia cuff test will then be performed. This is done by inflating the arm blood pressure cuff to ≥ 20 mmHg above the systolic blood pressure for a few minutes. The cuff will then be rapidly deflated resulting in an increased blood flow in the arm. If the subclavian steal syndrome is present, the reversal of blood flow in the ipsilateral vertebral artery will be observed.

It is important to distinguish the vertebral vein signal from a retrograde artery signal. The vertebral vein is located anteriorly to the vertebral artery. It is smaller compared to the vertebral artery. The venous signal varies with respiration, but retrograde arterial signal is pulsatile. Moreover, the Valsalva maneuver can be performed to differentiate the vertebral vein from the vertebral artery.

The next step will be an assessment of the transforaminal window of TCD. This test is done so that the intracranial vertebral arteries and basilar artery can be evaluated.

Analysis of Waveform

On the basis of the hemodynamic changes in the vertebral artery, there are three types of subclavian steal syndrome:

1. Occult subclavian steal;
2. Partial (incomplete) subclavian steal; and
3. Complete subclavian steal.

Occult and partial steal result in a decrease in systolic blood flow velocity. Occult steal shows minimal hemodynamic changes. In occult steal, duplex ultrasonography imaging may show antegrade flow with mid-systolic deceleration (a transient sharp decline in blood-flow velocities at mid-systole), the rounding of a subsequent second systolic peak, and restoration of forward flow in diastole. This may temporarily change into a more abnormal waveform (with reversed late-systolic flow) in response to reactive hyperemia in the ipsilateral arm after an arm exercise.

Partial subclavian steal syndrome shows moderate hemodynamic changes. The duplex ultrasonography shows partially reversed flow. The Doppler spectrum in occult and partial subclavian steal resembles the image of a rabbit (the "bunny rabbit" sign). The spectrum of a partial steal is illustrated in Figure 1. The duplex ultrasonography also reveals stenosis of the subclavian artery.

When the proximal subclavian stenosis is moderate and severe (> 50 percent), more than 90 percent of the patients will have either intermittent or continuous flow reversal in the vertebral artery. In severe subclavian steal syndrome, alternating directions of blood flow occurs in the vertebral artery on the side of the subclavian steal syndrome (Figure 2). There are also flow changes in the contralateral vertebral artery.

Case Illustration

A sixty-two-year-old man with a history of diabetes mellitus and hypertension presented to the neurology clinic with history of recurrent episodes of vertigo. A previous evaluation by the otorhinolaryngology clinic was unremarkable. On examination of his blood pressure, he had a pulse deficit of 52 mmHg. He also had a weaker radial pulse on the left arm.

Duplex ultrasonography revealed complete flow reversal in the left vertebral artery. The Duplex ultrasonography finding of the left vertebral artery is shown in Figure 3. In addition, the compensatory flow changes were present in both the common carotid arteries. He was diagnosed with subclavian steal syndrome (complete type). The etiology of the steal syndrome was likely to be atherosclerosis. The magnetic resonance angiography (MRA) of the brain and carotid showed subclavian occlusion.

Etiology of Subclavian Steal Syndrome

The most common cause of subclavian steal syndrome is atherosclerosis. This syndrome most commonly occurs on the left side, as compared to the right, with a ratio of 4:1. The risk factors for atherosclerotic subclavian steal syndrome are male gender and age of more than fifty years. An uncommon etiology of subclavian steal is Takayasu arteritis.

Clinical Features of Subclavian Steal Syndrome

Subclavian steal syndrome is characteristically asymptomatic and mostly does not lead to serious cerebral manifestations. Approximately 38.5

percent of the patients with subclavian steal syndrome with pulse deficit of ≥ 50 mmHg are symptomatic. Unilateral reversal of the flow in the vertebral artery uncommonly leads to vertebrobasilar transient ischemic attacks, whereas bilateral vertebral flow reversal is associated with non-lateralizing cerebral ischemia.

Management

The primary management is conservative with the aim of reducing the risk for atherosclerotic subclavian steal. The medical management consists of identification and optimizing the vascular risk factors. In symptomatic patients, refractory to the medical therapy, a carotid-subclavian bypass surgery or stenting of the affected subclavian artery may be considered.

