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Trans-Cranial Doppler (TCD) in clinical practice in medical sciences: a review article

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ABSTRACT

Trans cranial Doppler is a portable, repeatable, bed side investigation tool of immense importance but it has not been utilized to its full potentials in Medical Sciences even where it is available. A brief indications and role of TCD in different disorders are discussed.

Key words: Trans Cranial Doppler (TCD), Subarachnoid hemorrhage (SAH), Vasospasm.

Abbreviations: TCD – Trans cranial Doppler, SAH- Subarachnoid hemorrhage, CT Scan –computed tomography scan, MCA- Middle Cerebral Artery, SDH- sub Dural hematoma, SPECT-single photon emission tomography, ICBF-intra cranial blood flow, ICP-Intra cranial pressure, CBF- cerebral blood flow, ACA-anterior Cerebral artery, ICA- Internal Carotid Artery ,MCA –middle cerebral artery, C.P.P. –cerebral perfusion pressure, P.S.V- peak systolic velocities, E.D.V - end diastolic velocities, M.V -mean velocity , P.I.- gosling pulsatile index A.V.M.S- arterio-venous malformations

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1. INTRODUCTION

Routine trans cranial Doppler (TCD) examination of the intracranial arteries was demonstrated to be possible in 1982 (Newell et al. 1992). It shows the flow velocity in particular artery and unless the diameter of the vessel is established by some other mean it is not possible to determine the actual blood flow. Thus TCD is primarily a technique for measuring relative changes in flow (MacCarteny et al. 1997). The American academy of neurology technology (AAN.1990) assessment report published in 1990 stated that TCD has established value in the assessment of patient with intracranial stenosis, collaterals, sub-arachanoid hemorrhage and brain death. More recently a panel of international experts (Babikian et al.2000) critically reviewed the literature published up to 1998 and reported the specific clinical application of TCD based on strength and quality published literature (Table 1). There recommendations were endorsed by the American society of neurosurgeons. Vasospasm is the leading cause of death in initial sub arachnoid hemorrhage. Evidence of blood in sub arachnoid space on CT scan can often predict the occurrence of vasospasm (Falyar, 1999). Although vasospasm can only be definitively diagnosed by angiography, TCD sonography provides a non invasive low risk repetitive portable tool that can be used at bed side for anticipation of worsening of vasospasm by clinical data blood flow velocity and the neurological team to initiate and adjust appropriate therapy to improve the clinical outcome.

Clinically, the onset of new or worsening neurological symptoms is the most reliable indicator of vasospasm and TCD can further aid the neurological assessment for vasospasm by measuring the cerebral blood flow velocity. Physiological changes that occur during vasospasm cause the lumen of blood vessels to decrease, so increasing the blood velocity through affected area (Falyar, 1999). Vora (Vora et al. 1999) studied the correlation between TCD and angiographic vasospasm after SAH. He corrected MCA (Middle Cerebral Artery) flow velocity and MCA / ipsilateral - contra lateral i.c.a. (internal carotid artery) velocity ratio (Lindegaurd Ratio) with angiography. They found that although inter-observer assessment regarding angiographic vasospasm was good , and despite significant correlation between mean velocities and degree of vasospasm , the clinical dependability of TCD velocities was limited and concluded that only low or very high MCA flow velocity (<120 or >= 200cm/s) reliably predict the presence or absence of clinically significant angiographic vasospasm. Intermediate velocities were not dependable and should be interpreted with caution. Schmid (Schmid et al. 1998) in his experimental study in 100 Sprague doweled rats, found that continuous bilateral laser Doppler flowmetry is indispensible to monitor adequate MCA occlusion and is highly sensitive to recognize traumatic SDH. Soustiel (Soustiel et al .1998) found, using TCD in traumatic SAH, of vertibro- basilar system that, vertibro- basilar vasospasm is more common ,than previously thought especially in association with head injury, with which, it may significantly contribute to brain stem ischemic lesion , and therefore justify specific therapeutic measure.

