Transcranial Doppler Ultrasonography in Intensive Care Unit. Report of a Case with Subarachnoid Hemorrhage and Brain Death and Review of the Literature

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ABSTRACT

Transcranial Doppler (TCD) is increasingly utilized in patients with life-threatening neurologic injury and has several practical applications in neurocritical care. It holds promise for the diagnosis and monitoring of vasospasm (VSP) in patients with subarachnoid hemorrhage (SAH) and the detection of increased intracranial hypertension. In addition, it has the ability to estimate flow velocity alterations, associated with critical decrease in cerebral perfusion pressure, in patients with clinical diagnosis of brain death (BD). It is easily performed, it is relatively inexpensive and non invasive and can aid intensivists in optimizing neurovascular dynamics for the individual patient. In general, TCD findings in different clinical scenarios can be useful tools for screening neurocritical patients during clinical trials for the development of new therapeutic treatments, leading to improvement in final outcome. In this article we will try to describe basic technical issues regarding TCD instrument and examination and we will also try to provide a description of indications and findings in patients with subarachnoid hemorrhage and brain death clinical diagnosis, through the presentation of a case report with SAH and subsequent vasospasm leading to BD, treated in the Intensive Care Unit of a University Hospital.

Monitoring of physiologic functions is an important element of patient care in the Intensive Care Unit (ICU). Cardiovascular and pulmonary functions can be continuously monitored in ICU; however, the development of continuous monitoring of cerebrovascular function has lagged behind. Bedside measurement of cerebral blood flow (CBF) is a cumbersome task and requires expensive techniques that are not available in the ICU setting. Transcranial Doppler ultrasonography (TCD) was used in Neurology for the first time by Aaslid in 1982 [1] and is a non-invasive monitoring method that can be useful for bedside measurement of cerebral blood flow velocity (FV) and treatment response. It is portable, inexpensive, easily repeatable and non-invasive. Analysis of the FV waveform can indirectly provide information about CBF, cerebrovascular resistance and intracranial pressure (ICP) in neurocritically ill patients[2]. When monitoring cerebral heamodynamics, TCD can assess vessel patency, alterations in diameter of basal arteries of the Willis circle through the changes in FV, autoregulation (stable FV within a mean arterial pressure range of approximately 50-150 mmHg) and vascular reactivity to CO_2[2-5]. TCD can also monitor vasospasm after SAH and estimate blood flow patterns in patients with stroke, head trauma, hydrocephalus and intracranial masses [6-10]. Furthermore, TCD can be of significant value for guiding therapeutic decisions and prediction of outcome.

In this study we discuss methodology and basic guidelines for using TCD ultrasonography in

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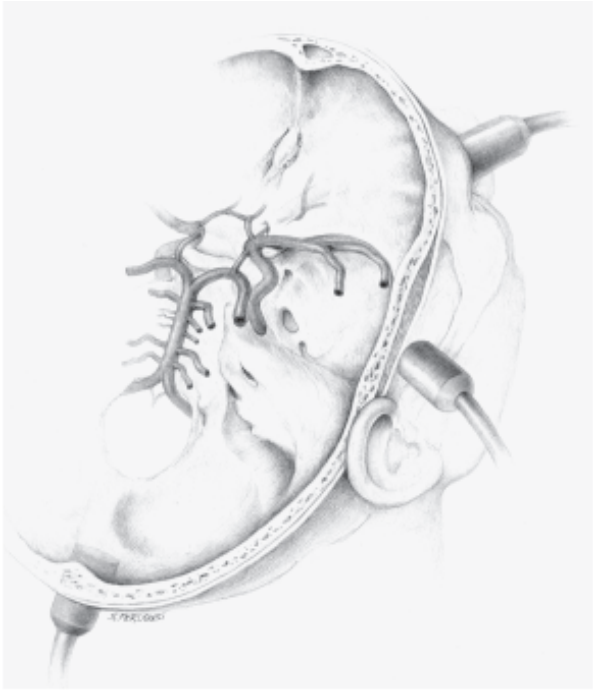
the ICU, along with clinical applications in the diagnosis and monitoring of SAH and brain death. Furthermore, we present major TCD findings of a patient with SAH, complicated by cerebral vasospasm with severe intracranial hypertension that led to cerebral circulatory arrest.

