Technology Update

American Society of Neurophysiologic Monitoring and American Society of Neuroimaging Joint Guidelines for Transcranial Doppler Ultrasonic Monitoring

Harvey L. Edmonds, Jr, PhD, FASNM (ASNM), Michael R. Isley, PhD, DABNM, FASNM (ASNM), Tod B. Sloan, MD, PhD, MBA, FASNM (ASNM), Andrei V. Alexandrov, MD, RVT (ASN), Alexander Y. Razumovsky, PhD, FAHA (ASN)

From the Department of Anesthesiology, University of Louisville, Louisville, Kentucky (HLE); Intraoperative Neuromonitoring, Orlando Regional Medical Center, Orlando, Florida (MRI); Department of Anesthesiology, University of Colorado, Colorado (TBS); Department of Neurology, Division of Cerebrovascular Disease and Comprehensive Stroke Research Center, The University of Alabama at Birmingham, Birmingham, Alabama (AVA); and Sentient NeuroCare Services, Inc., Hunt Valley, Maryland (AYR).

ABSTRACT

The American Society of Neurophysiologic Monitoring (ASNM) and American Society of Neuroimaging (ASN) Guidelines Committees formed a joint task force and developed guidelines to assist in the use of transcranial Doppler (TCD) monitoring in the surgical and intensive care settings. Specifically, these guidelines:
(1) delineate the objectives of TCD monitoring;
(2) characterize the responsibilities and behaviors of the sonographer during monitoring;
(3) describe methodological and ethical issues uniquely relevant to monitoring.
The ASNM and ASN strongly support the positions that (1) acquisition and interpretation of intraoperative TCD ultrasonograms be performed by qualified individuals, (2) service providers define their diagnostic criteria and develop on-going self-validation programs of these performance criteria in their practices. We agree with the guidelines of other professional societies regarding the technical and professional qualifications of individuals responsible for TCD signal acquisition and interpretation (Class III evidence, Type C recommendation). On the basis of current clinical literature and scientific evidence, TCD monitoring is an established monitoring modality for the: (1) assessment of cerebral vasomotor reactivity and autoregulation; (2) documentation of the circle of Willis functional status; (3) identification of cerebral hypo- and hyperperfusion, recanalization and re-occlusion; and (4) detection of cerebral emboli (Class II and III evidence, Type B recommendation).

Keywords: Transcranial Doppler, monitoring, cardiothoracic surgery, vascular surgery, intensive care.

Acceptance: Received May 15, 2009, and in revised form October 19, 2009. Accepted for publication October 23, 2009.

Correspondence: Address correspondence to Alexander Razumovsky, PhD, FAHA, Vice-President and Director, Sentient NeuroCare Services, Inc. 1101 McCormick Rd, Suite 200, Hunt Valley, MD 21031. E-mail: arazumovsky@sentientmedical.com.

J Neuroimaging 2011;21:177-183.
DOI: 10.1111/j.1552-6569.2010.00471.x

Introduction

The Intersocietal Commission on Accreditation of Vascular Laboratories (www.ICAVL.org) has established guidelines for the certification of laboratories making or interpreting diagnostic ultrasonic measurements of cerebral blood flow velocity (CBFV) with transcranial Doppler (TCD) ultrasonography. However, of the more than 950 ICAVL-approved facilities, less than 2% are certified for intracranial or TCD measurements.[1] Practice standards describing the performance of diagnostic TCD examination were published in 2007 by the Journal of Neuroimaging.[2] Previously, the published evidence for diagnostic TCD applications, including peri-operative monitoring and sonothrombolysis, had been assessed by the American Academy of Neurology panel of experts.[3] However, neither ICAVL nor any other recognized professional organization provides guidelines or certification specifically for the unique skills involved in continuous peri-operative or critical care TCD monitoring in order to immediately influence patient care. Thus, Guideline Committees of the American Society of Neurophysiologic Monitoring (ASNM) and American Society of Neuroimaging (ASN) formed a joint task force to develop these practice guidelines for TCD monitoring in the surgical and intensive care settings. The primary goals of this document are to:

(1) delineate the objectives of TCD monitoring;
(2) characterize the responsibilities and behaviors of the sonographer during monitoring; and
(3) describe methodological and ethical issues uniquely relevant to TCD monitoring.