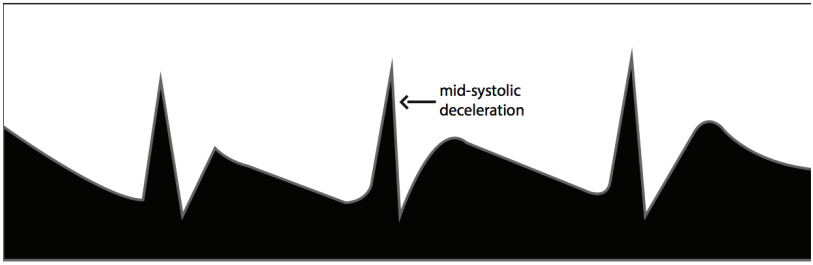


Figure 1. Partial subclavian steal spectrum, which resembles the image of a rabbit and mid-systolic deceleration.

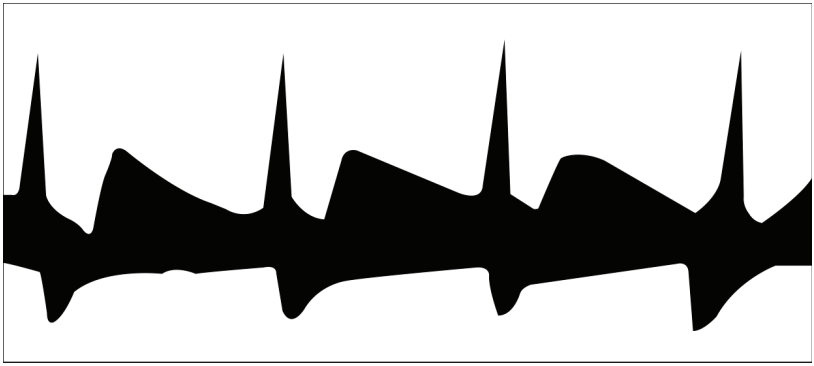


Figure 2. Alternating directions of blood flow in the vertebral artery on the side of the steal, consistent with severe subclavian steal syndrome.

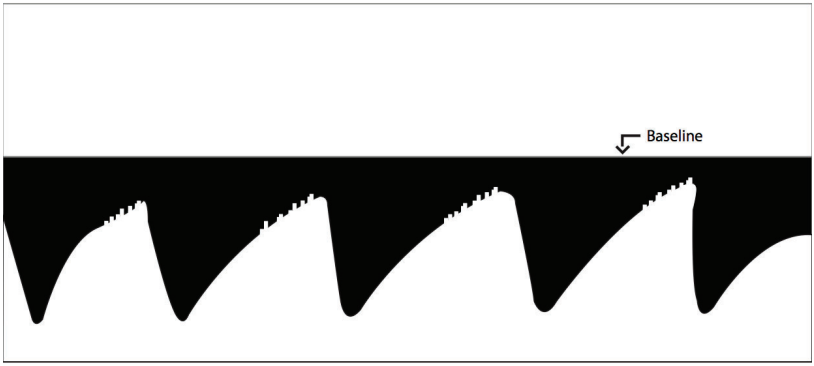


Figure 3. Complete flow reversal consistent with complete subclavian steal syndrome.

Intracranial Stenosis

Maria Cristina Z. San Jose
and Jose Danilo B. Diestro

Clinical Presentation

A sixty-one-year old hypertensive woman presented with a sudden-onset decrease in sensorium while taking a shower. Enroute to the hospital, she had a preferential movement of the right side of her body, which was noted by her relatives. Her past medical history included non-small cell lung cancer (stage four with metastases to the lung, eye, brain, and bone). She had received chemotherapy, radiotherapy, left-eye enucleation, and right upper lung lobectomy. Two months prior to this event, she was found to have a deep venous thrombosis and she had been on oral anticoagulant.

At the emergency department, she was stuporous. Her pupils were at 4 mm with sluggish constriction to light. Brainstem reflexes such as Corneal, Doll's eye, and gag reflexes were intact. Her right extremities showed withdrawal to pain, but her left upper extremity exhibited extension posturing.

