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Noninvasive Multimodality Cerebral Monitoring Modalities in Neurosurgical Critical Care

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Treatment decisions for patients with acute neurologic injury are increasingly guided by multiple monitoring modalities. These modalities together with the usual critical care end points examine intracranial pressure (ICP), cerebral blood flow (CBF), cerebral oxygenation, and cerebral autoregulation. The current gold standard for ICP monitoring, ventriculostomy, is used for transducing pressure and drainage of cerebrospinal fluid. Recent research efforts in neurologic critical care have sought to develop widely available, easy-to-use, noninvasive methods for monitoring ICP changes in patients with brain injuries and other aspects of cerebral autoregulation.

Several techniques use the physiology of the orbit as a surrogate for ICP. Fundoscopic examination has long been used to assess chronic changes in the form of optic disk swelling but is not reliable in acute brain injury or rapid ICP changes. Ultrasound measurement of optic nerve sheath diameter and optical coherence tomography are newer methods that may help assess real-time changes in ICP.[1,2] Optic nerve sheath diameter measurements between 5.0 and 5.9 mm were found to be 90% sensitive (95% confidence interval [CI], 0.80—0.95) and 85% specific (95% CI, 0.73—0.93) for ICP greater than 20 mm Hg in a recent meta-analysis of 6 such studies involving 231 total patients.[3] Pupillometry examines pupillary light reflex pathway, measuring a neurologic pupillary index. Neurologic pupillary index is significantly decreased in patients with ICP greater than 30 mm Hg, but not below.[4] In certain studies, visual evoked potentials have shown correlations between latency and elevated ICP; however, these findings are inconsistent and require further evaluation.[5-7]

Other techniques use the ear and its surroundings for indirect analysis of ICP. Tympanic membrane displacement (TMD) analyzers measure changes in the tympanic membrane kinematic properties to estimate ICP because of the communication between the subarachnoid membrane and cochlear perilymph.[8,9] Although a significant relationship has been demonstrated between TMD and ICP, its clinical feasibility is problematic in achieving an accurate TMD measurement, large standard error, and intersubject variability.[9] Distortion product otoacoustic emissions are produced by sound waves emanating from the cochlea and middle ear after stimulation with set frequencies.[10,11] ICP is transmitted to the stapes via the cochlear aqueduct, and the increased strain applied to the stapes is thought to measurably alter transmission of sound through the middle ear in patients without severe hearing loss.[10,11] Studies have found limited or no change to distortion product otoacoustic emissions magnitude or phase with small changes in ICP, restricting its use to changes in ICP of 15 mm Hg or higher.[12,13]

Ultrasound techniques for exploring the intracranial compartment represent another exciting direction in noninvasive methods for analyzing ICP. Insonation of cerebral arteries using transcranial Doppler (TCD) has been particularly useful in detecting trends in mean flow velocities in patients with subarachnoid hemorrhage[14]; however, more recently, TCDs have been used to assess ICP by comparing flow velocities in the intra- and extracranial portions of the ophthalmic artery.[5,15] By applying progressively greater external pressure to the orbit using specialized goggles, the intra- and extracranial pressure can be matched, therefore estimating ICP.

The pulsatility index generated by TCDs has been shown to be up to 81%—100% sensitive and 82%—97% specific for detecting elevated ICP when a pulsatility index cutoff of between 1.26 and 1.335 is selected.[4,10,16-19] Ultrasonic time-of-flight measures the effects of intracranial media on sound waves of various frequencies as they pass through the skull to a receiever.[15,20] As ICP increases, so does the density of the cranial compartment, causing a measurable change in ultrasonic time-of-flight, primarily through changes in speed of conduction and attenuation of the signal.[20] This method requires further studies to develop clinical utility.

Emerging spectroscopy-based modalities for assessing CBF, oxygenation, perfusion, and metabolism are beginning to shape our critical care management of neurologic injury. The volumetric impedance phase shift spectroscopy (VIPS) detects bioimpedance variations by transmitting an array of varying frequencies of low energy radio waves from each side of the back of the head to a receiver. The VIPS technology is able to identify patients with severe stroke with a sensitivity of 93%, specificity of 92%, and an area under the curve of 0.93 (95% CI, 0.85—0.97).[5] Near-infrared spectroscopy (NIRS) is an optical technology based on differential absorption and scattering of near-infrared spectrum light (650—900 nm) to noninvasively evaluate cerebral oxygenation indices in the brain cortex by differentiating between oxyhemoglobin and deoxyhemoglobin, resulting in a measurement of regional tissue hemoglobin oxygen saturation (rSO_2).[10,15] rSO_2 as measured using NIRS has been shown to correlate well with measurements of CBF using TCD ultrasound.[6,10] Moreover, rSO_2 can be used to derive the total hemoglobin reactivity index, another measurement of cerebral autoregulation.[6,7] NIRS therefore presents a continuous monitoring modality that can identify changes in cerebral autoregulation and perfusion before patients become symptomatic of cerebral ischemia.[21]

Monitoring cerebral metabolism and oxygen consumption in patients with brain injury is useful in screening cerebral ischemia. Jugular venous bulb oximetry uses a flexible catheter that is advanced through the jugular vein into the jugular venous bulb to measure the cranial venous blood rSO_2. By subtracting this value from the arterial rSO_2, the cerebral oxygen extraction percent is estimated.[22] Factors affecting jugular bulb saturation include CBF and cerebral oxygen extraction rate, meaning that this noninvasive method can describe trends in both global cerebral perfusion and metabolic rate.[21,23] However, there are several limitations to jugular bulb oximetry, including catheter malpositioning and injury to the vessel wall.[24]

Electroencephalography (EEG) patterns may facilitate the detection of cerebral hypoperfusion and loss of homeostasis to provide early identification of vasospasm and elevated ICP.[25] As a continuous form of monitoring, EEG can also be used to noninvasively collect information on changes in cerebral autoregulation in certain types of brain injury. Decrease in the alpha-delta ratio was shown to accurately predict delayed cerebral ischemia in patients with subarachnoid hemorrhage.[26-28] Changes in the alpha-delta ratio preceded clinical diagnosis of vasospasm by an average of 7 hours, indicating that continuous EEG monitoring may allow for early treatment of this complication in patients with subarachnoid hemorrhage.[26,27]

In conclusion, noninvasive multimodality monitoring modalities carry the potential to curtail unnecessary use of invasive monitors; however, they remain limited by regional, technical, and/or operator-dependent factors. Although substantial progress has been made, the greatest utility remains adjunctive, and further improvements could optimize the accuracy and efficiency of diagnosis to further decrease our reliance on invasive monitoring methods.

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