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



Examination and Doppler instrument

Ultrasound examination of a vessel by means of TCD is referred to as insonation. The TCD probe is placed over different 'acoustic windows' that are specific areas of skull where the cranial bone is thin (Figure 1). The transtemporal window is used to insonate the middle cerebral artery (MCA), the anterior cerebral artery (ACA), the posterior cerebral artery (PCA) and the terminal portion of the internal carotid artery (TICA) before its bifurcation. The transorbital window is used for insonating the ophthalmic artery (OA) and the internal carotid artery at the siphon level, whereas the transforaminal (occipital) window allows insonation of the distal vertebral arteries (VA) and the basilar artery (BA)[11,12]. Although the temporal bone allows the best insonation of the brain and its vessels, there are still important limitations. An inadequate temporal acoustic window is the major cause of inaccurate TCD measurements and has a prevalence of approximately 14.5%. In addition, bone has the highest acoustic impedance, resulting in great attenuation of the ultrasonic signal before it reaches the brain[13].

All TCD devices use a pulsed Doppler system with a low-frequency, 1-2 MHz ultrasonic signal that provides adequate penetration through thin areas of the cranium and exploits the Doppler effect, in order to determine the speed and direction (velocity) of flow in blood vessels. According to Christian Andreas Doppler, the Doppler effect is the change in the frequency or wavelength of a wave due to relative movement between the sound source and the receiver. The change in frequency is called Doppler frequency shift and is the difference between the transmitted and reflected frequencies. A positive shift indicates flow towards the transducer and a negative shift implies the opposite. Accurate estimation of the Doppler frequency shift requires knowledge of the angle between the sound beam and the flow direction (angle of insonation θ) and is inversely related to the cosine of this angle (cos θ)[11].

The ultrasonic beam is produced by piezoelectric crystals that have been stimulated electrically. This beam bounces off the erythrocytes within the insonated artery. The reflected signal is received by the transducer and converted to an electrical signal. This information is subtracted from the transmitting signal and then processed to obtain a waveform that allows accurate determination of blood flow velocities and direction of flow. Because the calculation of blood flow velocity depends on the cosine of the angle of insonation, the highest velocities are found with a 0-degree angle (cos θ=1), whereas it becomes impossible to accurately determine velocity at insonation angles approaching 90 degrees (cos 90=0).

There are two categories of Doppler transducers: those that transmit and receive frequencies continuously (continuous wave-CW Doppler) and those that transmit or receive intermittently (pulse wave-PW Doppler). During TCD ultrasonography, CW Doppler transducers cannot localize the depth of reflecting signal,

Table 1: Accepted Guidelines for a normal TCD examination

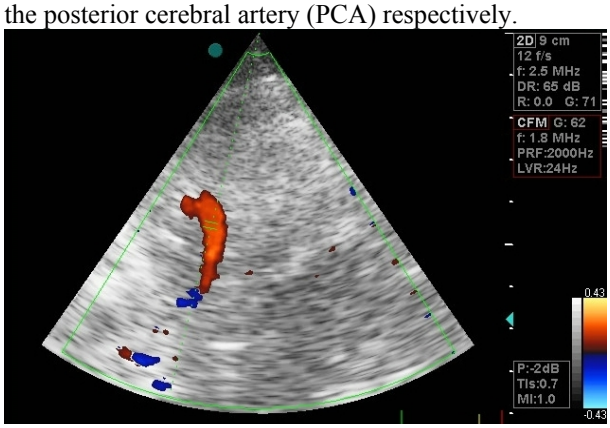
Artery	Window	Depth (mm)	Direction	Mean flow velocity
MCA	Temopral	30 to 60	Toward probe	55 ±12 cm/sec
ACA	Temporal	60 to 85	Away	50 ±11 cm/sec
PCA	Temporal	60 to70	Bidirectional	40 ±10 cm/sec
TICA	Temporal	55 to 65	Toward	39 ±10 cm/sec
ICA (siphon)	Orbital	60 to 80	Bidirectional	45 ±15 cm/sec
OA	Orbital	40 to 60	Toward	20 ±10 cm/sec
VA	Occipital	60 to 80	Away	38 ±10 cm/sec
BA	Occipital	80 to 110	Away	41 ±10 cm/sec