Instrumentation and Acquisition Parameters

Probe Choice

Adequate transcranial insonation of intracranial vessels generally requires ultrasonic probes with crystals operating at 2 MHz or less. Monitoring services should establish local probe performance standards and regularly document that each probe in current use meets these criteria. The crowded surgical/critical

care environment often requires TCD probe cables to have a minimum length of at least 4 m.

Microvascular probes are now available for intracranial application in the assessment of cerebral hemodynamics during cerebral aneurysm[4] and arteriovenous malformation surgeries.[5] These probes have a diameter of 1.5 mm or less and employ a carrier frequency of 16 or 20 MHz. The probes are placed on the vessel of interest using either a malleable wand or rigid suction cannula. Insonation depth and angle are typically .6-1.2 mm and $<30^{\circ}$, respectively.

Probe Fixation

Continuous TCD monitoring requires probe fixation to the insonation site. The probe holder should:

(1) prevent probe movement relative to the insonation site;
(2) avoid traumatic compression of cranial or facial tissues;
(3) permit access to critical cranial or neck regions.

Equipment Selection for TCD Monitoring

TCD low-profile and microvascular Doppler probes used in the critical care and peri-operative environments require capabilities that are often unnecessary in the diagnostic laboratory. However, three new TCD diagnostic applications approved by the Center for Medicare Services in January of 2005 do also require probe fixation. These new applications that bridge the gap between diagnosis and monitoring are (1) evaluation of cerebrovascular reactivity, (2) emboli quantification, and (3) emboli detection with air microbubble injection.

In addition to a low profile, monitoring probes should be relatively immune to radio-frequency contamination. Audio squelching should attenuate acoustic signals of an intensity >110 dB acoustic signals. TCD software should permit trending of CBFV and embolization for at least 8 hours.

Filter Settings

High-pass filtering of TCD signals is necessary to minimize the impact of vessel wall motion artifact. During surgery, very low CBFV signals may be encountered. Thus, filter settings as low as 50 Hz may be required to visualize these low CBFV. In some cases, it may become necessary to totally deactivate the filter.

CBFV Measurement

Although the peak CBFV is the preferred trended parameter by some sonographers because of a favorable signal-to-noise ratio, others rely primarily on the intensity-weighted mean CBFV because it has the highest correlation with independent estimates of cerebral blood flow (CBF).[6] During cardiopulmonary bypass, peak CBFV trending is problematic because of nonpulsatile flow (ie, peak = mean).

Flow Direction Convention

Blood flow direction should be clearly labeled (ie, velocities above baseline are away or toward the transducer).

Safety Considerations

Exposure to Ultrasound

Prolonged high-intensity ultrasonic insonation presents a risk of thermal injury because tissue dissipation of ultrasonic energy results in local warming.[7] Since all the energy studies have been performed in vitro, the potential intracranial heating effect associated with tissue perfusion is unknown. Consequently, there are as yet no internationally established maximum ultrasound intensity output standards for TCD devices and the effects of long-term exposure are not well understood. Due to these concerns, transorbital insonation is currently not recommended for intraoperative or continuous critical care monitoring.

The maximum output of 100 mW/cm^2 recommended by the American Institute of Ultrasound in Medicine has been adopted by all manufacturers of devices cleared for use by the U.S. Food and Drug Administration (FDA). In addition, the lowest pulse repetition frequency should be used which just permits measurement of the highest CBFV. However, the effects of continuous long-term exposure, such as may occur in the surgical/critical care environments, are not fully understood. Therefore, the acoustic power and pulse amplitude should be kept to a minimum commensurate with the production of waveforms that can be accurately measured. Sonographers and monitoring technologists should adhere to the universally accepted ALARA (as low as reasonably achievable) output principle. This is particularly important with transorbital insonation, which places the ultrasonic probe over to the unprotected eye. Transorbital insonation is not recommended for intraoperative or continuous critical care monitoring at this time.

Probe Contamination

Non-sterile ultrasonic probes and gel may expose the eye or scalp wound margins to infectious material. Thus, probes should be carefully cleaned and decontaminated prior to use in these circumstances and sterile aquasonic gel must be used if applying a probe on or near wound margins.

Documentation of TCD Monitoring

CBFV Monitoring

The details of the Fast Fourier Transform (FFT) analysis used to display Doppler shift spectra, including windowing and other digital filtering techniques, should be fully described by the manufacturer. Interpreters of TCD signals should understand the impact of variations in these display techniques on CBFV and pulsatility indices.