Initial Neuroimaging Findings

Brain magnetic resonance (MR) diffusion weighted and apparent diffusion coefficient imaging done on presentation (see figures 1A and 1B) showed a hyperacute infarct in the right fronto-parietal region. Time of flight (TOF) MR angiography (see figure 1C) showed a cutoff at the distal portion of the right internal carotid artery (ICA). It is well known that TOF MR angiogram overestimates the findings and may not be sensitive to slow-flow states. A follow-up non-contrast brain computed tomography (CT) done five days later (see figure 1D) showed extensive infarction involving the entire territory of the right ICA. Notable edema in the region of the infarct has already caused significant midline shift leading to brain herniation.

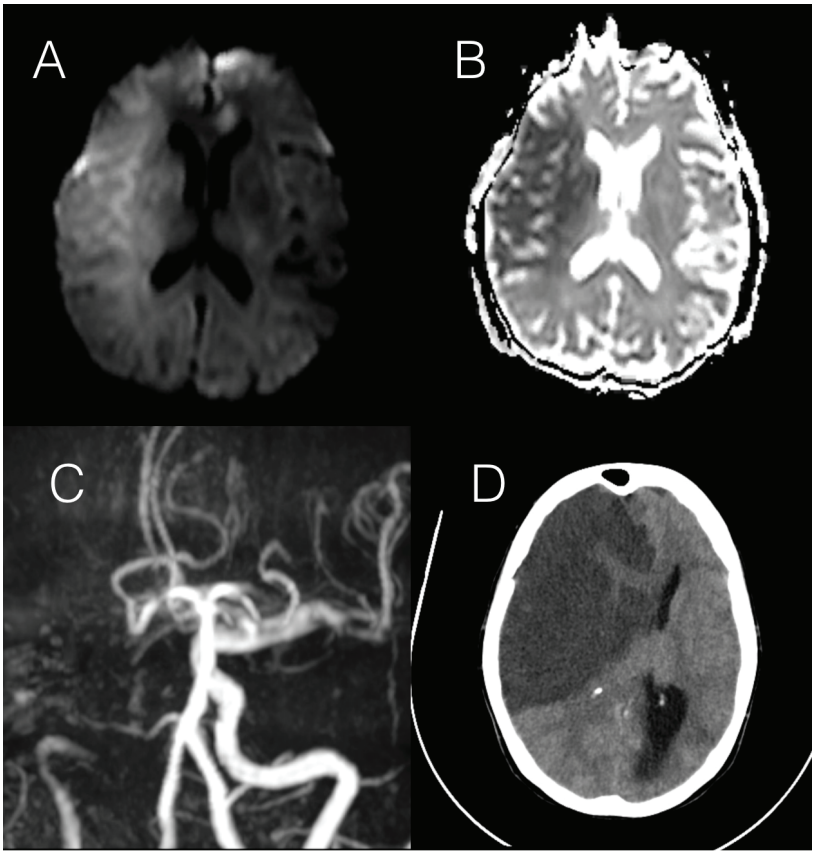


Figure 1. (A) Axial cranial magnetic resonance (MR) imaging diffusion weighted and apparent diffusion weighted imaging sequence showed a hyperintensity in the right frontoparietal area with (B) a corresponding signal drop in axial apparent diffusion coefficient sequence. (C) Time of Flight MR angiography shows absence of the distal part of the right internal carotid artery. (D) Plain axial computed tomography performed on day six showed a large hypodensity in the right ICA territory

Sonographic Findings: Carotid Duplex Sonography

B-mode imaging revealed a type-three plaque on the anterior and posterior wall of the right carotid bulb. Doppler spectra showed high resistance waveform pattern in the ICA (see figure 2A), suggestive of a more distal

stenosis. B-mode imaging and Doppler spectra in the left common carotid and the ICA showed smooth intimal lining and normal waveform patterns and velocities.

Sonographic Findings: Transcranial Color-Coded Doppler Sonography

Doppler spectra from the right MCA revealed a blunted waveform pattern compatible with severe steno-occlusive disease of the terminal ICA (see figure 2B). Mean flow velocities were uniformly low in the right MCA, with values approximately half of the left side. On the other hand, the left MCA had a normal waveform and velocities (see Figure 2C). All other vessels had normal and symmetric flow signals. Collateral flow toward the right side was not observed in any of the other vessels.