MCA: mean cerebral artery, **ACA**: anterior cerebral artery, **PCA**: posterior cerebral artery, **TICA**: terminal internal carotid artery, **ICA**: internal carotid artery, **VA**: vertebral artery, **BA**: basilar artery.

whereas PW Doppler transducers permit knowledge of depth of insonated vessels, and are exclusively used for blood flow velocity estimation. A hand-held transducer, which operates as both transmitter and receiver, is used by the technician who takes measurements at selected sites, 25-100 mm from the transducer[11,14].

In addition, transcranial ultrasonography may allow the examination of the parenchyma of the central nervous system (CNS) through the intact skull by means of B-mode ultrasonographic imaging techniques, with the aid of many different transducers of relative low frequency (2-3.5 MHz), allowing at the same time direct visualization of insonated vessels. Ultrasonographic B-mode imaging yields two-dimensional image plan segments that are liberally angled and have relatively free axial or coronal orientations. This method is the standard procedure for static imaging and allows vessel visualization through transcranial color-coded ultrasonography techniques (TCCS), (Figure 2)[15].

Figure 2: Transcranial color-coded B-Mode ultrasonography (TCCS) of the left anterior cerebral circulation. The upper red coded segment is the middle cerebral artery (MCA), followed by the blue coded A1 segment of ipsilateral anterior cerebral artery (ACA). The two spots at the bottom of the image that are placed next to each other (red and blue coded) are the controlateral right A1 segment of ACA and the right P1 segment of the posterior cerebral artery (PCA) respectively.



Except for flow velocities [peak systolic (PS), end-diastolic (ED), mean] estimation, TCD permits calculation of pulsatility index (PI) that is considered a reliable marker of resistance distal to the insonated site. It is easily calculated by the Gosling equation: PI= (Peak systolic-end diastolic FV)/Mean FV. Another useful measure is Pourcelot's resistance index (RI) that is a measure of peripheral flow resistance. R I= (Peak systolic-end diastolic FV)/Peak systolic FV. High vascular resistance is characterized by low diastolic flow velocities and a high RI (>0.8), whereas reduced RI (<0.8) is associated with high diastolic flow and low vascular resistance[16-18].

Different vascular scenarios are related to different patterns of blood flow velocities and PI[19].

1. Pure focal narrowing at the site of insonation will cause an increase in FV.
2. Narrowing or obstructing lesions proximal to the insonation site will cause a decrease in FV at the insonation site.
3. Distal (downstream) increased vascular resistance will decrease FV and increase PI proximal to lesion (PI>1.2).

Different depth range, flow direction and normal age-related flow velocities have been established for each vessel (Table 1)[12].

Report of a case

A 55-year-old man was transferred to the University Hospital of Alexandroupolis, Greece with a 30 minutes history of abrupt unconsciousness at his house. The initial Glasgow Coma Scale (GCS) score at the Emergency Department was 8, but the patient rapidly deteriorated to a GCS score of 6 and developed anisocoria with the right pupil greater. Computed tomography (CT) scan of the brain revealed massive right cerebral, intraventricular and subarachnoid hemorrhage, brain swelling and a midline shift from right-to-left >10 mm (Figure 3). Due to high clinical suspicion for SAH attributed to aneurysm rapture, an angiogram was performed a few hours later that demonstrated the presence of a large saccular aneurysm at the M1 segment of the right MCA (Figure 4). This patient underwent a large right frontotemporoparietal decompressive craniectomy with dural augmentation, a parallel aneurysmal clipping and an intraventricular ICP monitor placement and subsequently, he was admitted to the ICU.