Each TCD monitoring recording should, at minimum, include the following information:

(1) patient ID;
(2) procedure date;
(3) sonographer name;
(4) vessel identification;
(5) recording parameters (mean CBFV and PI);
(6) relevant physiologic variables;
(7) qualitative description of changes (if any) in the spectral morphology of waveforms.

With microvascular probes, documentation should include both qualitative and quantitative changes in the spectral waveforms.

Embolus Detection

The report of the International Consensus Committee on microembolus detection by TCD[8] recommended that both research and clinical materials be accompanied by a summary of the following technical parameters:

(1) make and model of ultrasound device;
(2) transducer type and size;
(3) insonated artery;
(4) algorithm for signal intensity measurement;
(5) velocity and time scales;
(6) embolus detection threshold;
(7) axial extension of sample volume;
(8) FFT size (number of points); length (time), and percentage overlap;
(9) high-pass filter setting;
(10) quantitative classification of emboli (if any).

Since these parameters may influence both emboli detection and CBFV measurement, sonographers be familiar with the impact of these parameters. Changes from established standard protocols should be documented, especially with services utilizing TCD instruments from multiple manufacturers.

Credentials for TCD Monitoring and Interpretation

The ASNM recognizes the importance of appropriately qualified intraoperative monitoring (IOM) personnel and refers the reader to its recent position statement regarding this sensitive issue.[9] Unfortunately, none of the ASNM-recommended certifying agencies directly address TCD monitoring competency. The American Registry of Diagnostic Medical Sonographers (ARDMS) certifies vascular ultrasound technologists. However, neither the content of the Registered Vascular Technologist (RVT) examination nor the required skills (www.ardms.org) reflect competencies necessary to perform TCD examination and/or intraoperative/critical care monitoring.

Training for physician proficiency in interpretation of cerebrovascular ultrasound studies with particular emphasis on applied principles of ultrasound physics, fluid dynamics, and various aspects of TCD examination has been offered for decades by the ASN (www.asnweb.org). Nevertheless, certification of technical sonographic expertise in TCD evaluation of intracranial vessels is currently not available in the United States. At the beginning of 2006, the Board of Directors of ASN approved creation of the first national examination to assess a technologist's ability to apply knowledge, concepts, and principles of neurovascular ultrasound that constitute the basis of safe and effective patient care. It is designed to measure the candidate's application of medical knowledge and understanding of biomedical and clinical sciences that are considered to be essential for the unsupervised performance of neurovascular ultrasound. The Neurovascular Technology (NVT) examination assesses proficiency in neurosonology (carotid duplex and TCD ultrasound). The examination is intended for technologists and physicians who, through years of training and experience in these neurovascular technologies, have acquired a foundation in the basic principles, techniques, and interpretation of neurosonology to record, interpret, and present preliminary findings to supervising physicians. The examination focus is on both diagnostic and monitoring TCD examinations and includes details relevant to continuous monitoring. Successful completion of the examination results in the designation of NVT, or registered NVT. This work is still in progress.

TCD Monitoring Rationale

Preoperative Considerations

Interpretation of cerebral hemodynamic changes occurring during surgery or critical care is heavily influenced by the patient's underlying pathology and the use of anesthetic and other vasoactive agents. Chronic hypertension, diabetes mellitus, cerebral atherosclerosis, and nicotine use may diminish cerebral autoregulation,[10] making both flow and flow-velocity dependent on systemic arterial pressure. Also lacking may be cerebral arterial reactivity to change in intravascular hydrogen ion concentration (ie, CO_2 reactivity). Especially with concomitant unilateral or asymmetric carotid stenosis or previous cerebral infarct, vascular reactivity in the two hemispheres may differ.[11] In such cases, unilateral insonation may result in a misperception about brain tissue at risk. Thus, preoperative intracranial hemodynamic information is often a helpful guide to intraoperative TCD monitoring.

The skull attenuates ~80% of the ultrasonic energy prior cerebral vessel insonation.[12] Because of temporal bone hyperostosis related to age, gender, and race,[13] inadequate temporal ultrasonic windows occur in a subpopulation of patients.[14] Identification of this situation prior to surgery may enable the sonographer to utilize a submandibular or foramenal insonation site.