Manno (Manno et al. 1998) found that in patient with disturbed auto regulation after SAH, induced hypertension can alter cerebral blood flow velocity. The level of auto regulation needs to be considered when interpreting TCD

Sub-arachanoid hemorrhage: Subarachnoid hemorrhage is bleeding in the area between the brain and the thin tissues that cover the brain. Subarachnoid hemorrhage caused by injury is often seen in the elderly who have fallen and hit their head. Among the young, the most common injury leading to subarachnoid hemorrhage is motor vehicle crashes. The main symptom is a severe headache that starts suddenly (often called thunderclap headache).

Table 1
Applications of TCD

Applcations	Rating	Evidence	
		Quality	Strength
Sickle cell disease	Effective	**Class i**	**Type a**
Ischemic cerebrovascular disease	Established	**Class ii**	Type b
Subarachnoid haemorrhage	Established	**Class ii**	Type b
Arterivenous malformations	Established	**Class iii**	Type c
Cerebral circulatory arrest	Established	**Class iii**	Type c
Perioperative monitoring	Possibly useful	**Class iii**	Type c
Meningeal infection	Possibly useful	**Class iii**	Type c
Periprocedural monitoring	Investigational	**Class iii**	Type c
Migraine	Doubtful	**Class ii**	Type d
Cerebral venous thrombosis	Doubtful	**Class iii**	Type d

velocities in patients after SAH. Wordlane (Wardlaw et al. 1998) in their prospective study concluded that routine TCD service provided accurate and useful in diagnosing and managing elevated blood velocity, and ischemic neurologic deficit following SAH. And if the information from TCD is used more often in patient management, outcome might be improved, however they felt that a randomized controlled trial is necessary to asses both their point definitively. Vasospasm in aneurismal SAH results in proliferative vasculopathy. Systemic hypertension also causes vascular hypertrophy. Both of these histological changes can lead to rigidity of cerebro-vascular system, reducing its auto regulatory capacity. Szabo (Szabo et al. 1997) in his TCD study with Acetazolamide concluded that proliferative vasculopathy developed at the time of vasospasm, most have resolved and did not reduce late vasoreactivity. Co-morbidity with hypertension also did not sow to influence the late vasoreactivity toward normalization. Ambruss et al., (1996) critically studied cerebral blood flow and compared SPECT and TCD in SAH in the detection of regional brain ischemia the sensitivity of SPECT study was 90%, the specificity was 28% and the accuracy was 50%, the corresponding value of TCD were 82%, 72% and 75%. Barring chronic neurological symptoms, the specificity of SPECT study was 67% at unchanged sensitivity. Based on the results they concluded that the brain perfusion SPECT and TCD investigation are useful methods for the detection of vasospasm after SAH the combination of the two methods is recommended for the correct diagnosis of vasospasm in SAH although they require further investigation for prognostic impact of the result of these investigations. SPECT was more sensitive but less specific and less accurate.

Johnson (Johnson et al. 1996) while continuously monitoring post operative intra cranial blood flow (ICBF) in aneurismal SAH patients using a combined intra cranial pressure - laser Doppler fiber optic probe. The combined ICP & laser Doppler flowmetry fiber optic probe that permits continuous monitoring of local C.B.F., was used in post-operative management and helped to differentiate cerebral ischemia from edema and hyperemia , and was useful in titrating blood pressure and fluid management , provided direct feedback about the effectiveness of instituted therapies, and determined early on which medical management can be based and that international neuroradiology was indicated and concluded that the pressure of angiographic vasospasm and increased velocities on TCD do not always correlate with ischemia in microcirculation and that direct measurement of intracranial blood flow are obtained at variance with calculations of cerebral perfusion pressure. Clyde (Clyde et al. 1996) studied the relationship of blood velocity as measured by TCD and stable xenon computed tomography studies after aneurismal SAH and concluded the therapeutic decision based solely on blood velocity revealed by TCD ultrasonography, might be inappropriate and potentially harmful. Xenon / CT studies of ICBF are useful in guiding the management of SAH. Oxymetry and SPECT may help in such cases to measure local cerebral blood flow. The great advantage of this technique is that it enables study of specific vessels separately. However resolution is not good and there is difficulty in detecting very high and very low flow

[p] (Spender et al. 1997) the Doppler shift or frequency shift refers to change in frequency when a sound wave comes in contact with a moving object (Aaslid et al. 1982). The change in frequency can now be recorded and analyzed mathematically by Fourier transformation.