Clinical Course

Owing to the patient's poor prognosis from lung cancer, her family eventually decided against decompressive hemicraniectomy. She passed away on day eight.

Final Diagnosis

The high-resistant waveform pattern of the extracranial right ICA, with blunted waveform pattern in the right MCA, supports the findings on MRI angiography of severe steno-occlusive disease of terminal ICA.

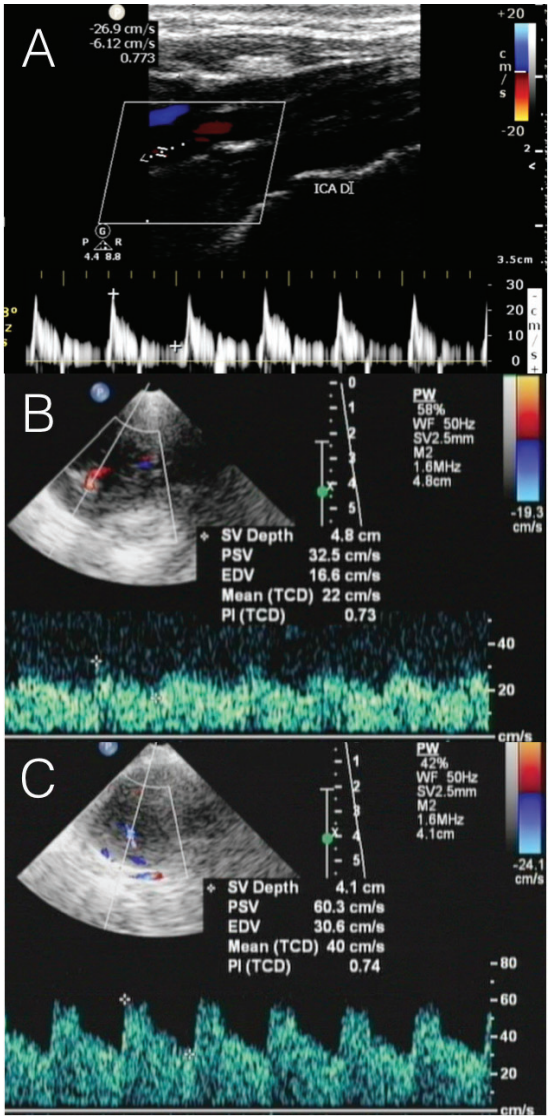


Figure 2. (A) Carotid duplex ultrasonography of the extracranial right internal carotid artery shows a higher resistance waveform pattern. (B) Transcranial color-coded duplex sonography (TCCS) of the right middle cerebral artery (MCA) showed a blunted waveform pattern. (C) TCCS of the left MCA showing a normal waveform pattern.

MOYAMOYA DISEASE

MARIA CRISTINA Z. SAN JOSE
AND JOSE DANILO B. DIESTRO

Clinical Presentation

A fifty-two-year-old male presented at the emergency department for a sudden onset of left-sided weakness accompanied with headache, facial asymmetry, dizziness, and multiple episodes of vomiting upon waking up. He had no co-morbid illnesses or prior illicit drug use but had a thirty pack-year history of smoking.

At the emergency room, except for an elevated blood pressure of 140/100, his systemic physical exam was unremarkable. He was drowsy, arousable to tapping, was able to follow commands, oriented to person only, and with a preferential gaze toward the right side. He had central facial palsy, hemiplegia, and a positive Babinski sign, all on the left side.

