Austin Neurosurgery: Open Access

Open Access

Austin Publishing Group

Special Article - Neurocritical Care Update

Transcranial Doppler Ultrasound as a Non-Invasive Intracranial Pressure Marker? Myth or Reality?

Alexander R*
Sentient Neurocare Services Inc., USA

***Corresponding author:** Razumovsky Alexander, Sentient Neurocare Services Inc., 11011 McCormick Rd, Suite 200, Hunt Valley, MD, USA

Received: June 14, 2016; **Accepted:** July 01, 2016; **Published:** July 05, 2016

Abstract

Monitoring the brain after acute injury is central to the practice of neurocritical care for patients with a wide range of disorders, including subarachnoid hemorrhage, traumatic brain injury, ischemic and hemorrhagic strokes, infectious disease of central nervous system as well status epilepticus and encephalopathy's of different origin. Developed over 30 years ago, Transcranial Doppler (TCD) and later Transcranial Color-Coded Duplex (TCCD) ultrasonography has now become part of the standard care in the Neuro-Critical Care Unit (NCCU). TCD and TCCD are of critical value in managing patients with cerebrovascular disease, sickle cell disease, thrombo-embolic occlusion of a large conductance vessel, cerebral vasospasm of any etiology, non-invasive assessment of intracranial pressure and have established value for the supportive diagnosis of brain death. Today, TCD and TCCD are the most innovative techniques for investigation of intracranial circulation and are important non-invasive modalities that can provide information about Cerebral Blood Flow Velocity (CBFV) and Intracranial Hypertension (ICH). This review article has addressed the rationale and clinical value of TCD in the NCCU for ICH. Although much of the data collected today is descriptive, TCD offers potential application to guide therapy and predict outcome in patients with ICH.

Keywords: Transcranial doppler ultrasound; Intracranial pressure; Neurocritical care

Introduction

In the contemporary neurointensive care of patients with acute stroke, Subarachnoid Hemorrhage (SAH), traumatic Brain Injury (TBI) and other illnesses where cerebral hemodynamics can be disturbed or impaired basic neurological monitoring should be expanded by extended multi-modality neuromonitoring including Transcranial Doppler (TCD) ultrasonography [1]. Growing evidence clearly supports the integration of extended neuromonitoring to unmask otherwise occult alterations and to differentially adapt the type, extent, and duration of therapeutic interventions. By expanding our knowledge and experience, the integration of TCD in the daily clinical routine in the Neuro-Intensive Care Unit (NICU) will provide us with the means to improve outcome, which has not been possible by relying only on neurological examination alone as practiced in the past. This review will describe specific advanced clinical application of TCD for the diagnosis and monitoring of patients with Intracranial Hypertension (ICH).

TCD and its Role for Evaluation of Intracranial Hypertension

TCD was introduced into the practice of medicine in 1986 and has been used extensively in a variety of in- and out-patient settings [2]. TCD ultrasonography utilizes a hand-held 2 MHz transducer that is placed on the surface of the scalp to measure the Cerebral Blood Flow Velocity (CBFV) and Pulsatility Index (PI) within the intracranial arteries in the circle of Willis. Due to its noninvasiveness and easy applications, TCD examinations have gained an important role in the very early phase, as well during the repetitive assessment of patients with acute stroke, to diagnose and monitor vasospasm for patients after SAH of a different nature (aneurysm rupture, tumor resection, TBI) [3-10]. A recent prospective observational study across 17 sites showed that TCD measurements upon admission may provide additional information about neurologic outcome after mild to moderate TBI [11]. Other important clinical TCD applications include emboli monitoring and management of patients with sickle-cell disease [12-14].

