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Anticardiolipin antibodies and transverse myelopathy: expanding our understanding of an elusive clinical problem.

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with an acute ischemic stroke. Information obtained from TCD was applied to adjust the duration and dosage of thrombolytic therapy; perhaps another important use of this modality. TCD offers important advantages over other techniques used for the assessment of cerebrovascular hemodynamics in its speed, safety, and cost-effectiveness. Why, then, does this method remain largely outside the mainstream of the neuroradiologist's techniques? A number of factors may create this paradox.

First, TCD measurements are extremely operator-dependent. The majority of neuroradiologists do not have significant hands-on experience performing Doppler examination and thus lack the necessary skills to perform or supervise the performance of such studies. This, combined with the neuroradiologist's proclivity for anatomy over physiology combined with a non-anatomical TCD-generated data format may create a subtle but very real bias against the technique.

A second possible explanation for the neuroradiologist's underutilization of TCD relates to a temptation to extract more information from examinations than is actually present in the data. As with a variety of other diagnostic modalities, this can result in an initial enthusiasm that is soon replaced by a mistrust

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of the approach. Using currently available instruments, TCD can accurately reveal the direction and velocity of blood flow at the level of the Circle of Willis, particularly in the middle and anterior cerebral arteries. The shape of the waveform provides information that can also be used to *estimate* the state of resistance in the downstream vasculature. Since it is not possible to determine and monitor the diameter of an examined artery accurately, TCD cannot determine if elevated velocity is caused by an increased blood flow or a decreased arterial diameter. The complexities of intracranial hemodynamics significantly limit TCD. The recent development of ultrasound devices suitable for intracranial duplex examinations offers promise for considerable improvement in this shortcoming.

Because of the intimate relationship between velocity changes, vessel diameter, and accurate measurement of the direction and velocity of blood flow, TCD is most widely used either to assess the state of collateral pathways or to monitor relative changes in flow. In the latter example, relative changes in flow can be assumed to result most often from perfusion pressure variation such as may occur during test occlusion of the internal carotid artery or development of caliber changes in the large distribution arteries as in post subarachnoid hemorrhage vasospasm. Although few direct correlations exist between velocity measurements and the perfusion of downstream tissue in the above applications, considerable experience indicates the technique still provides information that, when viewed in the context of individual patients, is helpful for making management decisions. Decreases in middle cerebral artery velocities of more than 65% have been associated with evidence of significant decreases in tissue perfusion in a variety of circumstances such as in tolerance tests of internal carotid artery occlusion, carotid endarterectomy, and evaluations of patients with position-related ischemic symptoms. Such observations, though empirical, often prove to be of significant practical value. Recently, TCD's capability to detect intracranial embolic events has spurred several investigations. This ability is unique to TCD and seems a potentially valuable aid in the monitoring of many endovascular procedures.

As TCD-related technology improves, this method of investigation has great potential to increase the quality of available information regarding the dynamics of cerebrovascular circulation. This will heighten the understanding of cerebrovascular hemodynamics. Neuroradiologists should seize the opportunity to participate in the development and application of this technology.

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Anticardiolipin Antibodies and Transverse Myelopathy: Expanding Our Understanding of an Elusive Clinical Problem

One of the most difficult problems in clinical neurology involves the diagnosis and treatment of a patient presenting with an acute or recurrent transverse myelopathy. When there is an abrupt onset of motor, sensory or autonomic spinal cord dysfunction, the primary aim of initial imaging studies is to rule out compressive mass such as a hematoma, tumor, bone/disc, or infectious process. When these and other underlying lesions such as a vascular malformation or spinal cord tumor have been excluded by history or imaging findings, other causes of acute transverse myelopathy (ATM) are sought. These include collagen-vascular disorders, multiple sclerosis, infectious myelitis, or a parainfectious/paraneoplastic process. When the exact origin for the first or recurrent episodes of ATM cannot be established, the term *idiopathic acute transverse myelopathy* is applied. *Myelopathy* is preferred over *myelitis* because it is unclear if an inflammatory condition exists in these patients. In this issue of the *American Journal of Neuroradiology* Campi et al (page 778) draw our attention to the possibility that many of these idiopathic and recurrent cases may be the result of underlying thromboses created by circulating anticardiolipin antibodies.

Antiphospholipid antibodies, including the lupus anticoagulant and the anticardiolipin antibodies, have been implicated in a number of clinical problems such as cerebral and myocardial infarctions, deep leg and hepatic venous thromboses, pulmonary emboli, valvular heart disease, and miscarriages. There has, however, been little mention of these immunoglobulins in relation to acute cord dysfunction. It is reasonable to suspect that because multiple systems can be affected, the spinal cord may also be a target for arterial or venous thrombosis. The series of 3 cases presented by Campi is admittedly small and pathologic proof of vascular thrombosis is lacking; however, the concept that elevated anticardiolipin antibodies may be responsible for an acute or a recurrent transverse myelopathy is important. The authors correctly assert that they are unsure whether the abnormal anticardiolipin antibodies are primary or the result of an otherwise undiagnosed disease in which the elevated anticardiolipin would represent an epiphenomenon. In either case, a vascular cause for myelopathy would be a strong possibility that could impact patient treatment.

There is nothing pathognomonic or unique about the abnormal patterns in ATM from an MR imaging standpoint. Usually thoracic cord enlargement over a number of segments, diffuse or patchy signal abnormalities on T1 or T2 weighted images, and a variable degree of contrast enhancement is expected. During clinical remission, many of these findings regress. The

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lack of a consistent pattern of white matter signal abnormality and the presence of long segment involvement diminishes the possibility of a primary demyelinating process as does the lack of a striking or exclusive central gray matter abnormality lessen the likelihood of a spinal artery thrombosis. The pattern of MR abnormality in Campi et al's cases, particularly with thoracic cord involvement, is reminiscent of venous ischemia, as may be seen in cases of venous hypertension in spinal dural arteriovenous malformations.

Increasing our awareness of the possible vascular origin of cord dysfunction that is secondary to the presence of anticardiolipin antibodies may decrease the diagnostic uncertainty surrounding patients who present with an acute or recurrent transverse myelopathy.

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