경두개 초음파 검사: 신경과 영역에서의 임상 이용

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Transcranial Doppler Ultrasound: Clinical Applications in Neurology

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Transcranial Doppler (TCD) is a noninvasive ultrasound study used to measure cerebral blood flow (CBF) velocity in the major intracranial arteries. It is relatively inexpensive, repeatable, and portable. TCD has been used for the evaluation of cerebrovascular disease for over a decade and provided useful information on intracranial steno-occlusive disease. It also has been used; 1) detection of microembolic signals, 2) quantification of right-to-left shunts, 3) diagnosis of subclavian steal syndrome, 4) assessment of cerebral vasomotor reactivity, CBF and intracranial pressure, 5) monitoring in patients with recurrent syncope, 6) intraoperative monitoring of carotid endarterectomy, 7) detection of vasospasm following subarachnoid hemorrhage and so on. There are several settings in which TCD has well established usefulness and some of the applications of TCD currently lack definitive evidence to the value of TCD is certainly to be expected from ongoing studies. This article aims to provide a basic understanding about the current use of TCD in clinical practice.

Key Words: Transcranial Doppler, Stroke, Clinical trials

Introduction

Transcranial Doppler ultrasonography (TCD) is introduced by Aaslid et al to measure blood flow velocity in the cerebral arteries.[1] In recent years, TCD has become a general practice in the diagnostic and monitoring study of cerebral hemodynamics. This is due to the fact that it is a non-invasive, non-ionizing, portable, dynamic and safe technique for assessment of intracranial circulation. TCD has been used for the evaluation of cerebrovascular disease for over a decade and has provided useful information on intracranial steno-occlusive disease. It also has been used; 1) detection of microembolic signals, 2) quantification of right-to-left shunts, 3) diagnosis of subclavian steal syndrome, 4) assessment of cerebral vasomotor reactivity (VMR), cerebral blood flow (CBF) and intracranial pressure (ICP), 5) monitoring in patients with recurrent syncope, 6) intraoperative monitoring of carotid Endarterectomy (CEA), 7) detection of vasospasm following subarachnoid hemorrhage and so on.

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1. Monitoring of transient microembolic signals

Gaseous or solid microemboli within the middle cerebral artery (MCA) can be detected by TCD as high intensity transient signals (HITS), also called microembolic signals (MES). They are characterized by: (1) having a duration ,300 ms; (2) an amplitude that is 3 dB higher than the background blood flow signal; (3) are typically unidirectional and occur randomly within the cardiac cycle; and (4) produce a characteristic sound like a "moan" or "chirp" on audio signal.[2] Regarding characterization of the nature of emboli, it has been shown that gaseous emboli have signals of higher amplitude and intensity compared to formed solid particles.[3] MES correlate with prior ischemic events and may represent a higher risk for future recurrence ischaemic events. Higher prevalence rates of MES have been reported in strokes caused by large vessel disease and in cardioembolic strokes as compared to lacunar strokes.[4] MES arising from cardiac sources have higher total signal power, longer signal duration, and higher percentage of signals occurring in diastole as compared to those seen in carotid artery disease.[5]

2. Diagnosis of right to left shunts

Transesophageal echocardiography (TEE) with a contrast agent was the way of diagnosing right-to-left intracardiac shunts. TCD with a gaseous contrast medium is now used for the diagnosis of right-to-left shunts. When a gaseous contrast agent is injected into the peripheral vein of a patient with shunts, microbubbles pass from the right to the left circulation during cardiac cycle, and enter the systemic circulation; TCD picks up the microbubbles as microembolic signals in the MCA. Two main contrast agents are in use: agitated saline containing tiny air bubbles, and a galactose based agent. Compared to TEE, TCD has a sensitivity of 95% and a specificity of 75% in detecting PFO.[6] TCD is a more sensitive tool than TEE for the detection of right-to-left shunt, and a shunt at non-cardiac level can be detected by this method. It can be repeated and can provide functional information on the quantification of the shunt which can be helpful in assessing the severity of the risk for stroke in patients with PFO.[7]

3. Diagnosis of subclavian steal syndrome

TCD can be used in conjunction with duplex vertebral sonography in the diagnosis of subclavian steal syndrome. Incomplete steal causes a decrease in systolic blood flow velocity, and in severe cases alternating directions of blood flow occurs in the vertebral artery on the side of the subclavian steal. Complete flow reversal is noted in the vertebral artery in cases of complete steal.[8] TCD findings may be potentiated by exercise of the arm on the affected side.

4-1. Assessment of cerebral vasomotor reactivity

This phenomenon is known as cerebrovascular autoregulation. The ability of the cerebral vascular system to constrict and dilate in response to changes in perfusion pressure is termed autoregulation. Decreased cerebral vasomotor reactivity (VMR) indicates the presence of preexisting vasodilatation, which reflects a reduced capacity of cerebral resistance vessels to adapt their caliber in response to changes in cerebral perfusion. TCD has been used extensively in the study of VMR. A variety of tests were introduced to evaluate intracranial hemodynamics using the phenomenon of VMR, including CO2 reactivity with TCD, acetazolamide testing with TCD, CBF scanning techniques, and the breath-holding index (BHI).[9] The latter is the simplest way of challenging VMR if a patient is compliant and capable of holding breath for 30 seconds. This index is calculated using the mean flow velocities (MFVs) obtained by TCD before breath holding and at the end of 4 seconds of breathing after 30 seconds of breath holding. The patient should be able to hold breath voluntarily for at least 24 seconds, preferably 30 seconds. BHI values of less than 0.69 are predictive of risk for stroke in patients who have asymptomatic severe ICA stenosis and symptomatic occlusions.[10] Although this test is least quantifiable in terms of achieved carbon dioxide levels and it requires patient cooperation, BHI does not require any gas monitoring equipment or intravenous injections. BHI may represent a screening test in the outpatient clinic to identify patients who have impaired VMR.