Assessment of Intracranial Hemodynamic

Hemodynamic considerations and TCD monitoring objectives should be discussed with the surgeon/anesthesiologist/intensivist prior to monitoring initiation. Continuous CBFV recordings from a single insonation site can provide clinically valuable trend information. TCD monitoring is well suited to identify flow direction or detect sudden dramatic CBFV change (ie, relative hypo- or hyperperfusion, recanalization, or re-occlusion).[15] Change in the ratio of velocities at peak systole and end-diastole also can be used to characterize change in cerebral vascular resistance (ie, venous obstruction or intracranial hypertension).[16] Finally, TCD monitoring can identify the sudden appearance of echogenic material (ie, emboli).[17] In certain clinical situations, each of these pieces of information, unobtainable by other means, may be literally life-saving.[18]

Circle of Willis Function

Correct identification of the circle of Willis vessels and accurate CBFV measurements within these vessels are based on a set of established criteria.[2] The M1 segment of the middle cerebral artery (MCA) is the most frequently utilized for TCD monitoring applications, since it carries up to 40% of the hemispheric blood flow. During carotid occlusion, the TCD signal from the internal carotid artery disappears but is often retained in the perfused MCA segment distal to the circle of Willis. With loss

of the MCA signal, a persistent anterior cerebral artery signal demonstrates flow reversal if collateralization via anterior communicating artery occurs or flow diversion away from the probe if the MCA becomes obstructed.[15]

Ischemic Threshold

Since the absolute CBFV ischemic threshold has not been established, common practice relies on a relative index of ischemia. In unanesthetized adult patients undergoing tilt-table testing, clinical signs of cerebral hypoperfusion appear with reductions in MCA CBFV >60%.[19]

Carotid Endarterectomy

Spencer et al. described an exponential relationship between carotid clamp-related decreases in MCA mean CBFV and carotid artery stump pressure.[20] The CBFV fell to undetectable levels at a stump pressure of 15 mmHg. The authors concluded that TCD "provided an excellent indicator as to the necessity of shunting." Similarly, Kalra et al. found that patients with stump pressures below 30 mmHg had significantly lower CBFV than those with higher pressures.[21]

The results of a large multicenter retrospective study of carotid endarterectomy (CEA) patients suggested that severe ischemia (ie, high probability of new neurodeficit) was associated with a >85% reduction in CBFV, while moderate ischemia represented a 60%-85% decrease.[22] During CEA under regional anesthesia, clamp-related reduction in mean CBFV >40% predicted clinical signs of developing neurologic dysfunction with a sensitivity of 92% and specificity of 75%.[23] To improve predictability, others have recommended a 70% reduction for the ischemic threshold.[24] With adequate leptomeningeal collateral flow, EEG activity occasionally may remain unchanged in the presence of severely decreased or absent MCA CBFV.

Intracranial Aneurysm Repair

Occlusion of an intracranial artery is often required for surgical treatment of a giant cerebral aneurysm. TCD monitoring during carotid occlusion has identified hemispheric dependence on ipsilateral carotid flow.[25] The appearance of neurologic sequelae correlated with the magnitude of the "relative" CBFV decrease from individualized pre-occlusion reference, but not with the "absolute" CBFV. Neurologic signs of transient focal deficit consistently occurred with CBFV decreases greater than 65%.

Cardiopulmonary Bypass

Cardiopulmonary bypass (CPB) may result in substantial alterations in red cell mass, blood viscosity, oxyhemoglobin dissociation, acid-base balance, and vasoneural coupling. Nevertheless, the TCD cerebral ischemic threshold has been defined as a mean CBFV decline of 80% below the pre-incision baseline CBFV in both adults[26] and children.[27]

Hyperperfusion Threshold

Following successful removal of a stenotic carotid lesion, cerebral hyperemia due to reperfusion may occur in up to 20% of cases.[28] CBFV more than 50% above normal may persist for up to 2 weeks postoperatively and may result in a cerebral hyperperfusion syndrome[29] or hemorrhagic infarct.[30]

Cerebral Emboli Detection

An international consensus committee has concluded that no commercially available automatic embolus detection system has the required sensitivity and specificity for clinical use.[8] The following comments are made to clarify the contentious issues and complexities of cerebral emboli monitoring.

Embolus Identification and Classification

Echogenic substances with acoustic impedances greater than erythrocytes have been referred to as microemboli, microbubbles, particulate emboli, formed-element emboli, and Doppler micro-embolic signals (MES), while the term high-intensity transient signals (HITS) appears currently to be the most widely used. The basic features of Doppler particulate embolic signals include:[8]

(1) transient character (duration <300 ms),
(2) duration dependent on the passage time through the sample volume,
(3) high-intensity (amplitude >3 dB above background),
(4) unidirectional
(5) acoustic resemblance to "snaps, tonal chirps, or moans."