Initial Neuroimaging Findings

Computed tomography (CT) scan on admission showed a 3 cc bleed on the right corona radiate with significant intraventricular (IV) extension up to the fourth ventricle and associated obstructive hydrocephalus (see figures 1A and 1B). The CT angiography showed cutoffs at both the distal intracranial internal carotid arteries; subsequently, a four-vessel angiography (4VA) was done (see figure 1C). The 4VA confirmed the findings seen on the CT—tapering of both internal carotid arteries with near total occlusion at its distal segment. Small vessels demonstrating abnormal blushing and "puff of smoke" appearance are seen at the bilateral proximal A1 and M1 territories (see figure 1D). Collateral supplies from the external carotid artery through its branches, i.e., the superficial temporal artery, middle meningeal artery, and occipital artery, were clearly demonstrated (see figure 1E). In addition, collateral supply from a prominent ophthalmic artery further augments blood flow to the anterior cerebral artery. The near occlusion of the distal ICA, prominent

collateral supply from the ECA, the faint outlines of the MCA and the ACA all point to a diagnosis of Moyamoya Suzuki, stage four classification.

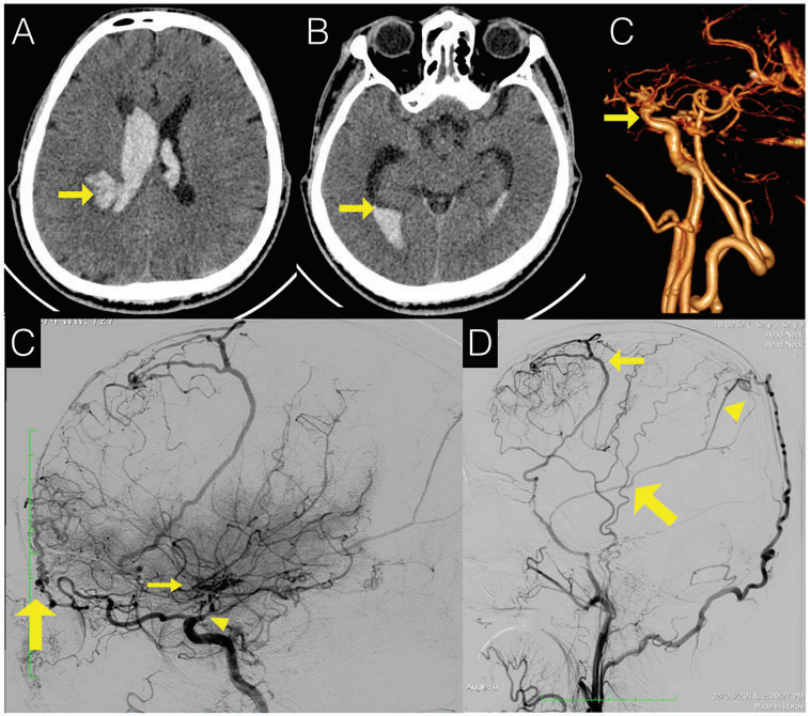


Figure 1**.** (A) Axial non-contrast cranial computed tomography (CT) scan. 3 cc bleed (arrow) in the right parietal lobe with intraventricular (IV) extension. (B) Axial non-contrast CT. Obstructive hydrocephalus and the IV extension (arrow) of the bleed in the temporal horns of the lateral ventricles. (C) Cranial CT angiography. Cutoff (arrow) on both internal carotid arteries (ICA). (D) Four vessel angiography (4VA) selective right ICA injection shows: near total occlusion of the distal part of the communicating segment of the ICA (arrowhead), Moyamoya vessels characterized by the net-like tangle of vessels over the ICA occlusion (small arrow), prominent ophthalmic artery (big arrow) consistent with extracranial-intracranial anastomosis. (D) 4VA selective right external carotid artery injection showing collateral supply to the territories of the anterior and middle cerebral arteries: occipital artery (arrow head), middle meningeal artery (small arrow), and superficial temporal artery (big arrow).

Neurosonologic Findings: Transcranial Color Doppler Sonography One Month Post-Ictus

Increased velocity, likely representative of a stenosis on the vessel site or flow diversion, is seen on the left anterior cerebral artery (ACA) (see figure 2A). In contrast, low-flow blunted waveform pattern is observed on the right ACA (see figure 2B). Both the ophthalmic arteries (OA) presented with bidirectional flow and low pulsatility. These findings on the OAs are consistent with the dilated ophthalmic arteries forming collateral circulation as seen on the 4VA (see figures 2C and 2D). Both the bilateral middle cerebral arteries (MCAs) have normal flow velocities but appear dampened. Similar to the blunted wave of the right ACA, dampened wave patterns suggest a more proximal significant stenosis or occlusion. Normal waveform patterns and velocities were observed in the posterior cerebral arteries and vertebrobasilar circulation.

