Transcranial Doppler (TCD) is a bedside, non-invasive, reproducible, non-expensive neuromonitoring device which can be used in many clinical scenarios. Based on the principle of the Doppler shift, blood flow velocity (FV) in the cerebral vessels can be measured. It should be noted that TCD measures blood FV and not the cerebral blood flow (CBF). However, in a given condition, FV can be used as a surrogate marker for vessel diameter or CBF. Indirectly, it can also measure the CBF and the intracranial pressure. This review describes briefly the method of using the equipment and the various indices that can be measured. The applications of TCD are varied. The review also gives an account of the various situations where TCD can be used. An inter-operator variability is an important limiting factor with the use of the TCD. However, in many of clinical scenarios, the TCD can still be used to guide for decision-making.

**Key words:** Cerebral autoregulation, cerebral blood flow velocity, cerebral vasospasm, middle cerebral artery, pulsatility index, transcranial Doppler

**INTRODUCTION**

Transcranial Doppler (TCD) is a non-invasive method of measuring blood flow velocity (FV) and its derived parameters in various intracranial arteries. In his historical article, Aaslid et al. first described this technique in 1982.1 Here, an ultrasound probe (frequency of 1–2 MHz) is used to insonate basal cerebral arteries [Figure 1]. Based on the principle of Doppler shift, the blood FV is measured. Sound waves are emitted by a piezoelectrical crystal in the probe of the TCD. These waves are directed towards basal arteries through TCD ‘acoustic windows’ by positioning the probe appropriately. The red blood cells (RBCs) in the blood stream reflect the sound waves which are captured back by the TCD probe.1,2 A positive deflection of the waveform indicates that the flow of vessel is towards the probe whereas, a negative deflection of the waveform suggests that the flow is away from the probe. As RBCs are moving particles, they change the frequency of reflected sound waves. The difference between the frequency of emitted and reflected waves measures FV of RBC and hence, the velocity of blood. It should be noted that TCD measures blood FV and not the cerebral blood flow (CBF). However, in a given condition, FV can be used as a surrogate marker for vessel diameter or CBF. TCD also measures vessel pulsatility and derives pulsatility index (PI) and resistance index (RI).

Now-a-days, transcranial colour Doppler is also available which makes insonation of various arteries easy. In some monitors, M-mode (motion mode) imaging is available which helps during the examination of the cerebral vessels.

**Angle of insonation**

The FV measured using TCD is given by the formula:

\[
FV \text{ (measured)} = FV \text{ (actual)} \times \cos \theta
\]

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If the probe is not in the same direction as of blood flow, it will create an angle (known as the angle of insonation). As evident from the formula above, measured FV will be less than actual FV. This causes two problems; first it provides inaccurate reading and second it brings inter-observer variation and makes the repetition of test erroneous. When the temporal window is used, this error is minimised by the fact that middle cerebral artery (MCA) can only be insonated within a narrow-angle. The second problem can be avoided by fixing the probe when serial testing is required. Using TCD, the variables that can be adjusted are the depth of insonation, power and filter ratio. The observed parameters are systolic FV, diastolic FV, mean FV (MFV), PI, and RI.

METHODS

Acoustic windows are parts of the skull that transmit sound waves to basal arteries. The commonly used windows are transtemporal (or temporal), suboccipital (or transforaminal), submandibular (or retromandibular) and transorbital. In many individuals (10–20%) it is not possible to get an adequate window. The probe is kept at various positions depending upon the acoustic window to be used. Commonly used windows and vessels insonated through them are provided in Table 1. The commonly observed vessel is the MCA. The probe is placed in front of the tragus and above the zygomatic arch. MCA is identified by the characteristic tracing in the upwards direction [Figure 2]. The bifurcation of the internal carotid artery (ICA) is shown in Figure 3. Anterior cerebral artery (ACA) is identified by the typical negative or downwards direction of the tracing [Figure 4].

**MEASURED AND CALCULATED VALUES**

**Flow velocity**

Systolic, diastolic and MFV is calculated by the waveform obtained. The most important of them is MFV as it shows the least variation. There are significant inter-individual variations. Therefore, serial values are more useful rather than a single value.