Gaseous microembolic signals are often differentiated by their bidirectionality and higher intensities (>25 dB above background). In contrast, acoustic artifacts, although also bidirectional, are predominantly of low frequency (<400 Hz). These artifacts may arise from electrical interference or movement of the probe, cable, or patient.

Although recent development of multifrequency and multigated Doppler instruments has led to claims of improved sensitivity and specificity of emboli detection, the U.S. FDA has yet to clear for clinical use ultrasonographs equipped with these technologies. A curious and notable limitation of the current generation of emboli measurement systems is the inability to detect the most serious clinical situation, a massive gaseous embolization. Since all current emboli detection algorithms focus on the discrete nature of individual transient embolic events, sustained high-intensity signals remain unrecognized.

Embolus Quantification

Presumably thrombotic or atheromatous HITS may be detected in as many as 80% of CEA.[24] These emboli appear to be responsible for the majority of CEA-associated cerebrovascular complications.[31] For example, Payne et al. found that in the first hours after endarterectomy, neurologic injury often accompanied HITS formation with a rate exceeding 2 per minute.[32] Aggressive thrombolysis may subsequently eliminate both HITS and the neurologic complications.

HITS composition during cardiac surgery is less certain because of the substantial opportunity for the unintentional introduction of air into the cerebral circulation during CPB and the production of cavitation bubbles by mechanical prosthetic devices. Some studies have implicated HITS in the etiology of both neurologic injury and cognitive decline after cardiac

surgery,[33] while others have not.[34] No study has proposed a critical HITS injury threshold.

Influence of Anesthetic Management

Induction and maintenance of general anesthesia may have a profound effect on cerebral hemodynamics.[35] An understanding of anesthetic techniques and action of agents commonly used by anesthesia providers is essential to interpret TCD changes occurring during surgery. Whenever practical, bilateral TCD probes should be positioned and fixed prior to preoxygenation and anesthetic induction. This permits establishment of baseline hemodynamic in the conscious patient prior to patient positioning, hyper- or hypocapnea and the administration of an array of vasoactive drugs. A wide range of variables under the influence of the anesthesia provider may affect CBFV. These include:

(1) hypertension–blood pressure increases above the upper limit of cerebral autoregulation;
(2) hypotension–blood pressure decreases below the lower limit of cerebral autoregulation may lead to a precipitous decline in CBFV (ie, the vascular waterfall);
(3) hypoxia–with intact cerebral CO_2 reactivity and autoregulation, low oxygen tension should result in increased CBFV;
(4) hyper- and hypocapnea–with intact CO_2 reactivity, change in CBFV should be proportionate to change in arterial CO_2 tension; and
(5) anesthetic agents–intravenous hypnotics tend to reduce CBFV, while halogenated volatile anesthetics tend to increase it in a dose-dependent fashion.

Influence of Extracorporeal Circulatory Support

TCD is the only available method to continuously assess changes in cerebral hemodynamics during CPB. The specific objectives of TCD monitoring include the following:

(1) TCD indicates CBF presence and direction and may detect a malpositioned perfusion cannula or inadvertent great vessel occlusion. During attempted retrograde cerebral perfusion, TCD documents CBF direction.
(2) TCD may aid in the determination of safe upper and lower limits for pump flow and perfusion pressure.
(3) End-diastolic CBFV change is inversely related to change in cerebrovascular resistance. A sudden increase in resistance may indicate compromised venous return, perhaps due to a malpositioned or partially occluded venous cannula. Post-CPB persistence of the pattern may suggest developing cerebral edema and a need for ultrafiltration.
(4) TCD quantitative identification of embolization may improve surgical and perfusion technique,[35] facilitate correction of technical problems such as an air leak[36] and improve patients outcome.[37]

Intervention Criteria

The presence of radio-frequency interference from the immediate or neighboring operating rooms or inadequate probe fixation may result in an ambiguous, obscured, or evanescent CBFV spectrum. Similarly, the absence of adequate supporting physiologic data may preclude effective interpretation of high-quality TCD spectra. It is the responsibility of the sonographer to notify the surgical/anesthesia/critical care team of these circumstances in order to prevent a false sense of security. No monitoring is preferable to the provision of potentially misleading information.