The findings on the TCD are supportive of the 4VA findings of bilateral ICA occlusion with active ECA collaterals.

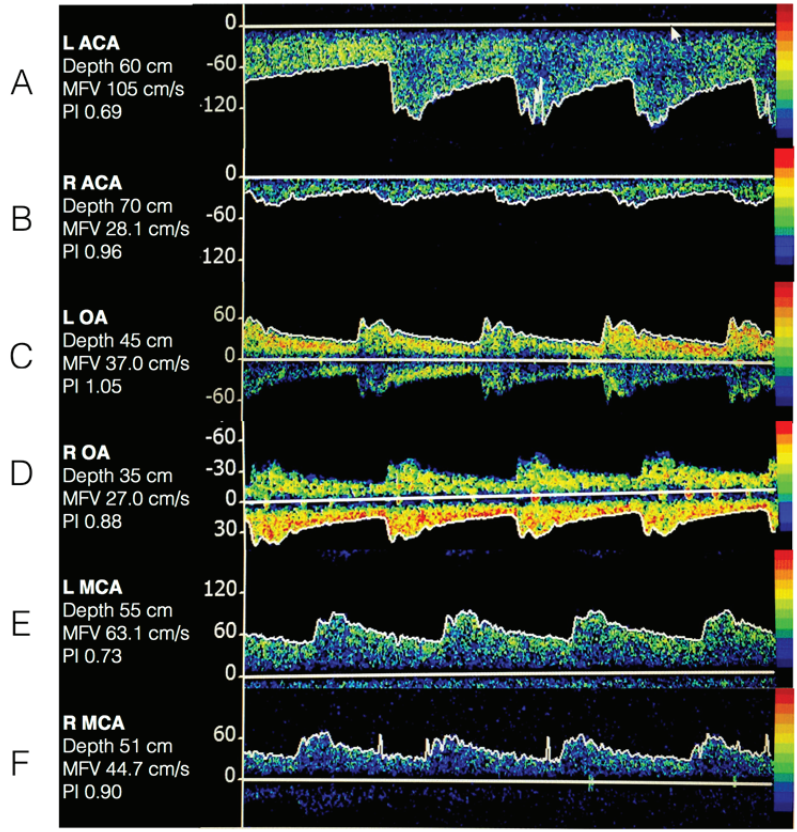


Figure 2. (A) TCD transtemporal approach, left-sided insonation. Increased MFV in the left ACA. (B) TCD transtemporal approach, right-sided insonation. Low flow and blunted waveform of the right ACA. (C and D) TCD transorbital approach. Both OAs with bidirectional flow and low pulsatility indices. (E and F) TCD transtemporal approach. Dampened waveform patterns on both middle cerebral arteries.

Clinical Course

After the patient was started on medical decompression, his sensorium improved. He was discharged on anti-hypertensive medications after a week. By then, he was already alert and conversant but still had a modified

Rankin Scale (MRS) score of four on account of the persistent left-sided hemiplegia.

Final Diagnosis

The presence of an intracranial bleed associated with severely stenotic distal internal carotid arteries and abnormal blushing of collateral vessels support the diagnosis of Moyamoya disease. Findings on the TCD further support the diagnosis—showing dampened waveform of the bilateral MCAs and blunted right ACA distal to the obstruction, increased flow velocity in the left ACA and bidirectional flow, and low pulsatility of the bilateral ophthalmic arteries.