Raised Intracranial Pressure (ICP) is a life-threatening condition that can result in brainstem compression and compromised brain circulation [15-17]. Increased ICP is the most common cause of death in patients with severe TBI. However, even in patients initially diagnosed with mild or moderate TBI, slow growth of a hematoma with consequent development of ICH will adversely affect outcome [18]. Increased ICP is associated with increased morbidity and is an independent predictor of mortality and outcome in TBI patients [19,20] or in patients after SAH [21]. Therefore, ICP monitoring is a reasonable approach to discovering a progressive increase in ICP in patients, including patients with TBI [22]. Today, the ventricular catheter connected to an external strain gauge transducer is considered the most accurate, low-cost and reliable method for monitoring ICP and it is the established reference standard for measuring ICP [23-25]. However, there are few factors limiting direct ICP measurements, one is a requirement for neurosurgical expertise and this expertise might not be available where or when treatment decisions need to be made, and risk of tissue damage and infection, particularly for mild and moderate TBI. In real life, ICP is monitored in only a small fraction of patients that could benefit from

Citation: Alexander R. Transcranial Doppler Ultrasound as a Non-Invasive Intracranial Pressure Marker? Myth or Reality?. Austin Neurosurg Open Access. 2016; 3(1): 1047.

ICP measurement [25]. Non-invasive ICP monitoring would enable triage at the point of contact (battlefield, sport arena, ambulance, emergency room, out-patient clinic). It could help titrate therapy to ICP targets, and can provide long-term monitoring to evaluate effectiveness of treatment. Therefore, any non-invasive method that can provide information about ICP will be very beneficial. The key to successful TCD utilization for its application to make judgment about ICP is recognition of the simple fact that unless the right questions are asked, it will never contribute towards a sustained reduction in the risk of inappropriate utilization of TCD. The primary purpose of TCD ultrasonography is to determine the CBFV of flowing blood by quantitative interpretation of TCD waveforms. Although the qualitative contour of the TCD waveform during ICP elevation falls into a recognizable pattern, the interpretation depends on the experience and expertise of the TCD examiner and interpreter. Objective, reproducible, and verifiable measures of TCD waveform changes are necessary for TCD findings to be used with certainty for evaluation of ICH. One method of quantifying these changes is utilization of the PI which is a reflection of downstream resistance and will be affected by distal focal stenosis, ICP and/or diffuse atherosclerosis [26]. The pulsatility of the waveform reflects the amount of resistance in the more distal cerebral blood vessels and PI is a calculated index of the TCD waveform and takes into account the peak systolic CBFV (pCBFV) and the end-diastolic CBFV (edCBFV) and compares the changes in these variables against the change in the standard measure of the entire waveform, such as mean CBFV [26]. When ICP is above 15-20 mm Hg, the PI has been evaluated as an alternative to direct ICP measurement [18,27,28]. There is also a significant correlation between the CPP and PI [29]. In a prospective study, it was shown that TCD is valid in predicting the patient's outcome of 6 months, and correlates significantly with ICP and CPP values when it is performed within the first 24 hours after severe TBI [30]. The high sensitivity of admission TCD to predict ICH and abnormal CPP after severe TBI in children demonstrates that TCD is an excellent first-line examination to determine those who need urgent aggressive treatment and continuous invasive ICP monitoring [31,32]. In those children with TBI who initially do not meet clear criteria for invasive ICP monitoring but who are at risk for development of ICH, TCD may be used as a noninvasive tool to screen for the development of elevated ICP in the first 24 hours following severe TBI [31]. Prospective, observational cohort study enrolled 365 patients with mild and moderate TBI and an initial GCS score of 9 to 15, who's initial CT scan showed either absent or mild lesions. TCD measurements of bilateral MCA's were obtained on admission in the emergency room under stable conditions. Results demonstrated that in patients with no severe brain lesions on CT, a TCD on admission, complemented with brain CT scan, could accurately screen patients at risk for secondary neurological complications [11]. Another recent pilot study suggests that in adult patients with severe TBI, TCD could be used in pre-hospital care to detect patients whose CPP may be impaired [33]. In the ICU setting, serial TCD monitoring allowed identification of an imminently fatal complication in time to allow a life-saving intervention [34,35].