2. MATERIAL AND METHODS

Two types of Doppler devices are used, continuous wave and pulse wave Doppler. Although continuous type of devices has a good frequency resolution, there is a serious drawback in that all vessels in the path of ultrasound wave contribute to frequency shift. These problems were over come by using duplex Doppler introduced by Barber (Barber et al. 1974). This is combination of high resolution real time ultrasound and conventional Doppler imaging. The application of Doppler principle in medicine to measure blood was described, in 1960, by Satomura (Satomura et al. 1960). Aaslid (Aaslid et al. 1984) demonstrated that with certain modifications ultrasound wave could penetrate the intact skull and be used for measuring blood velocities in the basal arteries of brain. He used a 2 MHz pulse Doppler introducer through natural skull windows and developed the technique of Trans cranial Doppler (TCD). These acoustic windows are ---

1) Trans-Temporal Windows described by Aaslid (Aaslid et al. 1984)
2) Trans-Orbital Window (Spencer et al. 1986)
3) Trans- Foramina Window (Arnold et al. 1986)

Flow towards the transducer causes an upward deflection on scope and flow away from transducer a negative or downward deflection. Doppler waveform is studied for peak systolic velocities (P.S.V), end diastolic velocities (EDV), mean velocity (M.V), and gosling pulsatile index (P.I.). The pulsatile index is given by (PSV-- E.D.V/M.V). The mean velocities are--- 55+/- 12 cm/sec for MCA, 50+/-11 cm/sec for ACA, 39+/- 9 cm/sec for terminal ICA. MCA velocities more than 120cm/sec correlate with spasm seen on angiography, mean velocity (M.V.) more than 200cm/sec correlate with severe spasm and presence of ischemic defects (Aaslid et al. 1984; Seiler et al. 1986). Velocity elevation more than 50% above the basal suggests vasospasm. Lindegaard (Lindegaard et al. 1988) proposed a velocity ratio of MCA velocity/ ICA velocity in the neck. The normal ratio is 1.7 +/-0.4. A ratio of >3 corresponds to angiographic spasm and >6 to severe MCA spasm. Abnormalities in cerebral blood flow are described after head injury and can cause secondary damage (Rosner MJ et al.1990), changes in flow velocities correlates with the cerebral perfusion pressure and could results in secondary ischemic damage. Increased velocity in cerebral blood vessels after head injury could be due to vasospasm or cerebral hyperemia. This can be differentiated by studying the velocity ratio, in which MCA/ICA ratio would be higher in vasospasm than in hypermia. The increased velocity would be in all vessels in cerebral hyperemia but would be focal in vasospasm (Grolimund et al. 1988). With raised I.C.P. and reduced C.P.P. there is reduced diastolic velocity and therefore an increased pulsatility. As the I.C.P. rises further the diastolic flow becomes zero and there is flow reversal in diastole. When I.C.P. and C.P.P. becomes equal, there is a cerebral circulatory arrest, used to diagnose brain death (Hassler et al. 1989, Newell et al. 1989 and Powers et al. 1989). When these findings were found in at least 2 intra-cranial vessels, they had 91.3% sensitivity and 100% specificity for brain death (Petty et al. 1990). TCD may reveal high velocities correlating to intracranial stenosis (Lindegaard et al. 1986). TCD can detect stenosis greater than 60% in the MCA and the intracranial ICA with a sensitivity of 91% (Caplan et al. 1990). Transcranial color coded real time sonography is anew technique which has been tried as an initial modality of investigation for

suspected intracranial neoplasm and A.V.M.S (Baker G et al. 1992). Becker et al were able to identify aneurysm in 20 of 26 patients in their study (Baker et al.1991).

