The Role of Transcranial Doppler Ultrasonography in the Diagnosis and Management of Vasospasm After Aneurysmal Subarachnoid Hemorrhage

Scott A. Marshall, MD[a,b,*], Paul Nyquist, MD, MPH[a,c], Wendy C. Ziai, MD, MPH[a,c]

KEYWORDS

• Transcranial Doppler • Vasospasm
• Subarachnoid hemorrhage • Lindegaard Index

Aneurysmal subarachnoid hemorrhage and its accompanying sequelae are management challenges for the neurosurgeon and neurointensivist. Transcranial Doppler ultrasonography (TCD) has emerged as a tool used extensively by many centers for the surveillance and monitoring of vasospasm after aneurysmal subarachnoid hemorrhage (SAH).[1] The overall management of the primary and secondary complications of SAH is complex, and the use of appropriate tools and diagnostic strategies is helpful. TCD has emerged as an inexpensive, noninvasive tool used not only for bedside monitoring of intracerebral hemodynamic changes seen with SAH. TCD can also be used to evaluate other neurologic conditions in the Neurosciences Critical Care Unit such as

The views and opinions herein belong solely to the authors. They do not nor should they be construed as belonging to, representative of, or being endorsed by the Uniformed Services University of the Health Sciences, the US Army, The Department of Defense, or any other branch of the federal government of the United States.

[a] Division of Neurosciences Critical Care, Departments of Anesthesiology Critical Care Medicine, Johns Hopkins University School of Medicine, Meyer 8-140, 600 North Wolfe Street, Baltimore, MD 21287, USA
[b] Department of Neurology, Uniformed Services University of the Health Sciences, 4301 Jones Bridge Road, Bethesda, MD 20814, USA
[c] Departments of Neurosurgery and Neurology, Johns Hopkins University School of Medicine, Meyer 8-140, 600 North Wolfe Street, Baltimore, MD 21287, USA
* Corresponding author. Department of Neurology, Uniformed Services University of the Health Sciences, 4301 Jones Bridge Road, Bethesda, MD 20814.
E-mail address: scott.marshall@amedd.army.mil

Neurosurg Clin N Am 21 (2010) 291–303
doi:10.1016/j.nec.2009.10.010
1042-3680/10/$ – see front matter

intra- and extracranial vascular stenosis, arteriovenous malformations, intraoperative emboli, venous sinus thrombosis, ischemic stroke, sickle cell disease, and brain death.[2–4] This article provides a brief review of the pathophysiology of vasospasm, and other devices used to detect vasospasm. Also reviewed are the indices and technical aspects of TCD ultrasonography, the interpretation of data obtained from TCD studies, and TCD-based management algorithms for vasospasm.

VASOSPASM AFTER SUBARACHNOID HEMORRHAGE

The diminution of blood flow transiting through the cerebral vasculature seen after aneurysmal SAH due to vasoconstriction is referred to as vasospasm.[5,6] Arterial spasm after SAH was originally described by Ecker, and has since been the subject of decades of laboratory research and clinical investigation.[7] Various definitions of vasospasm are employed, including vasospasm seen on digital subtraction angiography or computed tomography angiography referred to as ''angiographic vasospasm'' and ''clinical vasospasm,'' which includes ''delayed ischemic neurologic deficit'' (DIND) and ''delayed cerebral ischemia.'' (DCI) DIND and DCI refer to clinical signs of transient or permanent neurologic deficits occurring remotely from the initial SAH or surgery, after other complications of SAH potentially causing neurologic deficits have been excluded.[5] The exact cause of vasospasm is not clearly understood, but it is thought that extra-arterial blood products surrounding the arterial wall trigger a cascade of events at the cellular level, that culminate in vasoconstriction.[1,4,5] Other factors involved include decreased vascular autoregulation, reversible vasculopathy, and relative hypovolemia.[8,9] A further review of the current pathophysiology of vasospasm is presented in this edition of *Neurosurgical Clinics*. Vasospasm occurs most intensely adjacent to the subarachnoid clot, but can occur distantly from the majority of the subarachnoid blood, and is predicted by clot volume, age, location, and density of the SAH seen on the initial computed tomography (CT) scan.[10,11] In the past, the most likely cause of mortality after SAH was from aneurysmal rerupture in the early period after SAH. Due to more aggressive early surgical and endovascular treatment of ruptured aneurysms, this has now been replaced by hydrocephalus and vasospasm.[12,13]

The incidence of angiographic vasospasm after aneurysmal subarachnoid hemorrhage has been estimated to occur in 50% to 70% of patients with aneurysmal SAH, with approximately 50% of those exhibiting symptoms of clinical vasospasm.[14] A review of angiography studies of more than 2700 cases of aneurysmal SAH found the average incidence to be approximately 67%, with the highest incidence occurring between days 10 and 17 after SAH.[15] Vasospasm classically is reported to occur from days 4 to 14 after aneurysmal SAH, but variations on this rule abound.[1,5,12–14,16,17] The incidence of *early* angiographic vasospasm, detected within 48 hours of aneurysm rupture, occurs in 10% to 13% of SAH patients and is associated with prior aneurysmal SAH, large aneurysms, intraventricular hemorrhage, and with reduced morbidity at 3 months.[18] The impact of clinical vasospasms on outcome has been established, with both morbidity and mortality estimates ranging from 10% to 20%.[15,19]

MODALITIES USED FOR MONITORING CEREBRAL VASOSPASM

It should be emphasized that vasospasm is a clinical diagnosis, and radiographic studies and other markers of brain perfusion support this diagnosis through evidence of diminished vessel caliber. Left unchecked, patients with vasospasm may progress from diffuse neurologic signs such as confusion, increasing somnolence, and combativeness to focal neurologic deficits suggestive of infarction. Radiographic findings often precede such clinical deficits, and thus offer the opportunity to intervene to prevent neurologic injury. To this effect, in 1982 Aaslid and colleagues[20,21] provided the first descriptions of the use of TCD for such purposes, by monitoring flow in intracranial arteries and later used TCD in the assessment of arterial vasospasm. Much work has been done on the use of this technology in the evaluation of cerebral blood flow, due to its relative inexpensiveness, bedside availability, and noninvasive nature. The gold standard for the diagnosis of cerebral vasospasm has remained digital subtraction angiography. Because of its expense, potential for severe complications, and the need to move the patient to the angiography suite, this test is impractical for use as a frequent monitor of vasospasm.[22] The major advantage of angiography is the potential for both diagnosis and therapeutic intervention, discussed elsewhere in this issue. Computed tomography angiography (CTA) has emerged as a potentially helpful tool in the evaluation of vasospasm, with relatively good sensitivity and specificity for discovery of severe vasospasm in the proximal arteries of the circle of Willis, and with a high negative predictive value.[23] Some have raised concern that sending