TCD is the non-invasive ultrasound modality capable of identifying patients who are progressing to ICH, and it can also monitor the effectiveness of any pharmacological intervention, and detect normalization of the ICP and PI. However, three conditions must be fulfilled: mean arterial pressure, carbon dioxide tension, and cardiac output must be within normal limits and not significantly different compared to the previous day. Several publications indicate the clinical value of TCD for measuring the MCA CBFV and PI as possible predictors of outcome in severe TBI management [10,36-39]. Some authors suggest that early use of PI measurement permits identification of patients with low CPP and high risk of cerebral ischemia, and in emergency situations PI can be used alone, when ICP monitoring is contraindicated or not readily available [40].

TCD can be used to evaluate ICP, either independent of or in conjunction with other invasive and non-invasive imaging studies. To the best of our knowledge, nobody yet suggests use of PI as an accurate method to quantitatively express ICP in mm Hg. Nevertheless, in numerous publications it was shown that PI correlates well with ICP as measured by invasive methods and support use of TCD as a predictor of elevated ICP and showed positive linear correlation between PI and ICP [28,36,41,42].

At present the role of TCD for the detection of high ICP and low CPP due to the presence of ICH could be suggested as:

1) TCD wave-form changes indicates abnormally high ICP, especially above 20 to 30 mm Hg (PI must be 1.2 or higher and edCBFV 25 cm/sec or lower for all anterior circulation vessels uni- or bilaterally);

2) TCD changes may alert Neuro-ICU personnel and may indicate malfunctioning of ICP probe;

3) Abnormally globally decreased pattern of the CBFV's in parallel with increased PI's indicates onset of diffuse ICH;

4) Sudden onset of asymmetrical CBFV's and PI's changes may indicate a potential mid-line shift.

However, we would like to stress that at present TCD alone cannot substitute invasive and quantitative ICP monitoring modalities. Nevertheless, in cases when invasive ICP measurements are unobtainable TCD can be used to evaluate ICP either independent of or in conjunction with other non-invasive imaging studies [43,44].

Conclusion

The strong correlation observed between ICP and PI through the management period of patients with suspected ICH can lead us to use TCD ultrasonography-derived PI as a guide if invasive ICP monitoring is not available [11,28,36,41-45]. At the same time, some authors argue that PI is not a reliable predictor of ICP [46,47]. Therefore, more studies are needed before we can substitute direct measurement of ICP with TCD. In 2012 Kashif et al. presented a retrospective proof-of-concept, model based approach to continuous estimation and tracking of ICP by use of time synchronized, minimally invasive measurements of MAP and CBFV by TCD [48]. We completely agree with Kristiansson et al's. opinion - that TCD is the most promising non-invasive techniques for quantitative ICP evaluation, especially in the emergency care or NICU settings [49]. It is clear the quest for "fine tuning" of this TCD application is still not over but even today quantitative and qualitative changes in TCD measured CBFV's and PI's values and wave-form morphologies may persuade physicians

to undertake other diagnostic steps or to change medical treatment, thereby improving care of patients and their outcomes. To conclude, TCD can be used by experienced examiners to evaluate the presence of high ICP/low CPP and TCD as a non-invasive and simple procedure could be engaged in the daily management of patients when ICH is suspected and/or must be confirmed; although more studies are needed before we can substitute direct measurement of the ICP with TCD.