3. RESULTS AND DISCUSSION

Trans-cranial Doppler: an overview of its clinical application- The state Neurosonologic service, at-Houston has adopted the following clinical indications for TCD examinations at Hermann hospital and outpatient Clinic (Alexendrov et al. 2000).

3.1. Subarachnoid hemorrhage (SAH)

TCD is indicated in patient with SAH to detect and monitor arterial vasospasm. Most of the patients we monitor have non-traumatic intracranial SAH due to ruptured intra cranial aneurysm. Performing daily TCD in all grades of SAH during days 3--10 after headache , if no vasospasm is detected by day 7-8 in patient with grade -1 SAH , TCD is usually discontinued . In higher grade SAH. TCD is performed every second day after days 8-10. If no spasm is found at end of second week, TCD is discontinued. If moderate or severe vasospasm is present during second week, TCD monitoring is continued daily until spasm resolve to a mild or low moderate degree.

Signs of resolving proximal vasospasm on TCD are:

1) At site of spasm velocity decrease to normal values with low pulsatility index (P I).
2) At the site of spasm: velocity increase and Lindegaard ratio decrease or return to normal values (<3).
3) Distal to site of spasm: pulsatility index increase from very low to normal values (i.e. from 0.3 to 0.6)
4) Branches proximal to site of spasm: flow velocity decrease with pulsatility index returning to normal (from 0.3 to 0.6)

The signs of resolving distal vasospasm on TCD (Alexendrov et al. 2000):

1) Velocity increases proximal to spasm
2) Pulsatility index decreases to normal value (0.6 - 1.1) proximal to site of spasm.
3) Velocity decreases to normal value in branches proximal to spasm.

Potential TCD pitfalls include:-

1) Limited ability to detect distal branch vasospasm (a2, m2, m3)
2) Poor sensitivity to mild (<20mm hg) intracranial pressure increases.
3) High resistant flow pattern appearing due to increased cardiac output vs. increased ICP.
4) Difficult grading of spasm severity in arteries other than MCA.

3.2. Ischeamic cerebro-vascular disease

In these cases TCD can detect (Alexendrov et al. 1994, Alexendrov et al.1999; Wilterdink et al 1997):

1) Proximal intracranial arterial stenosis.
2) Arterial occlusion.
3) Collaterals.
4) Evidence of micro-embolization.

TCD can be used in patient with acute ischemia (<12 hrs) to identify major arterial occlusion or stenosis as well as to document reperfusion. TCD has 94% sensitivity and 97% specificity for hemodynamic ally significant proximal I.C.A. lesion and 81% sensitivity and 96% specificity for distal ICA lesions (Demchuk, 2000). These TCD findings are extremely helpful for grading carotid stenosis and it is a useful addition to carotid duplex and M.C.A in evaluation of acute ischaemic stroke (Razumovasky et al. 1999). TCD can be used to detect cerebral micro embolic signals (MES) in patients with potential emboligenic sources including cardiac lesions such as atrial fibrillation and patent foramen ovale (Di Tullio et al. 1993), extracranial carotid stenosis and intracranial stenosis. TCD can help to localize the source of embolization with simultaneous bilateral MCA monitoring, or unilateral MCA and ICA monitoring (Alexendrov et al. 2000).

Carotid angioplasty: Carotid angiography, also called carotid angio or an arteriogram, is an invasive X-ray imaging procedure used to detect the presence of narrowing or blockage (atherosclerosis) in the carotid arteries and determine your risk for future stroke. Carotid angiography may be performed when carotid artery disease is suspected, based on the results of other tests, such as a carotid duplex ultrasound, computed tomography angiogram (CTA) or magnetic resonance angiogram (MRA).

3.3. Sickle cell disease

The stroke prevention trial in sickle cell anemia has demonstrated a 90% relative risk reduction of ischemic stroke, when the need for prophylactic blood transfusion was determined by TCD in children of 2-16 years old. TCD was used to obtain time-averaged maximum mean flow velocity in the middle cerebral artery (MCA) or intracranial internal carotid arteries (ICA). The stop study is the most successful stroke prevention trial to date and the data provide the strongest evidence for effective clinical application of TCD to prevent ischemic stroke in children with sickle cell anemia (Admas et al. 1998; Adams et al. 1999). Potential TCD pitfalls include:

1) Inability to obtain an optimized signal (aliasing, weak signal)
2) Mirror artifact.
3) Inability to differentiate compensatory vs stenotic velocity changes.
4) Progressing lesions at non -accessible sites or arteries other than the MCA.