SUGGESTED READINGS

1. Cerebrovascular Ultrasound in Stroke Prevention and Treatment. Andrei V. Alexandrov (Editor). Wiley-Blackwell. New Jersey, US. 2008.
2. Cerebrovascular Ultrasound in Stroke Prevention and Treatment, 2nd Edition. Andrei V. Alexandrov (Editor). Wiley-Blackwell, New Jersey US. 2011.
3. Sharma VK, Wong KS, Alexandrov AV. Transcranial Doppler. Front Neurol Neurosci. 2016;40:124-140.
4. Tai MS, Sharma VK. Role of Transcranial Doppler in the Evaluation of Vasculopathy in Tuberculous Meningitis. PLoS One. 2016 Oct 10;11(10):e0164266.
5. Kulkarni AA, Sharma VK. Role of transcranial Doppler in cerebrovascular disease. Neurol India. 2016 Sep-Oct;64(5):995-1001.
6. Sharma AK, Bathala L, Batra A, Mehndiratta MM, Sharma VK. Transcranial Doppler: Techniques and advanced applications: Part 2. Ann Indian Acad Neurol. 2016 Jan-Mar;19(1):102-7.
7. Wakerley BR, Kusuma Y, Yeo LL, Liang S, Kumar K, Sharma AK, Sharma VK. Usefulness of transcranial Doppler-derived cerebral hemodynamic parameters in the noninvasive assessment of intracranial pressure. J Neuroimaging. 2015 Jan-Feb;25(1):111-6.
8. Sharma VK, Yohanna K, Kawnayn G, Sarkar N, Batra A. Cerebrovascular ultrasonography for selecting patients for stroke intervention. Recent Pat CNS Drug Discov. 2013 Dec;8(3):205-19.
9. Sharma VK, Wong KS. Neurosonological examinations of transient ischemic attack. Front Neurol Neurosci. 2014;33:123-34.
10. Bathala L, Mehndiratta MM, Sharma VK. Transcranial doppler: Technique and common findings (Part 1). Ann Indian Acad Neurol. 2013 Apr;16(2):174-9.
11. Bathala L, Mehndiratta MM, Sharma VK. Cerebrovascular ultrasonography: Technique and common pitfalls. Ann Indian Acad Neurol. 2013 Jan;16(1):121-7.
12. Saqqur M, Tsivgoulis G, Nicoli F, Skoloudik D, Sharma VK, Larrue V, Eggers J, Perren F, Charalampidis P, Storie D, Shuaib A, Alexandrov AV. The role of sonolysis and sonothrombolysis in acute ischemic stroke: a systematic review and meta-analysis of

randomized controlled trials and case-control studies. J Neuroimaging. 2014 May-Jun;24(3):209-20.
13. Zhao L, Barlinn K, Sharma VK, Tsivgoulis G, Cava LF, Vasdekis SN, Teoh HL, Triantafyllou N, Chan BP, Sharma A, Voumvourakis K, Stamboulis E, Saqqur M, Harrigan MR, Albright KC, Alexandrov AV. Velocity criteria for intracranial stenosis revisited: an international multicenter study of transcranial Doppler and digital subtraction angiography. Stroke. 2011 Dec;42(12):3429-34.
14. Yeo LL, Sharma VK. Role of transcranial Doppler ultrasonography in cerebrovascular disease. Recent Pat CNS Drug Discov. 2010 Jan;5(1):1-13.
15. Lao AY, Sharma VK, Tsivgoulis G, Frey JL, Malkoff MD, Navarro JC, Alexandrov AV. Detection of right-to-left shunts: comparison between the International Consensus and Spencer Logarithmic Scale criteria. J Neuroimaging. 2008 Oct;18(4):402-6.
16. Tsivgoulis G, Sharma VK, Hoover SL, Lao AY, Ardelt AA, Malkoff MD, Alexandrov AV. Applications and advantages of power motion-mode Doppler in acute posterior circulation cerebral ischemia. Stroke. 2008 Apr;39(4):1197-204.
17. Alexandrov AV, Sharma VK, Lao AY, Tsivgoulis G, Malkoff MD, Alexandrov AW. Reversed Robin Hood syndrome in acute ischemic stroke patients. Stroke. 2007 Nov;38(11):3045-8.
18. Tsivgoulis G, Sharma VK, Lao AY, Malkoff MD, Alexandrov AV. Validation of transcranial Doppler with computed tomography angiography in acute cerebral ischemia. Stroke. 2007 Apr;38(4):1245-9.
19. Navarro JC, Lao AY, Sharma VK, Tsivgoulis G, Alexandrov AV. The accuracy of transcranial Doppler in the diagnosis of middle cerebral artery stenosis. Cerebrovasc Dis. 2007;23(5-6):325-30.
20. Sharma VK, Tsivgoulis G, Lao AY, Alexandrov AV. Role of transcranial Doppler ultrasonography in evaluation of patients with cerebrovascular disease. Curr Neurol Neurosci Rep. 2007 Jan;7(1):8-20.
21. Sonothrombolysis, by Jose C Navarro, Kristian Barlinn, Georgios Tsivgoulis and Andrei Alexandrov, Chapter 14 in Manual of Neurosonology Ed by Laszlo Csiba and Claudio Baracchini 2016 Cambridge University Press.