**Table 1: Commonly used acoustic windows, vessels insonated through them, depth, direction of flow and mean velocities**

<table>
<thead>
<tr>
<th>Artery</th>
<th>Acoustic window</th>
<th>Depth (mm)</th>
<th>Flow direction</th>
<th>MFV (cm/s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>MCA</td>
<td>Temporal</td>
<td>30-65</td>
<td>Towards</td>
<td>55±12</td>
</tr>
<tr>
<td>ACA</td>
<td>Temporal</td>
<td>60-75</td>
<td>Away</td>
<td>50±11</td>
</tr>
<tr>
<td>ICA</td>
<td>Temporal</td>
<td>40-70</td>
<td>Both sides</td>
<td></td>
</tr>
<tr>
<td>bifurcation</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PCA (P1)</td>
<td>Temporal</td>
<td>60-70</td>
<td>Towards</td>
<td>39±10</td>
</tr>
<tr>
<td>PCA (P2)</td>
<td>Temporal</td>
<td>60-70</td>
<td>Away</td>
<td>40±10</td>
</tr>
<tr>
<td>BA</td>
<td>Suboccipital</td>
<td>80-12</td>
<td>Away</td>
<td>41±10</td>
</tr>
<tr>
<td>VA</td>
<td>Suboccipital</td>
<td>60-75</td>
<td>Away</td>
<td>38±10</td>
</tr>
<tr>
<td>OA</td>
<td>Transorbital</td>
<td>45-55</td>
<td>Towards</td>
<td>21±5</td>
</tr>
<tr>
<td>Extra</td>
<td>Retromandibular</td>
<td>45-50</td>
<td>Away</td>
<td>30±9</td>
</tr>
<tr>
<td>cranial ICA</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

ICA = Internal carotid artery, MCA = Middle cerebral artery, ACA = Anterior cerebral artery, PCA = Posterior cerebral artery, BA = Basilar artery, VA = Vertebral artery, OA = Ophthalmic artery, MFV = Mean flow velocity

**Figure 1:** The transcranial Doppler probe

**Figure 2:** The transcranial Doppler waveform showing middle cerebral artery, identified by the characteristic tracing in upward direction

**Figure 3:** The transcranial Doppler waveform showing bifurcation of the internal carotid artery, identified by the characteristic tracing in both upward and downward direction

**Figure 4:** The transcranial Doppler waveform showing anterior cerebral artery, identified by the characteristic tracing in downward direction
It must be noted that there are normal variations in MFV of MCA; cyclical variations are around 10% while the difference of up to 14% may be found in right to left MCA FV. The important factors that can influence FV are haematocrit, carbon dioxide level, mean arterial pressure, age, subject arousal, exercise and pregnancy. When MFV is increased, it indicates either of two things: Decreased vessel diameter (vasospasm) or increased blood flow (hyperaemia). Decreased MFV can be due to hypotension, decreased CBF, raised intracranial pressure (ICP), or brain stem death [Table 2].[8]

**Gosling’s pulsatility index**
This derived index is given by the formula: PI = (systolic FV – diastolic FV)/MFV
PI provides information about the downstream cerebral vascular resistance. The normal value is 0.5–1.19. PI is decreased with proximal obstruction (resulting in distal vasodilation) and is increased with distal obstruction. It has been studied as a marker of ICP. It is hypothesised that with the rise in ICP, CVR increases. Although in many studies the correlation was not well-established.[9]

**Pourcelot resistivity index (RI):** Is a similar index and is not used widely. It is derived by the formula: RI = (systolic FV – diastolic FV)/systolic FV.

**Lindegaard ratio**
As we have seen that the increase in FV may be due to either hyperaemia or vasospasm, differentiation between the two is important. To differentiate between the two, the Lindegaard Ratio (LR) is used.[9]

LR = MCA MFV/extracranial ICA MFV
LR <3 indicates hyperaemia while more than 3 indicates vasospasm. Similar indices have been devised for basilar artery (BA) and ACA.

**MICROEMBOLIC SIGNALS DETECTION**
There is an easily identifiable and characteristic signal distortion when an embolus (either gaseous or particulate) passes through insonated artery. This kind of distortion is produced due to high intensity and narrow frequency signal [Figure 5]. These microembolic signals are useful in diagnosing right to left shunts and are highly accurate. They are characterised by their transient character, high intensity, chirping sound, and random appearance during a cardiac cycle.[9-11]

**APPLICATIONS OF TRANSCRANIAL DOPPLER**

**Vasospasm after subarachnoid haemorrhage**
The commonest cause of delayed neurological deterioration in a patient with subarachnoid haemorrhage (SAH) is vasospasm.[12-16] This occurs between 4th and 15th day of SAH and results in increased morbidity and mortality. If detected early, early institution of therapeutic measures can help in reducing the incidence of the adverse outcome.[17] The gold standard investigation to confirm vasospasm is angiography. However, use of this modality is limited by its being invasive, use of contrast and radiation, availability of facility and associated cost factors. TCD being easily reproducible, non-invasive, portable and cheaper is commonly employed for screening of patients at risk of vasospasm. Serial TCD is performed as single values are of less importance. One should remember that FV is inversely proportional to vessel diameter only when other variables such as CBF and viscosity of blood remain constant. The common confounding factors are haematocrit and CO2 level.[13]