References

1. Le Roux P, Menon DK, Citerio G, Vespa P, Bader MK, Brophy G, et al. The International Multidisciplinary Consensus Conference on Multimodality Monitoring in Neurocritical Care: Evidentiary Tables. A Statement for Healthcare Professionals from the Neurocritical Care Society and the European Society of Intensive Care Medicine. Neurocritical Care. 2014; 21 Suppl 2: 297-361.
2. Aaslid R, Markwalder TM, Nornes H. Noninvasive transcranial Doppler ultrasound recording of flow velocity in basal cerebral arteries. J Neurosurg. 1982; 57: 769-774.
3. Razumovsky AY, Gillard JH, Bryan RN, Hanley DF, Oppenheimer SM. TCD, MRA and MRI in acute cerebral ischemia. Acta Neurol Scand. 1999; 99: 65-76.
4. Sloan MA, Alexandrov AV, Tegeler CH, Spencer MP, Caplan LR, Feldmann E, et al. Assessment: transcranial Doppler ultrasonography: report of the Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology. Neurology. 2004; 62: 1468-1481.
5. Demchuk AM, Burgin WS, Christou I, Felberg RA, Barber PA, Hill MD, et al. Thrombolysis in brain ischemia (TIBI) transcranial Doppler flow grades predict clinical severity, early recovery, and mortality in patients treated with intravenous tissue plasminogen activator. Stroke. 2001; 32: 89-93.
6. Washington CW, Zipfel GJ and the Participants in the International Multi-disciplinary Consensus Conference on the Critical Care Management of Subarachnoid Hemorrhage. Detection and Monitoring of vasospasm and Delayed Cerebral Ischemia: A Review and Assessment of the Literature. Neurocrit Care. 2011; 15: 312-317.
7. Kumar G, Shahripour RB, Harrigan MR. Vasospasm on transcranial Doppler is predictive of delayed cerebral ischemia in aneurysmal subarachnoid hemorrhage: a systematic review and meta-analysis. J Neurosurg. 2016; 124: 1257-1264.
8. Ricarte IF, Funchal BF, Miranda Alves MA, Gomes DL, Valiente RA, Carvalho FA, et al. Symptomatic Cerebral Vasospasm and Delayed Cerebral Ischemia Following Transsphenoidal Resection of a Craniopharyngioma. J Stroke Cerebrovasc Dis. 2015; 24: e271-273.
9. Martin NA, Patwardhan RV, Alexander MJ, Africk CZ, Lee JH, Shalmon E, et al. Characterization of cerebral hemodynamic phases following severe head trauma: hypoperfusion, hyperemia, and vasospasm. J Neurosurg. 1997; 87: 9-19.
10. Ziegler D, Cravens G, Poche G, Gandhi R, Tellez M. Use of Transcranial Doppler in Patients with Severe Traumatic Brain Injuries. J Neurotrauma. 2016.
11. Bouzat P, Almeras L, Manhes P, Sanders L, Levrat A, David JS, et al. Transcranial Doppler to Predict Neurologic Outcome after Mild to Moderate Traumatic Brain Injury. Anesthesiology. 2016.
12. Markus HS, King A, Shipley M, Topakian R, Cullinane M, Reihill S, et al. Asymptomatic embolisation for prediction of stroke in the Asymptomatic Carotid Emboli Study (ACES): a prospective observational study. Lancet Neurol. 2010; 9: 663-671.
13. Naylor AR, Sayers RD, McCarthy MJ, Bown MJ, Nasim A, Dennis MJ, et al. Closing the loop: a 21-year audit of strategies for preventing stroke and death following carotid endarterectomy. Eur J Vasc Endovasc Surg. 2013; 46: 161-170.
14. Lee MT, Piomelli S, Granger S, Miller ST, Harkness S, Brambilla DJ, et al. Stroke Prevention Trial in Sickle Cell Anemia (STOP): extended follow-up and final results. Blood. 2006; 108: 847-852.
15. Kahraman S, Dutton RP, Hu P, Xiao Y, Aarabi B, Stein DM, et al. Automated measurement of "pressure times time dose" of intracranial hypertension best predicts outcome after severe traumatic brain injury. J Trauma. 2010; 69: 110-118.