3.4. Arterio-venous malformation (A.V.M)

TCD can detect abnormal flow velocity and pulsatility in an AVM - feeder, and bears a signature of an extremely low resistence flow. Although TCD should not be used as a screening tool to detect AVM, it can be used for patient follow up during staged embolization. A.V.M feeders may also be found accidently during routine TCD examination and sonographers should be able to identify these flow pattern. Hassler & burger suggested the following classification (Newell et al. 1992):

1) an exclussive AVM feeder has a peak systolic velocities (P.S.V) >180cm/sec and an end diastolic velocities (E.D.V) >140cm/sec with very low p.i. values of 0.4 or less due to decreased resistance to flow.
2) Main A.V.M feeders have peak systolic velocity (P.S.V) of 140-180 cm/sec and end diastolic velocity (EDV) 120-140 cm/sec with low P.I.
3) Partial A.V.M feeder may have end -diastolic velocities greater than 80cm/sec and low p.i. values of 0.4-0.6

3.5. Cerebral circulatory arrest

TCD is used as a confirmatory test to document reverberating flow in patients which indicates brain death due to cerebral circulatory arrest. It is of particular value in patients with high barbiturate level (Petty et al. 1990; Ducrocq et al. 1998). TCD is indicated when the clinical examination indicates brain death or is unreliable. TCD may be used to determine positive end diastolic flow and rule out cerebral circulatory arrest, or may demonstrate reverberating flow and confirm clinical finding. It is extremely important to document arterial blood pressure during TCD examination to rule out transient arrest of cerebral circulation due to hypotension. Once reverberating flow is identified; it should be confirmed in both MCA and basilar artery, and monitored for 30 min, to exclude effect of transient intra-cranial pressure increases. It is prudent to wait and repeat TCD 1-2 hrs later. Progression to arrest will manifest as reverberating flow, and complete arrest will make identification of any signals more difficult.

3.6. To determine vasomotor reactivity

TCD can be used to determine vasomotor reactivity (VMR) of intra-cranial vessels (Ringelstein, 1996). MCA flow velocity decreases during hypocapnia since constriction of arterioles increases resistance of flow, increases P.I. and dampens proximal flow velocity. Hypercapnia produces arteriolar vasodilatation, decreased resistance and results in increased flow velocities and low P.I. To perform vasomotor reactivity (V.M.R) testing, the transducer should be maintained at a fixed angle during all phases using a head frame. A constant angle of insonation is required to make velocity measurements comparable. Impaired vasomotor reactivity (V.M.R) is found in patients with hemodynamically significant I.C.A stenosis or occlusions and there is a potential association with an increased risk of stroke.

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However, prospective multicenter studies are necessary to determine the value of vasomotor reactivity (V.M.R) to identify high risk patient. TCD can also be used for monitoring during procedures, particularly during cardiopulmonary bypass (CABG), carotid endarterectomy, carotid angioplasty and stenting, balloon occlusion, etc. These special applications of TCD may be potentially useful since it allows real time assessment of brain micro-embolization, brain hypo perfusion, thrombosis (Spender, 1997).

4. CONCLUSION

It is very evident that TCD is portable, repetitive, bed side equipment of immense use. The reading can be verified confirmed and valid table. The information thus provided helps in diagnosis, prognosis and management of different disease entity. It can also be uses as to see the progress or otherwise of medical and surgical therapies and alter the course of therapy if so dictated by the TCD findings.

FUTURE ISSUE

The size of equipment needs to be further small in future to become practically portable. The equipment needs to be used frequently and the results to be compared with other diagnostic modalities like angiography, CT scan, MRI scan etc to understand the information better to decrease the cost effectiveness of other equipments and decrease the financial burden on the patients.

DISCLOSURE STATEMENT

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