The predictive values of TCD in the detection of vasospasm vary. The commonly accepted value for vasospasm detection is MFV >120 cm/s [Figure 6 and Table 3]. MFV of >120 cm/s has a good negative predictive value whereas >200 cm/s has a good positive predictive value.[18] Some of the findings in the vasospasm of MCA include MCA Vmean ≥180 cm/s, a sudden rise in MCA Vmean by >5 cm/s or 20% increase within a day during post-haemorrhage days 3–7, LR ≥6, and abrupt increase in PI >1.5 in two or more arteries suggesting increases in ICP and/or vasospasm.[4]

**Table 2: Factors affecting FV**

<table>
<thead>
<tr>
<th>Factor</th>
<th>Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>Increases up to 10 years of age, then decreases</td>
</tr>
<tr>
<td>Gender</td>
<td>Females &gt; males</td>
</tr>
<tr>
<td>Pregnancy</td>
<td>Decreases in third trimester</td>
</tr>
<tr>
<td>PaCO2</td>
<td>With decrease in PaCO2, FV decreases and vice versa</td>
</tr>
<tr>
<td>Haematocrit</td>
<td>Increases with haemodilution</td>
</tr>
</tbody>
</table>

PaCO2 = Arterial pressure of carbon dioxide, FV = Flow velocity

**Figure 5:** The transcranial Doppler waveform showing vasospasm of the middle cerebral artery. Note the encircled values
MFV = Mean flow velocity, LR = Lindegaard ratio, ICA = Internal carotid artery

Figure 6: The transcranial Doppler waveform showing various microemboli in the middle cerebral artery (white arrows)

Table 3: Vasospasm definition

<table>
<thead>
<tr>
<th>Severity of vasospasm</th>
<th>MFV (cm/s)</th>
<th>LR</th>
</tr>
</thead>
<tbody>
<tr>
<td>ICA/MCA</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mild</td>
<td>120-150</td>
<td>3-6</td>
</tr>
<tr>
<td>Moderate</td>
<td>150-200</td>
<td>3-6</td>
</tr>
<tr>
<td>Severe</td>
<td>&gt;200</td>
<td>&gt;6</td>
</tr>
<tr>
<td>BA</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mild</td>
<td>70-85</td>
<td>2-2.5</td>
</tr>
<tr>
<td>Moderate</td>
<td>&gt;85</td>
<td>2.5-3</td>
</tr>
<tr>
<td>Severe</td>
<td>&gt;85</td>
<td>&gt;3</td>
</tr>
</tbody>
</table>

MFV = Mean flow velocity, LR = Lindegaard ratio, ICA = Internal carotid artery, MCA = Middle cerebral artery, BA = Basilar artery

Although TCD has been shown to detect vasospasm with good sensitivity, its prognostic ability and potential to improve outcome is not well-established. Although the sensitivity of TCD is good in the detection of spasm of the BA its use in detection of spams in the ACA and posterior cerebral arteries is limited. American Heart Association/American Society of Anaesthesiologists has recommended TCD as a ‘reasonable tool’ to monitor for development of vasospasm. It can be concluded that although its influence over the outcome is yet not established, TCD is a useful guiding tool to screen patients with SAH. [18]

Acute ischaemic stroke

The use of TCD is increasing in the cases of acute ischaemic stroke. [19-22] It has proven to be a good diagnostic, monitoring and prognostic tool. TCD is particularly useful in the acute ischaemic stroke where repeated TCD studies can be used to track the course of an arterial occlusion before and after thrombolysis. [19] There is high sensitivity, specificity and predictive values of TCD in detecting proximal anterior circulation stroke. However, usefulness in the detection of BA and vertebral artery stroke is limited.

Continuous TCD recording significantly increased tissue plasminogen activator (tPA)-induced arterial recanalisation in the Clotbust trial. [22] In this trial, 83% of patients achieved either partial or complete recanalisation with tPA and TCD monitoring compared with 50% recanalisation with tPA treatment alone.

In addition, early TCD findings can be very useful for prognosis in patients presenting with acute ischaemic stroke. In these patients, intracranial arterial occlusion detected by TCD is associated with poor 90-day outcome, whereas a normal TCD study is predictive of early recovery. Delayed (>6 h) spontaneous recanalisation as demonstrated by TCD, is also independently associated with greater risk of haemorrhagic transformation (odds ratio: 8.9, 95% confidence interval: 2.1–33.3). In a more recent study of 489 patients with recent transient ischaemic attacks or minor stroke, MFV and the ratio of pulsatility to MFV were independent risk factors for not only stroke recurrence, but also the occurrence of other major vascular events (stroke, myocardial infarction and vascular death).

