

Transcranial Doppler

Manish K. Marda, Hemanshu Prabhakar¹

Abstract

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 clinical 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 scenario, 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).

Department of Neuroanaesthesia and Pain, Fortis Hospital, Noida, Uttar Pradesh, ¹Department of Neuroanaesthesiology and Critical Care, Neurosciences Centre, AIIMS, New Delhi, India

Address for correspondence:

Dr. Hemanshu Prabhakar, Department of Neuroanaesthesiology and Critical Care, Neurosciences Centre, AIIMS, New Delhi - 110 029, India.

E-mail: prabhakaraiims@yahoo.co.in

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:

$$FV(\text{measured}) = FV(\text{actual}) \times \cos(\text{angle of insonation})$$

This is an open access article distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 3.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as the author is credited and the new creations are licensed under the identical terms.

For reprints contact: reprints@medknow.com

How to cite this article: Marda MK, Prabhakar H. Transcranial Doppler. J Neuroanaesthesiol Crit Care 2015;2:215-20.

Access this article online	
Quick Response Code:	Website: www.jnaccjournal.org
	DOI: 10.4103/2348-0548.165042

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.^[3-5] 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

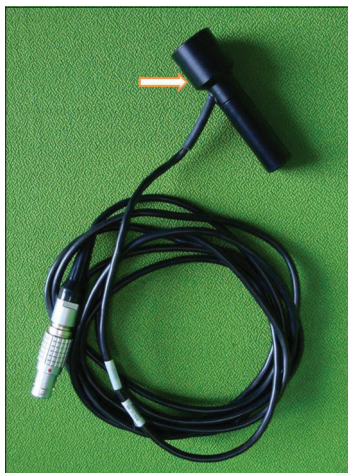


Figure 1: The transcranial Doppler probe



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

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

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

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

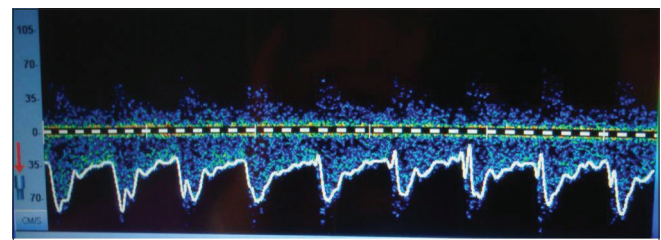


Figure 4: The transcranial Doppler waveform showing anterior cerebral artery, identified by the characteristic tracing in downward direction

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].^[6]

Gosling's pulsatility index

This derived index is given by the formula:

$$PI = (\text{systolic FV} - \text{diastolic FV}) / \text{MFV}$$

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.^[7]

Pourcelot resistivity index (RI): Is a similar index and is not used widely. It is derived by the formula: $RI = (\text{systolic FV} - \text{diastolic FV}) / \text{systolic FV}$.

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.^[8]

$$LR = \text{MCA MFV} / \text{extracranial ICA MFV}$$

LR < 3 indicates hyperaemia while more than 3 indicates vasospasm. Similar indices have been devised for basilar artery (BA) and ACA.

MICOEMBOLIC 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.^[15]

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.^[16] 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

Age	Increases up to 10 years of age, then decreases
Gender	Females > males
Pregnancy	Decreases in third trimester
PaCO ₂	With decrease in PaCO ₂ , FV decreases and vice versa
Haematocrit	Increases with haemodilution

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

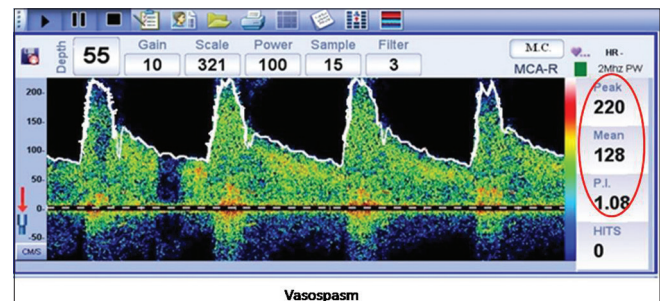


Figure 5: The transcranial Doppler waveform showing vasospasm of the middle cerebral artery. Note the encircled values