GE Medical Systems Vivid 3 Ultrasound technology was used for performing TCD examinations with the aid of a 2.5 MHz transducer. The 2D imaging and anatomical M-mode options permitted direct visualization of intracranial vessels and tracking spectral profile of flow velocities utilizing PW-Doppler technology. On postoperative day 1 TCD findings were normal, with right PS MCA FV of approximately 140 cm/sec, ED MCA FV 60 cm/sec and mean FV around 96 cm/sec. PI and RI were also within normal range (Figure 5). The patient remained under sedation with propofol and remifentanil, hemodynamically stable with no use of inotro-

Figure 3: Computed tomography (CT) scan of the brain demonstrating massive right cerebral, intraventricular and subarachnoid hemorrhage, brain swelling and a midline shift from right-to-left >10 mm.

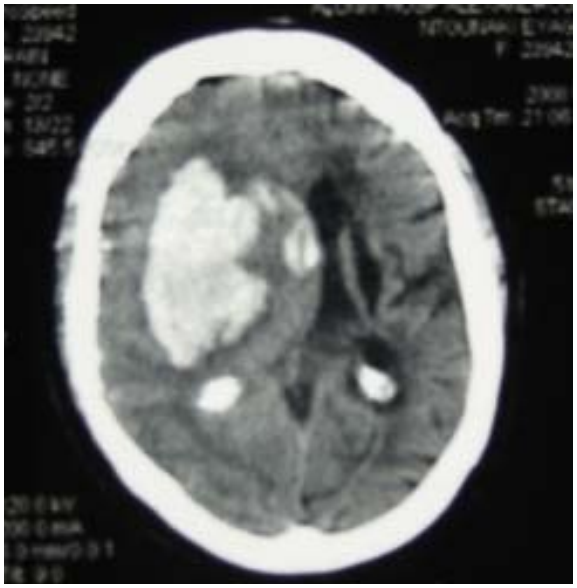


Figure 4: Classical angiogram of anterior cerebral circulation demonstrating the presence of a large saccular aneurysm at the M1 segment of the right MCA.

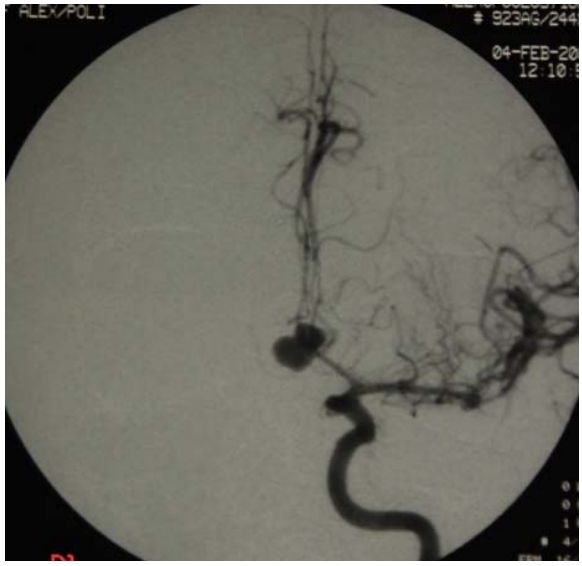
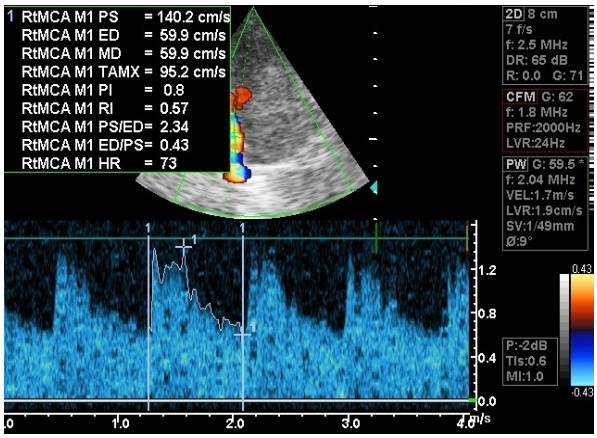
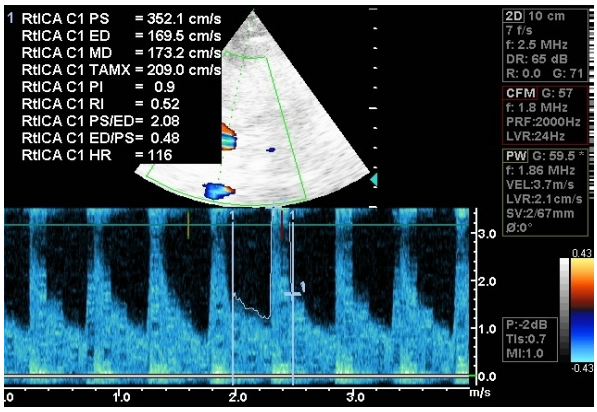