Emerging Applications of TCD Monitoring

Clinical neurological events may complicate carotid angioplasty and stenting (CAS) procedures.[38] Obstructive carotid lesions are known to contain friable thrombotic and atherosclerotic components that can embolize during CAS. In fact, the presence of new Diffusion-Weighted (DWI) Magneto-Resonance Imaging (MRI)-detected ischemic cerebral microinfarcts is highly correlated with the number of HITS.[39] Thus, TCD detection of emboli and potentially adverse CBFV changes during and immediately after CAS may improve patients' outcome.

Another emerging application of TCD monitoring is coronary and cerebral angiographic catheterization.[40] Although TCD-detected cerebral microembolism often occurs during cardiac catheterization, its clinical relevance remains uncertain. After percutaneous cardiac interventions, the incidence of asymptomatic cerebral infarction detectable by DWI MRI is as high as 22%.[40] The current SCIPION (Silent cerebral infarct after cardiac catheterization as detected by diffusion weighted Magnetic Resonance Imaging: a randomized comparison of radial and femoral arterial approaches. NIH Registry NCT 00329979) trial addresses the clinical relevance of the TCD embolic signals. The study uses serial DWI MRI to assess the rate of silent cerebral infarction after cardiac catheterization.[41] In a TCD M-mode-monitored subgroup, pre- and post-procedure neuropsychological testing will assess the impact of ultrasonic microembolic signals and silent brain injury on cognitive decline.

Initial attempts have been made to monitor intraarterial revascularization procedures for acute ischemic stroke patients.[42] TCD monitoring offers unique information about often unexpected flow changes such as re-occlusion, air embolization and hyperperfusion. This information may be particularly valuable in sedated and ventilated patients, since clinical examination is limited. TCD provides a tool to expand hyper-acute stroke treatment monitoring from the emergency department to catheterization laboratories and intensive care settings in an extended time window.[43]

Medico-Legal and Ethical Considerations

The Washington State Blue Shield carrier has published the position that TCD monitoring is reimbursable for selected surgical procedures.[44]

Current TCD Limitations

On the basis of current clinical literature and expert opinion, TCD monitoring has limitations as a monitoring tool. TCD is an effective means of identifying sudden change in large vessel CBFV or flow direction during CEA, repair of intracranial aneurysms and cardiovascular surgery. However, optimal monitoring also involves other technologies which may include electroencephalography, sensory- and/or motor-evoked potentials, and transcranial near-infrared spectroscopy. Current technology provides, at best, a semi-quantitative estimate of the number of cerebral microemboli to appear within a specific vascular

segment. Nevertheless, ultrasonic emboli monitoring can be an effective technique for the detection of cerebral embolization.

The reliable TCD distinction between particulate and gaseous emboli is still under development. It is not currently possible to determine either the size or composition of individual emboli. In the absence of an anatomical anomaly or occlusive vascular disease, insonation of the intracranial anterior circulation transorbitally, and posterior circulation from a foramenal approach is possible in nearly all patients. Cranial hyperostosis, however, may prevent transtemporal insonation of the anterior circulation in a small percentage of patients (10%-20%).[45]

Other limitations include the need for the trained physicians (neurologists, anesthesiologists, intensivists, radiologists, etc), sophisticated equipment, knowledgeable technologists, extensive experience with documented reliability, and compatibility with the other numerous monitoring devices in the operating room or in the ICU.

Major Recommendations

(1) The ASNM and ASN strongly support the positions that (1) acquisition and interpretation of intraoperative TCD ultrasonograms should be performed by qualified individuals and (2) service providers define their diagnostic criteria and develop ongoing self-validation programs of these performance criteria in their practices. It agrees with the guidelines of other professional societies regarding the technical and professional qualifications of individuals responsible for TCD signal acquisition and interpretation. (Class III evidence, Type C recommendation).

(2) On the basis of current clinical literature and scientific evidence, TCD monitoring is an established monitoring modality for the: (1) assessment of cerebral vasomotor reactivity and autoregulation; (2) documentation of the circle of Willis functional status; (3) identification of relative cerebral hypo- and hyperperfusion;and (4) detection of cerebral emboli. (Class II and III evidence, Type B recommendation.)

The authors are grateful to Dr. Viken Babikian, MD, for his manuscript review, thoughtful comments, and suggestions.

