Diagnosis of carpal tunnel syndrome

A jointly sponsored AAN/AAEM and AAPM & R practice parameter reviews the literature through December 2000 and sets standards for electrodiagnostic assessment of CTS. The article also provides concise recommendations for future assessment of diagnostic studies for CTS.

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Chang et al. found that wrist–palm motor conduction velocity is more sensitive than wrist–palm sensory conduction velocity in the early diagnosis of CTS, suggesting that sensory fibers are no more susceptible than motor fibers to compression in carpal tunnel. They recommend that in patients with suspected CTS in whom conventional nerve conduction studies are normal, both motor and sensory wrist–palm conduction studies should be carried out to increase the diagnostic yield.

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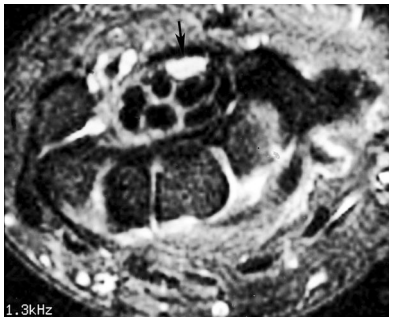
MR for carpal tunnel syndrome



Axial STIR image (swollen and markedly hyperintense median nerve [arrow]).

Jarvik et al. prospectively evaluated the accuracy of high-resolution MR of the carpal tunnel in patients with suspected carpal tunnel syndrome. MR proved highly reliable but only moderately accurate compared to a reference standard that combined electrodiagnostic studies and a hand pain diagram.

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The editorial by Fleckenstein and Wolfe accompanying these three papers on CTS considers the relative value of neurophysiologic studies vs MRI for CTS diagnosis. Reviewing clinical settings where MRI will provide valuable data on anatomic abnormalities of peripheral nerve—tumor, arthropathies, developmental anomalies, and following unsuccessful surgery—they conclude that at best MRI falls short of electrodiagnostic assessment of CTS and is more expensive. MRI of peripheral nerve also requires expertise not as widely available as that for electrodiagnostic studies. They also note that the search for a diagnostic test that is 100% sensitive and 100% specific should not necessarily be the goal; in mild cases, conservative management is appropriate and spontaneous improvement is possible, so that some degree of diagnostic uncertainty is acceptable.

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The prevalence of frontotemporal dementia (FTD)

Ratnavalli et al. identified/examined 108 cases of early-onset dementia in Cambridgeshire, UK (population 326,019). The age-adjusted prevalence of FTD and AD in the 45- to 64-year age group was the same (15 per 100,000), suggesting that FTD is a relatively common cause of early-onset dementia.

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Accuracy of clinical criteria for FTD: Autopsy assessment

Rosen et al. compared 30 autopsy-proven FTD cases and 30 AD cases and found that five features best distinguished FTD from AD: social conduct disorders, hyperorality, akinesia, and absence of amnesia and absence of a perceptual disorder.

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CSF tau and Aß42 for diagnosis in patients with FTD

Riemenschneider et al. studied CSF tau and Aß42 levels in 34 patients with FTD, 74 patients with AD, and 40 controls. These CSF markers differed between FTD and AD, but contributed mainly to negative predictive value in differential diagnosis.

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The accompanying editorial on FTD by Galasko and Marder points out that the Ratnavalli et al. study gives the first estimate of the frequency of FTD in the community—and that somewhat surprisingly, it is equal in prevalence to early-onset AD. Diagnosis of FTD remains a challenge and although low CSF tau and Aß42 levels can serve to make FTD unlikely, no single or combination of diagnostic tests can establish the diagnosis. Clinical criteria are improving as evidenced by the Rosen et al. report. Prospective studies of both brief office screening criteria and more detailed assessments are now needed to further strengthen diagnostic criteria.

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Cerebral amyloid angiopathy: Impaired cognitive function

Cerebral amyloid angiopathy (CAA) is commonly found in neuropathologic investigations of the elderly, but its functional significance is not known. In a population-based autopsy study, Pfeifer et al. found CAA alone was not associated with antemortem cognitive function, but in combination with AD it was associated with a significant decrement in cognitive function.

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The accompanying editorial by Greenberg notes that vascular amyloid, now recognized as the major cause of intracerebral lobar hemorrhage in the elderly, can cause dementia by vascular deposition of amyloid: amyloid distinct from that of AD tissue, but nonetheless, associated with cognitive loss.

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Nontyphoidal salmonella encephalopathy

Arii et al. report eight children (age 1 to 14 years) presenting with seizures or loss of consciousness caused by noninfectious encephalopathy associated with bacteriologically confirmed salmonellosis. There was no evidence of severe dehydration, sepsis, or endotoxemia. This encephalopathy is a distinctive clinical entry that can be differentiated from Reye's syndrome.

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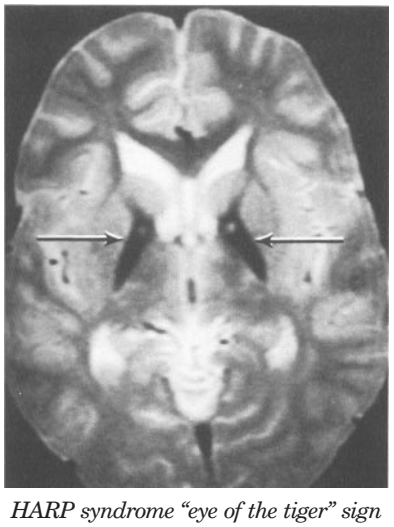
Genetic basis of HARP syndrome



HARP syndrome "eye of the tiger" sign

Ching et al. have identified mutations in the gene mutated in pantothenate kinase associated neurodegeneration (PKAN, formerly Havervorden–Spatz syndrome) in the original patient reported in *Neurology* with hypoprebetalipoproteinemia, acanthocytosis, retinitis pigmentosa, and pallidal degeneration (HARP).

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