16. Alali AS, Fowler RA, Mainprize TG, Scales DC, Kiss A, de Mestral C, et al. Intracranial pressure monitoring in severe traumatic brain injury: results from the American College of Surgeons Trauma Quality Improvement Program. J Neurotrauma. 2013; 30: 1737-1746.
17. Badri S, Chen J, Barber J, Temkin NR, Dikmen SS, Chesnut RM, et al. Mortality and long-term functional outcome associated with intracranial pressure after traumatic brain injury. Intensive Care Med. 2012; 38: 1800-1809.
18. Bouzat P, Francony G, Declety P, Genty C, Kaddour A, Bessou P, et al. Transcranial Doppler to screen on admission patients with mild to moderate traumatic brain injury. Neurosurgery. 2011; 68: 1603-1609.
19. Kawoos U, McCarron RM, Auker CR, Chavko M. Advances in Intracranial Pressure Monitoring and Its Significance in Managing Traumatic Brain Injury. Int J Mol Sci. 2015; 16: 28979-28997.
20. Talving P, Karamanos E, Teixeira PG, Skiada D, Lam L, Belzberg H, et al. Intracranial pressure monitoring in severe head injury: compliance with Brain Trauma Foundation guidelines and effect on outcomes: a prospective study. J Neurosurg. 2013; 119: 1248-1254.
21. Magni F, Pozzi M, Rota M, Vargiolu A, Citerio G. High-Resolution Intracranial Pressure Burden and Outcome in Subarachnoid Hemorrhage. Stroke. 2015; 46: 2464-2469.
22. Bor-Seng-Shu E, Hirsch R, Teixeira MJ, De Andrade AF, Marino R Jr. Cerebral hemodynamic changes gauged bytranscranial Doppler ultrasonography in patients with posttraumaticbrain swelling treated by surgical decompression. J. Neurosurg. 2006; 104: 93-100.
23. The Brain Trauma Foundation. The American Association of Neurological Surgeons. The Joint Section on Neurotrauma and Critical Care. Recommendations for intracranial pressure monitoring technology. J Neurotrauma. 2000; 17: 497-506.
24. Bratton SL, Chestnut RM, Ghajar J, McConnell Hammond FF, Harris OA, Hartl R, et al. Guidelines for the management of severe traumatic brain injury. VII. Intracranial pressure monitoring technology. J Neurotrauma. 2007; 24 Suppl 1: S45-54.
25. Popovic D, Khoo M, Lee S. Noninvasive Monitoring of Intracranial Pressure Biomedical Engineering. 2009; 2: 165-179.
26. Gosling RG, King DH. Arterial assessment by Doppler-shift ultrasound. Proc R Soc Med. 1974; 67: 447-449.
27. Ursino M, Giulioni M, Lodi CA. Relationships among cerebral perfusion pressure, autoregulation, and transcranial Doppler waveform: a modeling study. J Neurosurg. 1998; 89: 255-266.
28. Wakerley BR, Kusuma Y, Yeo LL, Liang S, Kumar K, Sharma AK, et al. Usefulness of transcranial Doppler-derived cerebral hemodynamic parameters in the noninvasive assessment of intracranial pressure. J Neuroimaging. 2015; 25: 111-116.
29. Zweifel C, Czosnyka M, Carrera E, de Riva N, Pickard JD, Smielewski P. Reliability of the blood flow velocity pulsatility index for assessment of intracranial and cerebral perfusion pressures in head-injured patients. Neurosurgery. 2012; 71: 853-861.
30. Tan H, Feng H, Gao L, Huang G, Liao X. Outcome prediction in severe traumatic brain injury with transcranial Doppler ultrasonography. Chin J Traumatol. 2001; 4: 156-160.
31. Melo JR, Di Rocco F, Blanot S, Cuttaree H, Sainte-Rose C, Oliveira-Filho J, et al. Transcranial Doppler can predict intracranial hypertension in children with severe traumatic brain injuries. Childs Nerv Syst. 2011; 27: 979-984.
32. O'Brien NF, Maa T, Reuter-Rice K. Noninvasive screening for intracranial