Figure 5: TCCS ultrasonography with spectral waveforms obtained from the right MCA of the patient, 1 day after surgery, demonstrating normal findings [right peak systolic (PS) MCA FV of approximately 140 cm/sec, end-diastolic (ED) MCA FV 60 cm/sec and mean FV around 96 cm/sec]. PI and RI are also within normal range limits. Velocity scale (m/sec) is displayed on the right of the screen.



pes, whereas his blood gases were normal under controlled mechanical ventilation. Three days later, TCD measurements were consistent with severe arterial narrowing with increased PS (352 cm/sec), ED (170 cm/sec) and mean FVs (210 cm/sec), mainly in the right MCA (Figure 6). The above flow velocity pattern could have been related to a severe-to-critical vasospasm

Figure 6: TCCS ultrasonography with spectral wave-forms obtained from the right MCA of the patient, 3 days after surgery demonstrating severe arterial narrowing with increased PS (352 cm/sec), ED (170 cm/sec) and mean FVs (210 cm/sec). This pattern is consistent with vasospasm or cere-bral hyperemia. Since ICP was not increased, TCD findings support the diagnosis of spasm. Velocity scale (m/sec) is displayed on the right of the screen (see text for details).



with no parallel upstroke in ICP values, indicating absence of hyperemia. Triple H therapy (hypertension, hemodilution, hypervolaemia) was instituted, however on postoperative day 7, the ICP reached values between 35-40 mmHg with the appearance of bilateral pupil dilatation. Despite cerebrospinal fluid (CSF) draining, mannitol administration and application of hyperventilation for a few minutes, ICP remained unchanged and an urgent CT scan was performed, showing a large area of hypo-density involving the right MCA. That was consistent with an acute cerebral ischemic infarct due to vasospasm.

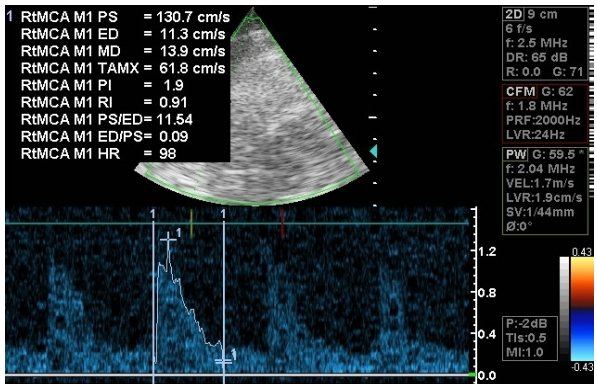
Since neurosurgeons had nothing to offer, sedation was interrupted while serial TCD examinations were performed until the clinical establishment of brain death diagnosis. Figure 7 reveals a severe reduction in ED MCA FV (14 cm/sec) with a parallel increase in PI (1.9) and RI (0.9), findings that are consistent with severe intracranial hypertension (ICP=45 mmHg). Figure 8 was performed one day before undertaking the 1st diagnostic tests for BD and shows systolic peaks of approximately 60 cm/sec with diastolic flow reversal (oscillating blood flow) whereas Cerebral perfusion pressure (CPP) was 20 mmHg.