References

1. Cited by http://intersocietal.org/iac/news/div_icavl/2009/tcd_intro4.htm IAC *Newslett* 2009;1:6-9.
2. Alexandrov AA, Sloan MA, Wong LKS, et al. for the ASN. Practice standards for transcranial Doppler ultrasound: part I – test performance. *J Neuroimaging* 2007;17:11-18.
3. Sloan MA, Alexandrov AV, Tegeler CH, 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.
4. Bailes JE, Tantuwaya LS, Fukushima T, et al. Intraoperative microvascular Doppler sonography in aneurysm surgery. *Neurosurg* 1997;40:965-972.
5. Akdemir H, Oktem S, Menkü A, et al. Image-guided microneurosurgical management of small arteriovenous malformation: role of neuronavigation and intraoperative Doppler sonography. *Minim Invasive Neurosurg* 2007;50:163-169.
6. Larsen FS, Olsen KS, Hansen BA, et al. Transcranial Doppler is valid for determination of the lower limit of cerebral blood flow autoregulation. *Stroke* 1994;25:1985-1988.
7. Nyborg WL. Optimization of exposure conditions for medical ultrasound. *Ultrasound Med Biol* 1985;11:245-260.
8. Ringlestein EB, Droste DW, Babikian VL, et al. Consensus on microembolus detection by TCD. *Stroke 1998*;29:725-729.
9. Isley MR, Edmonds HL Jr, Stecker M. Guidelines for intraoperative neuromonitoring using raw (analog or digital waveforms) and quantitative electroencephalogrphy: a position statement by the American Society of Neurophysiololgical Monitoring. *J Clin Monitor Comput* 2009; DOI:10.1007/s10877-009-9191-y
10. Muller M, Schimrigk K. Vasomotor reactivity and pattern of collateral blood flow in severe occlusive carotid artery disease. *Stroke* 1996;27:296-299.
11. Eames PJ, Blake MJ, Dawson SL, et al. Dynamic cerebral autoregulation and beat to beat blood pressure control are impaired in acute ischaemic stroke. *J Neurol Neurosurg Psychiatr* 2002;72:467-472.
12. Grolimund P. Transmission of ultrasound through the temporal bone. In: Aaslid R, ed. *Transcranial Doppler Sonography*. New York: Springer-Verlag, 1986: 10-21.
13. Saver JL, Feldman F. Basic transcranial Doppler examination: technique and anatomy. In: Babikian VL and Wechsler LR, eds. *Transcranial Doppler Ultrasonography*. St. Louis: Mosby, 1993: 11-28.
14. Bass A, Krupski WC, Schneider PA, et al. Intraoperative transcranial Doppler: limitations of the method. *J Vasc Surg* 1989;10:549-553.
15. Alexandrov AV (ed.) *Cerebrovascular Ultrasound in Stroke Prevention and Treatment*. New York: Futura/Blackwell Publishing, 2003.
16. Hassler W, Steinmetz H, Gawlowski J. Transcranial Doppler ultrasonography in raised intracranial pressure and in intracranial circulatory arrest. *J Neurosurg* 1988;68:745-750
17. Albin MS, Bunegin BS, Garcia C, et al. Transcranial Doppler can image microaggregates of intracranial air and particulate matter. *J Neurosurg Anesth* 1989;1:134-135.
18. Guerra WK, Gaab MR, Dietz H, et al. Surgical decompression for traumatic brain swelling: indications and results. *J Neurosurg* 1999;90:187-196.
19. Edmonds HL Jr, Singer I, Sehic A, et al. Multimodality neuromonitoring for neurocardiology. *J Interven Cardiol* 1998;11(3):197-204.
20. Spencer MP, Thomas GI, Moehring MA. Relation between middle cerebral artery blood flow-velocity and sump pressure during carotid endarterectomy. *Stroke* 1992;23:1439-1445.
21. Kalra M, al-Khaffaf H, Farrell A, et al. Comparison of measurement of stump pressure and transcranial measurement of flow-velocity in the middle cerebral artery in carotid surgery. *Ann Vasc Surg* 1994;8:225-231.
22. Halsey J. Monitoring blood flow velocity in the middle cerebral artery during carotid endarterectomy. In Babikian VL and Wechsler LR, eds. *Transcranial Doppler Ultrasonography*. St. Louis: Mosby, 1993: 216-221.
23. McCarthy RJ. The value of transcranial Doppler in predicting cerebral ischemia during carotid endarterectomy. *Eur J Vasc Endovasc Surg* 2001;21:408-412.
24. Babikian VL, Cantelmo NL, Wijman CAC. Neurovascular monitoring during carotid endarterectomy. In Babikian VL and Wechsler LR, eds. *Transcranial Doppler Ultrasonography*. 2nd ed. Boston: Butterworth-Heinemann, 1999: 231-245.
25. Schneweis S, Urbach H, Solymosi L, et al. Preoperative risk assessment for carotid occlusion by transcranial Doppler ultrasound. *J Neurol Neurosurg Psychiatr* 1997;62:485-489.
26. Edmonds HL Jr. Protective effect of neuromonitoring during cardiac surgery. *Ann NY Acad Sci* 2005;1053:12-19.
27. Austin EH 3rd, Edmonds HL Jr, Auden SM, et al. Benefit of neurophysiologic monitoring for pediatric cardiac surgery. *J Thorac Cardiovasc Surg* 1997;114:707-715.
28. Jorgensen LG, Schroeder TV. Defective cerebrovascular autoregulation after carotid endarterectomy. *Eur J Vasc Surg* 1993;7:370-379.
29. Muller M, Behnke S, Walter P. Cerebrovascular resistance and blood flow-velocity changes after carotid endarterectomy. *Vasa* 1999;28:279-282.
30. Piepgras DG, Morgan MK, Sundt TM Jr, et al. Intracerebral hemorrhage after carotid endarterectomy. *J Neurosurg* 1988;68:532-536.