hypertension in children with acute, severe traumatic brain injury. J Neurosurg Pediatr. 2015; 16: 420-425.

33. Tazarourte K, Atchabahian A, Tourtier JP, David JS, Ract C, Savary D, et al. Pre-hospital transcranial Doppler in severe traumatic brain injury: a pilot study. Acta Anaesthesiol Scand. 2011; 55: 422-428.

34. Reinhard M, Petrick M, Steinfurth G, Ziyeh S, Hetzel A. Acute increase in intracranial pressure revealed by transcranial Doppler sonography. J Clin Ultrasound. 2003; 31: 324-327.

35. Mayans DR, Meads DB, Reynolds PS. Transcranial Doppler identifies a malfunctioning extraventricular drain. J Neuroimaging. 2014; 24: 518-519.

36. 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.

37. Murillo-Cabezas F, Arteta-Arteta D, Flores-Cordero JM, Muñoz-Sánchez MA, Rincón-Ferrari MD, Rivera-Fernández MV, et al. The usefulness of transcranial Doppler ultrasonography in the early phase of head injury. Neurocirugia (Astur). 2002; 13: 196-208.

38. Lewis S, Wong M, Myburgh J, Reilly P. Determining cerebral perfusion pressure thresholds in severe head trauma. Acta Neurochir Suppl. 1998; 71: 174-176.

39. Cardim D, Robba C, Bohdanowicz M, Donnelly J, Cabella B, Liu X, et al. Non-invasive Monitoring of Intracranial Pressure Using Transcranial Doppler Ultrasonography: Is It Possible? Neurocrit Care. 2016.

40. Voulgaris SG, Partheni M, Kaliora H, Haftouras N, Pessach IS, Polyzoidis KS. Early cerebral monitoring using the transcranial Doppler pulsatility index in patients with severe brain trauma. Med Sci Monit. 2005; 11: CR49-52.

41. Moppett IK, Mahajan RP. Transcranial Doppler ultrasonography in anaesthesia and intensive care. Br J Anaesth. 2004; 93: 710-724.

42. Moreno JA, Mesalles E, Gener J, Tomasa A, Ley A, Roca J, et al. Evaluating the outcome of severe head injury with transcranial Doppler ultrasonography. Neurosurg. 2000; 8: e8.

43. Robba C, Bacigaluppi S, Cardim D, Donnelly J, Bertuccio A, Czosnyka M. Non-invasive assessment of intracranial pressure. Acta Neurol Scand. 2016; 134: 4-21.

44. Pappu S, Lerma J, Khraishi T. Brain CT to Assess Intracranial Pressure in Patients with Traumatic Brain Injury. J Neuroimaging. 2016; 26: 37-40.

45. Gura M, Elmaci I, Sari R, Coskun N. Correlation of pulsatility index with intracranial pressure in traumatic brain injury. Turk Neurosurg. 2011; 21: 210-215.

46. Figaji AA, Zwane E, Fieggen AG, Siesjo P, Peter JC. Transcranial Doppler pulsatility index is not a reliable indicator of intracranial pressure in children with severe traumatic brain injury. Surg Neurol. 2009; 72: 389-394.

47. Behrens A, Lenfeldt N, Ambarki K, Malm J, Eklund A, Koskinen LO. Transcranial Doppler pulsatility index: not an accurate method to assess intracranial pressure. Neurosurgery. 2010; 66: 1050-1057.

48. Kashif FM, Verghese GC, Novak V, Czosnyka M, Heldt T. Model-Based Noninvasive Estimation of Intracranial Pressure from Cerebral Blood Flow Velocity and Arterial Pressure. Sci Transl Med. 2012; 4: 129ra44.

49. Kristiansson H, Nissborg E, Bartek Jr J, Andresen M, Reinstrup P, Romner B. Measuring Elevated Intracranial Pressure through Noninvasive Methods: A Review of the Literature. J Neurosurg Anesthesiol. 2013; 25: 372-385.

Austin Neurosurg Open Access - Volume 3 Issue 1 - 2016
Submit your Manuscript | www.austinpublishinggroup.com

Citation: Alexander R. Transcranial Doppler Ultrasound as a Non-Invasive Intracranial Pressure Marker? Myth or Reality?. Austin Neurosurg Open Access. 2016; 3(1): 1047.