Discussion

1. Clinical application of TCD in subarachnoid hemorrhage

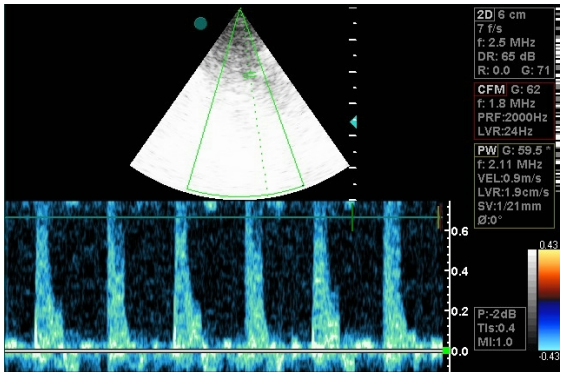
Subarachnoid hemorrhage is commonly caused by rupture of an intracranial saccular aneurysm. It occurs with a rate of 10-28 cases per 100.000 persons per year[20]. Cerebral vasospasm is the constriction of cerebral blood vessels due to the presence of blood in the subarachnoid space and if severe enough, may result in ischemia to the brain, whereas it is associated with an increase in mortality by 1.5 to 3-fold during the first 2 weeks after SAH. Vasospasm typically occurs within 3 to 21 days after SAH and may last from 12 to 16 days. Arterial narrowing on angiography occurs in a majority of patients, although actual clinical deficits develop in only 30% of patients[21]. Fisher et al used the term delayed ischemic defect attributable to this vessel narrowing[22]. According to the Report of the Therapeutics and Technology Assessment Subcommittee of the American Academy

Figure 7: TCCS ultrasonography with spectral wave-forms obtained from the right MCA of the patient, 1 day after confirmation of cerebral ischemia with CT scan, attributed to vasospasm. There is an apparent severe reduction in ED MCA FV (14 cm/sec) with a parallel increase in PI (1.9) and RI (0.9), findings that are consistent with severe intracranial hypertension (ICP=45 mmHg). Velocity scale (m/sec) is displayed on the right of the screen (see text for details).



of Neurology, published in 2004 and concerning transcranial Doppler Ultrasonography, TCD is of established value in the detection and monitoring of angiographic vasospasm after spontaneous SAH (incidence of 21-70%)[23].

Figure 8: TCCS ultrasonography with spectral waveforms obtained from the right MCA. The TCD examination was performed one day before undertaking the 1st diagnostic tests for BD (2 days after performance of the examination being displayed in figure 7) and shows systolic peaks of approximately 60 cm/sec with diastolic flow reversal (oscillating blood flow), whereas cerebral perfusion pressure (CPP=mean arterial pressure-ICP) was 20 mmHg. Velocity scale (m/sec) is displayed on the right of the screen (see text for details).