4-2. Assessment of cerebral perfusion pressure and cerebral blood flow

Cerebral perfusion pressure (CPP) is the difference between arterial pressure (AP) and the effective downstream pressure (EDP) of the cerebral circulation. Direct measurement of ICP is not always possible when the risk of a reduction in CPP is maximal. In this instance TCD evaluation of MCA velocity has been proposed as an alternative for neurological monitoring. Several methods of estimating CPP using measured TCD velocities have been described but more work is needed as none of the recently described methods has been fully validated.

TCD velocities have been used to provide information about CBF. A number of studies have assessed the validity of comparing flow velocity (FV) with techniques for measuring CBF, including IV xenon, the Kety-Schmidt method, magnetic resonance imaging, single photon emission computed tomography and laser Doppler flowmetry.[11] Whilst the TCD represents a non-invasive method of obtaining information about CBF, the linear relationship between CBF and FV is only present if neither the diameter of the insonated vessel nor the angle of insonation change during the examination.[12]

4-3. Assessment of intracranial pressure

In a pilot study, changes in ICP were compared with the TCD findings of the MCA. Increases in ICP were accompanied by an increase in pulsatility index (PI) (owing to a decrease in diastolic and mean velocities).[13] It is unlikely that TCD will replace invasive ICP monitoring in the near future. However, it may have a role in situations where the role of invasive monitoring is not clearly established: stroke, pediatric patients, liver failure, and minor head injury assessed on accident and emergency wards and preeclampsia.[12]

5. Monitoring in patients with recurrent syncope

Syncope is a transient loss of consciousness with inability to maintain postural tone due to cerebral hypoperfusion. Recovery is typically spontaneous. The most frequent cause of syncope is a dysfunction of the autonomic nervous system (neurocardiogenic syncope).[14] Patients with neurocardiogenic syncope have changes in CBF regulation during the event. TCD monitoring during head upright tilt table testing (HUT) helps to assess these alterations. Since TCD changes precede the fall in blood pressure, heart rate and symptoms, this technique allows early interruption of HUT before symptoms arise, preventing the unpleasant experience for the patients. TCD monitoring also help to differentiate patients with exaggerated response to nitrates from those with true neurocardiogenic vasodepressor syncope. TCD during HUT may improve the well-known usefulness of the method for the diagnosis of neurocardiogenic syncope.[15]

6. Intraoperative monitoring of CEA and cardiac surgery

Thromboembolism, hypoperfusion, and hyperperfusion are the primary causes of stroke during carotid interventions and stroke rates range between 2% and 10%. TCD monitoring of the MCA during surgeries is a valuable tool that quickly alerts clinicians to these complications. Because TCD monitors in real time, clinicians can make procedural changes sooner than with other monitoring modalities. Intraoperative TCD involves monitoring the velocity of flow of blood in the MCA during carotid endarterectomy.[9] It assesses the adequacy of CBF while the carotid artery is cross-clamped during carotid endarterectomy.[16] Ischemia during cross clamping is a classic complication and is considered severe if reduction in flow velocity is less than 85%, mild to moderate if reduction is between 60-85%, and absent if there is less than 60% reduction.[2] Hyperemic phenomena may occur and result in a sudden increase in blood flow velocity in the MCA. Microemboli (MES/HITS) caused by operative maneuvers such as shunt insertion or removal can be documented in real time with TCD as well. In addition to CEA, TCD has been applied to other surgical procedures for intraoperative monitoring. Cardiac surgery with cardiopulmonary bypass has been an area of research with TCD. Use of TCD allows guided management of perfusion pressure to maintain appropriate CBF velocities. TCD does allow detection of cerebral emboli during bypass surgery and the embolic load has been associated with postoperative neuropsychological dysfunction.[2]

7. Detection of vasospasm following subarachnoid hemorrhage

Although in most situations changes in flow velocity as detected by TCD indicate proportional changes in flow, cerebral vasospasm following subarachnoid hemorrhage (SAH) is the exception to this paradigm. The correlation between flow velocity and flow is lost. Cerebral angiography is the gold standard for diagnosing cerebral vasospasm, but TCD has become an important tool to detect its onset, location, severity, and response to therapy.[17]

Lindegaard index,[18] the ratio of the MCA flow velocity to the extracranial ICA flow velocity, was developed to distinguish true vasospasm from hyperdynamic flow. In vasospasm, the flow velocity should be elevated only in the intracranial vessel, and the index will be high, whereas a hyperdynamic state would be expected to increase velocity in all vessels, with little change in the index. A recent evidence-based assessment of the literature on the use of TCD supported its application in monitoring for vasospasm following SAH, particularly for vasospasm in the middle cerebral and basilar arteries.[19] Causes of acute neurological deterioration in patients with SAH other than cerebral vasospasm include further hemorrhage, cerebral edema, hydrocephalus, and even cerebrospinal fluid hypovolemia. Although a computed tomography (CT) scan of the head is essential in the diagnosis, TCD may give clues to the diagnosis of subacute changes, however. Elevated intracranial pressure from cerebral edema or hydrocephalus increases the pulsatility index.[20] TCD may therefore be useful to guide further workup when the diagnosis is unclear.

Conclusion

Although there are several settings in which transcranial Doppler has well established usefulness, TCD has its limitations. One major disadvantage of TCD is that it is highly operator dependent and requires skilled interpretation. A suboptimal interrogation window is also less of a limiting factor. Some of the applications of TCD currently lack definitive evidence to the value of TCD is certainly to be expected from ongoing studies. With rapid advancements being made in technology, newer applications of TCD for quantitative assessment of CBF are observed in the near future.

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