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

Table 3: Vasospasm definition

Severity of vasospasm	MFV (cm/s)	LR
ICA/MCA		
Mild	120-150	3-6
Moderate	150-200	3-6
Severe	>200	>6
BA		
Mild	70-85	2-2.5
Moderate	>85	2.5-3
Severe	>85	>3

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 spasms in the ACA and posterior cerebral artery 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

of ICA and to detect microemboli during the surgery. Severe reduction (>90%) at the onset of clamping and an increase in PI (>100%) at the release are associated with intra- and post-operative stroke. Detection of microemboli during CEA may lead to modifications in surgical and anaesthetic technique thus improving outcome.^[27] Although correlation of TCD monitoring with improved outcome has not been established yet, this provides a real-time feedback to the surgical and anaesthetic team on haemodynamics, as well as microemboli.

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, $CVRe = \text{Mean blood pressure} / FV$. 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.

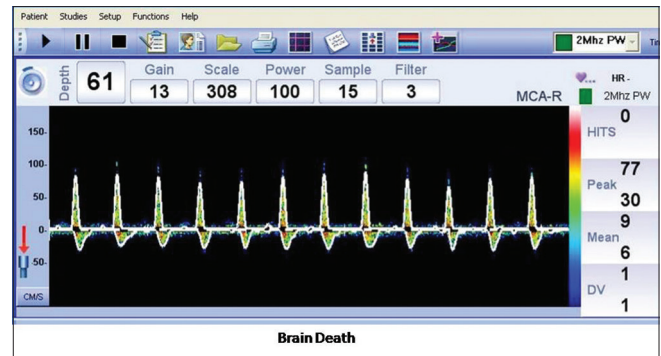


Figure 7: The transcranial Doppler waveform showing an oscillating or to and fro flow pattern, characteristic of brain death

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

tPA: Tissue plasminogen activator; PFO: Patent foramen ovale; RL: Right to left shunt; TCD: Transcranial Doppler

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.