Vasospasm after SAH causes increased FV inside intracranial vessels. This can be detected by TCD imaging, indicating the need for treatment before the onset of ischemia. The diagnosis of spasm with a TCD device is based on the hemodynamic principle that the velocity of blood flow in an artery is inversely related to the area of the lumen of that artery[24]. Usually a bedside TCD is performed at day 0 and subsequently during days 3 to 10. A mean FV of 120 cm/sec is considered a sign of mild spasm whereas a value above 180 cm/sec indicates severe spasm. In general, MFV < 120 cm/sec correlate with mild angiographic VSP (<25% diameter narrowing), MFV of 120-200 cm/sec correlate with moderate angiographic VSP (<25-50% diameter narrowing) and MFV >200 cm/sec imply severe angiographic VSP (>50% diameter narrowing)[25,26]. Nevertheless, different studies suggest that these observations are imperfect as grade IV and V Hunt-Hess patients (Table 2)[27] tend to have the lowest flow velocities. These discrepant results might reflect treatment effect with nimodipine, presence of adequate collateral pathways and lack of clear criteria for defining VSP by angiography or TCD. In addition, important VSP may have occurred in vessel segments imperfectly insonated. Furthermore, TCD findings are influenced by different physiologic and pathologic factors and by vasoactive medications. Increased age, ICP, central venous pressure (CVP), cardiac output, blood viscosity and vasoconstrictive drugs are associated with decreased FV, whereas anemia, drugs with vasodilatation properties and hypercarbia cause increase in FV[28,29]. It is important to point out that TCD MF Vs do not allow calculation of cerebral blood flow volume and cannot be substituted for direct CBF measurements. The information provided by TCD provides prediction of the degree of vessel narrowing, spasm progression or regression and compensatory vasodilation.

Patients with cerebral VSP are often treated with medical therapies designed to increase CBF, such as volume loading and induced hypertension. However, SAH is associated with impaired autoregulation, meaning that these measures may increase TCD velocities (flow changes parallel changes in mean blood pressure), thus complicating their interpretation. For that reason many researchers have recommended that TCD velocities be evaluated before and after medical maneuvers in order to assess

Table 2: Hunt-Hess Grading Scale of Subarachnoid Hemorrhage

Grade I	Asymptomatic or minimal headache and slight nuchal rigidity
Grade II	Moderate to severe headache, nuchal rigidity, no neurologic deficit other than cranial nerve palsy
Grade III	Drowsiness, confusion, or mild focal deficit
Grade IV	Stupor, moderate-to-severe hemiparesis, possibly early decerebrate rigidity and vegetative disturbances
Grade V	Deep coma, decerebrate rigidity, moribund appearance

the influence of these changes[30,31].

Table 3: TCD's mean flow velocity criteria for cerebral vasospasm

Severity of vasospasm	Mean flow velocity (cm/sec)	MCA/ICA ratio (Lindegaard ratio)
Normal	< 85	< 3
Mild	< 120	< 3
Moderate	120-150	3 to 5.9
Severe	151-200	> 6
Critical	> 200	> 6

Increased FV may be caused from generalized hyperaemia instead for true VSP. The Lindegaard ratio (LR) can differentiate those two conditions, increasing TCD accuracy in the diagnosis of cerebral VSP (Table 3)[19]. The LR is the ratio between the MCA or ACA and ipsilateral internal carotid artery (ICA) mean flow velocity (MFV_{MCA}/MVF_{ICA} or MFV_{ACA}/MFV_{ICA}) and it changes very little when MCA FVs and CBF increase as a result of a hyperemic stimulus, such as hypercarbia. A MFV_{MCA}/MVF_{ICA} ratio of less than 3 is rarely found in patients with VSP, whereas a ratio above 6 appears to distinguish severe from moderate MCA vasospasm[7,25,26].

TCD measurements may be predictive of outcome after SAH. An average rise in flow velocities of more than 20 cm/sec per day between days 3 and 7 after SAH, a rapid early rise in FV (>25%/day) and a high Lindegaard ratio have been found in patients with SAH who develop a delayed ischemic neurologic deficit. An increase in MCA FV of greater than 50 cm/sec predicts the presence of more than 20% angiographic vessel narrowing. A PS FV in the range of 270-302 cm/sec has been associated with clinical MCA VSP, whereas an increased PI indicates poor prognosis[32].

