# Spinal cord abnormalities in metabolic, nutritional, and toxic disorders.

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rollment in the ECASS study). Another important finding of Kucinski et al's report is that complete or incomplete recanalization was achieved in only 37% of patients with carotid "T" occlusions, compared to a 61% recanalization rate for the entire cohort of patients. Successful recanalization was proven to be a statistically significant prognosticator; for the entire cohort of patients, successful recanalization was followed by death in 13% of patients compared to 61% when recanalization failed. The authors conclude that thrombolytic treatment for patients with carotid "T" occlusions is not useful and may even be dangerous because of the higher rate of hemorrhagic transformation observed in this subset of patients.

Important questions regarding thrombolytic treatment for acute stroke patients remain unanswered. Which subset of patients will have better outcomes with intraarterial treatment compared to intravenous treatment and vice versa? Which patients will do better with no thrombolytic treatment? Answers to these questions will likely depend heavily upon the site of vascular occlusion. Many neurologists and neuroradiologists intuitively agree that intraarterial thrombolysis should have better outcomes than intravenous thrombolysis since more drug is delivered to the site of occlusion. That concept, however, still remains to be proven in a randomized, controlled study. For all physicians participating in the evaluation and management of the acute stroke patient, a crucial question remains. What is the best neuroimaging method to distinguish between already-infarcted tissue and potentially salvageable ischemic tissue at risk for infarction; the ischemic penumbra?

It is certainly understandable why angiography was not obtained prior to randomization in the ECASS and NINDS trials. The inherent delay of mobilizing the angiography team and the performance of the procedure would have pushed many participants out of the therapeutic time window. Therefore, by default, CT was the only neuroimaging procedure employed in both trials; primarily to exclude patients with hemorrhage in the NINDS trial and to exclude patients with hemorrhage and an infarct greater than one third of the middle cerebral artery territory in the ECASS trial. I believe a noncontrast CT is an inadequate screening examination when done prior to instituting thrombolytic therapy. It is fraught with problems of poor interobserver agreement regarding the extent of already-infarcted brain tissue. In addition, it often gives us little information regarding the site of vascular occlusion and no information regarding the size of the ischemic penumbra. The time has come for us to start employing currently available, noninvasive imaging techniques that were not readily available prior to 1995 during the conduction of the ECASS and NINDS trials. Numerous articles have appeared in the literature in recent years demonstrating the utility of MRA, diffusion/perfusion echo-planar MR imaging, MR spectroscopy, xenon CT, helical CT with bolus contrast technique, and CT angiography in the evaluation of the acute stroke patient. As we continue to upgrade CT and MR scanners, many of these techniques and modalities will become readily accessible at the community level within the accepted time necessary for the evaluation and management of acute stroke patients. It is our responsibility to determine which exam or combination of exams is optimal in the pretherapeutic evaluation of the acute stroke patient.

We know that the site of vascular occlusion and the volume of the ischemic penumbra are important indicators for the efficacy and safety of thrombolytic treatment. The final chapter on acute stroke therapy was not written with the publication of the ECASS and NINDS trials. We, as diagnostic and interventional neuroradiologists, will need to play an important role in the design and implementation of future acute stroke therapy trials.

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## Spinal Cord Abnormalities in Metabolic, Nutritional, and Toxic Disorders

It is well known that certain metabolic and nutritional disorders preferentially affect particular areas and tracts of the spinal cord. In this issue of the journal, Pema et al (page 894) describe and illustrate the MR findings of a patient in whom discrete dorsal column involvement resulted from nitrous oxide abuse and secondary vitamin B12 inactivation. The predominate, but not exclusive, dorsal column abnormality in diseases such as subacute combined degeneration and vacuolar myelopathy raises the question of why such selectivity should exist if these diseases are biochemical in nature; in other words, why, as beautifully demonstrated in Figure 1B of Pema's report, is this the preferential site?

The high signal on T2 weighted images in this patient, confined to the dorsal columns, correlated well with findings of diminished light touch and vibratory sensation, ataxia (loss of joint position sense),

and the absence of upper motor neuron signs. Curiously, while these neurologic abnormalities involved mainly the lower extremities, inspection of Figure 1B suggests at least partial sparing of the fasciculus gracilis (mid-line) but apparent full involvement of the fasciculus cuneatus. Based simply on this topographic distribution, one might have suspected that the upper extremities would have been affected equally or to a greater extent than the lower extremities. The reason for this incongruity is unclear. Another apparent discrepancy between the clinical and MR findings is the presence of spinothalamic tract dysfunction (decreased pin prick) with apparent lack of T2 image abnormalities in this region. It is probable that the spinothalamic tracts are spared because they carry second-order axons.

In this patient it is possible that the dorsal column abnormalities could have been the result of an associated peripheral neuropathy, particularly in light of the fact that nitrous oxide toxicity as well as a primary vitamin B12 deficiency may cause a peripheral neuropathy. Although no nerve conduction studies were performed, one might have expected small or absent sensory potentials indicating the presence of a peripheral axonal degeneration which then might have resulted in dorsal column abnormalities (a dying back phenomena).

One cannot help but be struck by the similarities between the pattern of involvement in this particular case and that seen in vacuolar myelopathy, as noted in patients with AIDS in which vacuolization of the white matter mainly in the lateral and posterior columns of the thoracic cord has been described. Although a nutritional deficiency of vitamin B12 is common in AIDS, most AIDS patients with vacuolar myelopathy have normal serum vitamin B12 levels and it is possible that other nutritional deficiencies such as methionine deficiency may be responsible for the myelin vacuolization. In Dr. Pema's patient, who suffered from nitrous oxide toxicity, the reversal of symptoms and the return of the MR to normal (Figure 2B) after cessation of nitrous oxide abuse and the addition of vitamin B12 therapy is striking. This suggests a recovery from the intracellular fluid accumulation in the myelin sheaths and a reconstitution of normal myelin.

In Dr. Pema's report, we are left with MR findings that are to a certain degree in concert with the presenting symptoms. Nonetheless, certain questions concerning the pattern of involvement remain unanswered. In an upcoming issue of the *American Journal of Neuroradiology*, a review of peripheral nervous system (PNS) imaging will appear. With new MR techniques to detect and characterize abnormalities in the PNS, it may be possible to establish, by imaging alone, what the contributions of a peripheral neuropathy are to the clinical status of patients with metabolic, nutritional, and toxic disorders.

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