REFERENCES

1. Aaslid R, Markwalder TM, Nornes H. Noninvasive transcranial Doppler ultrasound recording of flow velocity in basal cerebral arteries. *J Neurosurg* 1982;57:769-74.
2. DeWitt LD, Wechsler LR. Transcranial Doppler. *Stroke* 1988;19:915-21.
3. Moppett IK, Mahajan RP. Transcranial Doppler ultrasonography in anaesthesia and intensive care. *Br J Anaesth* 2004;93:710-24.
4. Tsivgoulis G, Alexandrov AV, Sloan MA. Advances in transcranial Doppler ultrasonography. *Curr Neurol Neurosci Rep* 2009;9:46-54.
5. Marinoni M, Ginanneschi A, Forleo P, Amaducci L. Technical limits in transcranial Doppler recording: Inadequate acoustic windows. *Ultrasound Med Biol* 1997;23:1275-7.
6. Nicoletto HA, Burkman MH. Transcranial Doppler series part III: Interpretation. *Am J Electroneurodiagnostic Technol* 2009;49:244-59.
7. Gosling RG, King DH. Arterial assessment by Doppler-shift ultrasound. *Proc R Soc Med* 1974;67:447-9.
8. Lindegaard KF, Nornes H, Bakke SJ, Sorteberg W, Nakstad P. Cerebral vasospasm after subarachnoid haemorrhage investigated by means of transcranial Doppler ultrasound. *Acta Neurochir Suppl (Wien)* 1988;42:81-4.
9. Dunne VG, Besser M, Ma WJ. Transcranial Doppler in carotid endarterectomy. *J Clin Neurosci* 2001;8:140-5.
10. King A, Markus HS. Doppler embolic signals in cerebrovascular disease and prediction of stroke risk: A systematic review and meta-analysis. *Stroke* 2009;40:3711-7.
11. Kobayashi K, Iguchi Y, Kimura K, Okada Y, Terasawa Y, Matsumoto N, *et al.* Contrast transcranial Doppler can diagnose large patent foramen ovale. *Cerebrovasc Dis* 2009;27:230-4.
12. Lysakowski C, Walder B, Costanza MC, Tramèr MR. Transcranial Doppler versus angiography in patients with vasospasm due to a ruptured cerebral aneurysm: A systematic review. *Stroke* 2001;32:2292-8.
13. Vora YY, Suarez-Almazor M, Steinke DE, Martin ML, Findlay JM. Role of transcranial Doppler monitoring in the diagnosis of cerebral vasospasm after subarachnoid haemorrhage. *Neurosurgery* 1999;44:1237-47.
14. Sloan MA, Burch CM, Wozniak MA, Rothman MI, Rigamonti D, Permutt T, *et al.* Transcranial Doppler detection of vertebrobasilar vasospasm following subarachnoid hemorrhage. *Stroke* 1994;25:2187-97.
15. Staalsø JM, Edsen T, Romner B, Olsen NV. Transcranial Doppler velocimetry in aneurysmal subarachnoid haemorrhage: Intra- and interobserver agreement and relation to angiographic vasospasm and mortality. *Br J Anaesth* 2013;110:577-85.
16. Frontera JA, Fernandez A, Schmidt JM, Claassen J, Wartenberg KE, Badjatia N, *et al.* Defining vasospasm after subarachnoid hemorrhage: What is the most clinically relevant definition? *Stroke* 2009;40:1963-8.
17. Biller J, Godersky JC, Adams HP Jr. Management of aneurysmal subarachnoid hemorrhage. *Stroke* 1988;19:1300-5.
18. Connolly ES Jr, Rabinstein AA, Carhuapoma JR, Derdeyn CP, Dion J, Higashida RT, *et al.* Guidelines for the management of aneurysmal subarachnoid hemorrhage: A guideline for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke* 2012;43:1711-37.
19. Rasulo FA, De Peri E, Lavinio A. Transcranial Doppler ultrasonography in intensive care. *Eur J Anaesthesiol Suppl* 2008;42:167-73.
20. Demchuk AM, Christou I, Wein TH, Felberg RA, Malkoff M, Grotta JC, *et al.* Accuracy and criteria for localizing arterial occlusion with transcranial Doppler. *J Neuroimaging* 2000;10:1-12.
21. Christou I, Alexandrov AV, Burgin WS, Wojner AW, Felberg RA, Malkoff M, *et al.* Timing of recanalization after tissue plasminogen activator therapy determined by transcranial Doppler correlates with clinical recovery from ischemic stroke. *Stroke* 2000;31:1812-6.
22. Alexandrov AV. Ultrasound enhancement of fibrinolysis. *Stroke* 2009;40 3 Suppl: S107-10.
23. Adams RJ, McKie VC, Carl EM, Nichols FT, Perry R, Brock K, *et al.* Long-term stroke risk in children with sickle cell disease screened with transcranial Doppler. *Ann Neurol* 1997;42:699-704.
24. Adams RJ, McKie VC, Hsu L, Files B, Vichinsky E, Pegelow C, *et al.* Prevention of a first stroke by transfusions in children with sickle cell anemia and abnormal results on transcranial Doppler ultrasonography. *N Engl J Med* 1998;339:5-11.
25. Van Santbrink H, Schouten JW, Steyerberg EW, Avezaat CJ, Maas AI. Serial transcranial Doppler measurements in traumatic brain injury with special focus on the early posttraumatic period. *Acta Neurochir (Wien)* 2002;144:1141-9.
26. Bellner J, Romner B, Reinstrup P, Kristiansson KA, Ryding E, Brandt L. Transcranial Doppler sonography pulsatility index (PI) reflects intracranial pressure (ICP). *Surg Neurol* 2004;62:45-51.
27. Ackerstaff RG, Moons KG, van de Vlasakker CJ, Moll FL, Vermeulen FE, Algra A, *et al.* Association of intraoperative transcranial Doppler monitoring variables with stroke from carotid endarterectomy. *Stroke* 2000;31:1817-23.
28. Naqvi J, Yap KH, Ahmad G, Ghosh J. Transcranial Doppler ultrasound: A review of the physical principles and major applications in critical care. *Int J Vasc Med* 2013;2013:629378.