2. Clinical application of TCD in the confirmation of brain death

Descriptions of TCD findings in BD first appeared in the literature in the mid-1980s [33,34]. The TCD flow profile in BD has been shown to change with cardiac output, varying from sharp and pulsatile to damped and sluggish. BD has also been associated with an oscillating pattern of FV, correlated with a CPP below 20 mmHg and reversal of intracranial diastolic blood flow, due to increased ICP. There are 5 patterns of FV seen with BD[35-37]:

1. *Low diastolic flow.* Increased ICP and preserved CBF, both being reversible, can be associated with low ED MCA FV due to the fact that augmented ICP approaches diastolic blood pressure. In such cases, there is a rapid increase in systolic FV, followed by a sharp decrease in diastolic FV to near zero.
2. *Systolic peaks.* No diastolic flow is present, whereas only systolic peaks of FV are detected. This pattern can also be reversible.
3. *Oscillating blood flow.* Anterograde short systolic peaks alternate with retrograde diastolic blood flow of brief duration and sharp morphology.
4. *Short systolic spikes.* In cases of absence of cerebral blood flow there are only anterograde systolic spikes that last briefly (contrary to systolic peaks that last longer).
5. *No TCD signal.* In cases of angiographic absence of flow in all vessels there is intracranial circulatory arrest. Attention is needed for cases with thickened skull with inadequate 'acoustic windows'.

The first 2-to-3 patterns of TCD findings in BD accompany nearly the majority of cases with increased ICP, associated with an increased PI (>1.2) and RI (>0.8). Distal cerebrovascular resistance is very high in cases of severe intracranial hypertension where blood flow compromise progresses from a distal to a proximal direction. In conclusion, basal arteries remain patent, allowing for blood flow detection with TCD, whereas peripheral capillaries are the major site of flow obstruction.

Despite high specificity (100%) and sensitivity (91.3%), false positive results can occur. For that reason, the entire intracranial circulation must be examined, with both the anterior and vertebrobasilar system evaluated through TCD examination. There is also need for insonating arteries from both haemispheres[34].

Finally, the reliability of TCD confirmation of BD depends on the examiner's experience and skill and must be interpreted with caution,

exclusively in patients who have already met the clinical criteria for brain death.

Conclusions

TCD appears to be a practical and noninvasive method for assessment of circulatory derangements of the cerebral circulation. When TCD is applied carefully to study the entire intracranial circulation in neurocritically ill patients and particularly in those suffering from SAH, it reveals a great deal of information that is valuable to the caring physician. The limitations of TCD monitoring demonstrate that the results must be interpreted cautiously and always in relation with clinical and neuroimaging data. TCD examination can also be a useful confirmatory test in patients who have already met the clinical criteria of BD. Nevertheless, further studies are required for the evaluation of TCD examination as a long-term prognostic tool in other selected cases or for the establishment of TCD measurements as end points in different clinical trials assessing new therapeutic regimens for management of neurocritically ill patients.

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Abbreviations

ACA = Anterior cerebral artery
BA = Basilar artery
BD = Brain death
CBF = Cerebral blood flow
CNS = Central nervous system
CPP = Cerebral perfusion pressure
CSF = Cerebrospinal fluid
CT =Computed tomography
CVP = Central venous pressure
CW = Continuous wave
ED = End diastolic
FV = Flow velocity
GCS = Glasgow Coma Scale
ICA = Internal carotid artery
ICP = Intracranial pressure
ICU = Intensive Care Unit
LR = Lindegaard ratio
MCA = Middle cerebral artery
OA = Ophthalmic artery
PCA = Posterior cerebral artery
PI = Pulsatility index
PS = Peak systolic
PW = Pulse wave
RI = Resistance index
SAH = Subarachnoid hemorrhage
TCCS=Transcranial color-coded ultrasonography
TCD = Transcranial Doppler
TICA = Terminal portion of the internal carotid artery
VA = Vertebral artery
VSP = Vasospasm

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