31. Ackerstaff RGA, Moons KGM, van de Vlasakker CJW, et al. Association of intraoperative transcranial Doppler monitoring variables with stroke from carotid endarterectomy. *Stroke* 2000;31:1817-1823.
32. Payne DA, Jones CI, Hayes PD, et al. Beneficial effects of clopidogrel combined with aspirin in reducing cerebral emboli in patients undergoing carotid endarterectomy. *Circulation* 2004;109:1476-1481.
33. Stump DA, Kon NA, Rogers AT, et al. Emboli and neuropsychological outcome following cardiopulmonary bypass. *Echocardiogr* 1996;13:555-558.
34. Georgiadis D, Kaps M, Berg J, et al. Transcranial Doppler detection of microemboli in prosthetic heart valve patients: dependency upon valve type. *Eur J Cardio-Thorac Surg* 1996;10:253-257.
35. Edmonds HL Jr. Advances in neuromonitoring for cardiothoracic and vascular surgery. *J Cardiothorac Vasc Anesth* 2001;15:241-250.
36. Yeh TJ Jr, Austin EH 3rd, Sehic A, et al. Role of neuromonitoring in the detection and correction of cerebral air embolism. *J Thorac Cardiovasc Surg* 2003;126:589-591.
37. Razumovsky AY, Gugino L, Owen JH. Advanced neuromonitoring for cardiothoracic and vascular surgery. *Sem Cerebrovasc Dis Stroke* 2005;5:141-154.
38. Barbato JE, Dillavou E, Horowitz MB, et al. A randomized trial of carotid artery stenting with and without cerebral protection. *J Vasc Surg* 2008;47:760-765.
39. Gossetti B, Gattuso R, Irace L, et al. Embolism to the brain during carotid stenting and surgery. *Acta Chir Belg* 2007;107:151-154.
40. Wong SC, Minutello R, Hong MK. Neurological complications following percutaneous coronary interventions (A report from the 2000-2001 New York State angioplasty registry). *Am J Cardiol* 2005;96:1248-1250.
41. Hamon M, Gomes S, Oppenheim C, et al. Cerebral microembolism during cardiac catheterization and risk of acute brain injury. A prospective diffusion-weighted magnetic resonance imaging study. *Stroke* 2006;37:2035-2038.
42. Ribo M, Molina CA, Alvarez B, et al. Intra-arterial administration of microbubbles and continuous 2-MHz ultrasound insonation to enhance intra-arterial thrombolysis. *J Neuroimaging*. 2009 Feb 13. [Epub ahead of print] PubMed PMID: 19226340.
43. Chernyshev OY, Garami Z, Calleja S, et al. The yield and accuracy of urgent combined carotid-transcranial ultrasound testing in acute cerebral ischemia. *Stroke* 2005;36:32-37.
44. Anonymous. Medical policy. Washington State Blue Shield Provisions. March 1999.
45. Marinoni M, Gianneschi A, Forleo P, et al. Technical limits in transcranial Doppler recording: inadequate acoustic windows. *Ultrasound Med Biol* 1997;23:1275-1277.