Sickle cell disease

In sickle cell disease, RBCs that are irreversibly sickled adhere to the vascular endothelium and lead to vessel occlusion. This may result in subclinical infarction, acute stroke and haemorrhage. FV of more than 200 cm/s in asymptomatic children is associated with greater risk of stroke. If a blood transfusion is instituted in such a situation, the risk of stroke can be reduced by 90% [23,24] There is the class I evidence of TCD screening of children between 2 and 6 years of age every 6 or 12 months. Blood transfusion is done if the FV is >200 cm/s to decrease sickle cells <30%.

Traumatic brain injury and measurement of intracranial pressure

PI has been most widely studied TCD parameter to measure ICP. However, there are conflicting results. To date, PI has not been validated to measure ICP. However, change in PI can be used to see trends of ICP. The other uses of TCD in cases of TBI include detection of low flow states, detection as well as monitoring of vasospasm and predicting the outcome. [25,26]

Brain stem death

According to the Indian laws, brain death is certified after clinical criteria are met. For the purpose of brain death declaration, no other test is recommended. However, TCD shows 100% specificity and 96% sensitivity for the diagnosis of brain death. [6] The typical flow patterns are demonstrated in Figure 7. There can be one of following pattern in case of brain death: An oscillating or to and fro flow (antegrade flow during systole and retrograde flow during diastole), small systolic spikes with no diastolic flow or no intracranial flow.

Carotid endarterectomy

During carotid endarterectomy (CEA), TCD can be used to monitor the need of a shunt during cross-clamping
Cerebral autoregulation
Cerebral autoregulation means maintenance of CBF despite the change in cerebral perfusion pressure between 50 and 150 mmHg. Loss of autoregulation is associated with worse outcome. Autoregulation is tested with TCD by three methods: Static autoregulation (by infusion phenylephrine), dynamic autoregulation (by inflating and deflating thigh cuff) and transient hyperaemic response test (by compressing ICA at neck). Clinical utility of these tests is not well defined. They can be used to test and compare neuro-vascular properties of various anaesthetics and other agents.[3]

Static autoregulation is tested by increasing the mean blood pressure using 0.01% phenylephrine infusion and simultaneously recording the FV. Subsequently, the estimated cerebral vascular resistance (CVRe) is calculated by the formula, CVRe = Mean blood pressure/FV. The index of autoregulation (IOR) is the ratio of percentage change in CVRe to the percentage change in mean blood pressure. An IOR of 1 implies perfect autoregulation and IOR of 0 implies disruption of autoregulation.

Dynamic autoregulation is tested by measuring the recovery of FV after a rapid decrease in mean blood pressure. Large thigh cuffs are applied and inflated to 50 mmHg above the systolic blood pressure for 3 min and then deflated to produce approximately 20 mmHg drop in the mean blood pressure. Using an algorithm, the rate of dynamic cerebral autoregulation is calculated which normally is 20%/s and is described as the rate of restoration of the FV. This means that the process is complete within approximately 5 s.

The transient hyperaemic response is performed by compressing the common carotid artery for 5–8 s and observing the change in the FV after release. A transient increase in the FV occurs due to hyperaemia, only when autoregulation is intact.

Miscellaneous uses
Besides the monitoring of the blood flow velocities in the cerebral vessels, the TCD has varied applications.[28] Some of them are tabulated in Table 4.

Table 4: Uses of TCD

| Diagnosis of extracranial and intracranial stenosis and occlusion |
| Detection and monitoring of vasospasm following aneurysmal subarachnoid haemorrhage |
| Detection of PFO and RLS |
| Detection and counting of emboli |
| Evaluation of the brain vasomotor reserve |
| Support for brain death diagnosis |
| Monitoring during carotid endarterectomy or carotid stenting |
| Monitoring during coronary artery bypass grafting |
| Monitoring during tPA treatment for acute stroke patients, identifying the point in time at which recanalisation occurs |
| Screening children with sickle cell disease uses of TCD |

CONCLUSION
TCD is a bedside, non-invasive, reproducible, non-expensive monitor which can be used in many clinical scenarios. It is also a research tool. A very important consideration is looking for confounding factors during analysis of TCD values. The serial examination is more helpful. As assumptions are made about CBF while monitoring vessel diameter, the accuracy is limited. Moreover, inter-operator variability is also a limiting factor. However, in many of clinical scenario, the TCD can be used to guide for decision-making.

Acknowledgement
We would like to thank J Adlin (President, RIMED Ltd.,) and Gaurav Seth (Arena Medical Care Pvt Ltd.,) for some of the photographs they have kindly provided for this manuscript.

Financial support and sponsorship
Nil.
Conflicts of interest
There are no conflicts of interest.

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