CONTRIBUTORS

Lokesh Bathala, MD, DM
Department of Neurology,
Aster CMI Hospital
Bangalore, India

Amit Batra, MD, DM
Department of Neurology
Max Hospital, Patparganj
New Delhi, India

Hui Meng Chang, MD
Department of Neurology
Singapore General Hospital,
Singapore

Jose Danilo B. Diestro
Stroke Fellow
Department of Neurosciences
College of Medicine-Philippine General Hospital
University of the Philippines Manila

Cyrus Escabillas
Consultant staff
Jose R. Reyes Memorial Medical Center, Department of Neurology
Asst. Professor and Active Consultant
Section of Neurology, Department of Internal Medicine
Far Eastern University Dr. Nicanor Reyes Medical Foundation Medical Center
Quezon City, Philippines

Maria Cristina Z. San Jose
Clinical Associate Professor
Department of Neurosciences
College of Medicine-Philippine General Hospital
University of the Philippines Manila

Head, Stroke Service
Institute for Neurosciences
Head, Health Services Outcomes Research Unit
Research and Biotechnology Group
St. Luke's Medical Centre Quezon City

Amit Kulkarni, MD, DM
Sagar Hospital
Bangalore, India

Komal Kumar, MD, DM
Department of Neurology
Yashodha Hospital
Hyderabad, India

Annabelle Lao-Reyes
Chair, Department of Neurosciences
Brookenshire Medical School
Davao City, Philippines

Katreen Jane Lara
Head, Stroke Unit
Cagayan Valley Medical Center
Associate Professor
Cagayan State University - College of Medicine and Surgery
St Paul University of the Philippines - College of Medicine
Cagayan Valley, Philippines

Man Mohan Mehndiratta, MD, DM
Department of Neurology
Janakpuri Superspecialty Hospital
New Delhi, India

Jose C. Navarro
University of Santo Tomas
Jose R. Reyes Medical Center
Institute of Neurosciences, St Luke’s Medical Center
Manila, Philippines

Thang Nguyen, MD
Head, Stroke Science,
115 The People Hospital,
Ho Chi Minh city, Vietnam

Prakash Paliwal, MD
Division of Neurology
National University Hospital
Singapore

N Venketasubramanian Ramani, MD
Department of Neurology
Raffles Hospital
Singapore

Reza Bavarsad Shahripour
University of Tennessee, Neurology Department
USA

Arvind Sharma, MD, DM
Department of neurology
Zydus Hospital
Ahmedabad, Gujarat, India

Suryanarayana Sharma P, MD, DM
Department of Neurology
BGS Geneagles Hospitals
Bangalore, India

Vijay K Sharma, MD
Yong Loo Lin School of Medicine
National university of Singapore
Division of Neurology
National University Hospital
Singapore

Nijasri C Suwanwela, MD
Department of Neurology
Chulalongkorn University
Bangkok, Thailand

Mei-Ling Sharon Tai, MD
Department of Neurology
University of Malaya
Kuala Lumpur, Malaysia

Kay Sin Tan, MD
Professor & Senior Consultant Neurologist
Division of Neurology
Dept of Medicine, Faculty of Medicine
University of Malaya
Pantai Valley, 50603 Kuala Lumpur
Malaysia

Girianto Tjandrawidja
Neurovascular Lab, Siloam Hospital Lippo Village - Karawaci,
Tangerang, Indonesia

Benjamin R Wakerley, MD, PhD
Gloucestershire Royal Hospital,
Gloucester GL13NN,
United Kingdom

Kusuma Yohanna , MD
Department of Neurology
National Brain Center
Jakarta, Indonesia