## Pain and neurologic complaints in whiplash

Kasch et al. prospectively followed 141 whiplash patients and 40 ankle-injured controls for 1 year postinjury. Headache and neck pain occurred with high frequency but low intensity in both groups. Cognitive and other neurologic complications were two to three times more frequent after whiplash. Persistent disability was infrequent and only seen in whiplash injured.

**see page 743**

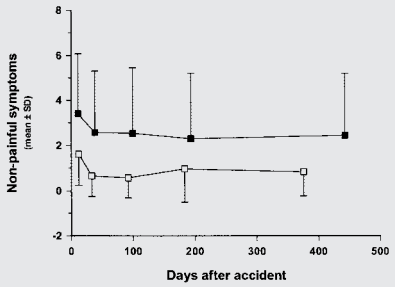
## Whiplash: Chronic pain and cognitive symptoms

*Commentary by Michael P. Alexander*

Whiplash is the most common residual problem after car accidents and accounts for a large percentage of lost work and medical costs. It may be the most common traumatic injury seen in neurologic practice. What do we know about it?

Upward thrust and straightening of the thoracic spine generate axial compression and posterior shear forces that damage cervical facet joints. Stretching injury to cervical muscles and ligaments also contributes to acute pain. Acute whiplash resolves in approximately 80% of patents within 6 months.[1] Optimal treatment is *limited* to nonsteroidal analgesics and early mobilization.[2] Risk factors for persistent pain include very severe initial neck pain, radicular arm pain, age above 60, and perhaps female gender.[3]

The basis for disability months after whiplash is controversial. Chronic pain may be due to inflammation in facet joints;[4] radiofrequency neurotomy may provide lasting relief. It may be due to central hypersensitization,[5] demonstrable even at a distance from the neck; pharmacological treatment may be effective although optimal treatment is not defined. Chronic pain certainly has psychological consequences that may come to dominate the clinical picture (even after ankle sprain), but there is little evidence that chronic whiplash is primarily a psychogenic disorder. Multimodal pain programs that include psychological management may be effective in reducing disability.




*Nonpainful neurologic complaints after whiplash and ankle injury. Whiplash recovered (■); ankle-injured controls (□).*

The report of Kasch et al. confirms much of what we claimed to know above and hints at what we do not. Both acute and chronic whiplash are associated with more regional pain than ankle sprains. There are more cognitive and affective symptoms than with ankle sprain, but excessive inquiry about cognitive symptoms may produce them. Patients with poor recovery from whiplash carry a high load of cognitive and psychological symptoms. Cognitive symptoms do not mean brain damage. Psychological symptoms do not mean malingering. But both are fertile fields for sowing iatrogenesis. Acute whiplash has a good functional prognosis. Intervene lightly, but do not ignore treatment if whiplash becomes chronic.

### References

1. Radanov BP, Sturzenegger M. DiStefano G. Long-term outcome after whiplash injury: a 2-year follow-up considering features of injury mechanism and somatic, radiologic, and psychosocial findings. Medicine 1995;74:281–297.
2. Rosenfeld M, Gunnarson R, Borenstein P. Early intervention in whiplash-associated disorders: a comparison of two treatment protocols. Spine 2000;25:1782–1787.
3. Suissa S, Harder S, Veilleux M. The relationship between initial symptoms and signs and the prognosis of whiplash. Eur Spine J 2001;10:44–49.
4. Lord SM, Barnsley L, Wallis BJ, McDonald GJ, Bogduk N. Percutaneous radiofrequency neurotomy for chronic cervical zygapophysial joint pain. N Engl J Med 1996;335:1721–1726.
5. Curatalo M, Petersen-Felix S, Arendt-Nielsen L, Zbinden AM, Radanov BP. Central hypersensitivity in chronic pain after whiplash injury. Clin J Pain 2001;17:306–315.

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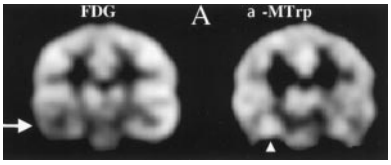
## Serotonin synthesis and receptors in temporal lobe epilepsy




*Images of FDG and α-MTrp PET in a TLE patient with a left-sided focus.*

Toczek et al. used PET imaging to show reduced $5HT_{1A}$ receptor binding in mesial and lateral temporal cortex from patients with temporal lobe epilepsy even when MRI was normal.

**see page 749**

Natsume et al. found an increased hippocampal uptake of α-[$^{11}$C]MTrp/PET in TLE patients with normal hippocampal volumes. Frontal lobe decrease FDG uptake relates to an increase of α-Trp uptake in temporal lobe. Thus there is evidence of the serotonergic system dysfunction and potential localizing value of α-MTrp in TLE patients with normal hippocampal volumes.

**see page 756**

*In an Editorial accompanying these two papers Diane and Harry Chugani suggest that abnormalities in serotonin function demonstrated here as well as their own work reflect functional reorganization in and around the seizure focus. They further suggest that the lack of hippocampal atrophy seen in some patients could reflect serotonin-mediated neurogenesis. Whatever the basis for the abnormalities, these new PET tracers may prove valuable for localization of the seizure focus in patients who do not show hippocampal atrophy on MRI or glucose hypometabolism by PET.*

**see page 736**

## Postmenopausal hormones, caffeine, and Parkinson's disease

Ascherio et al. in a longitudinal study of over 70,000 women found that caffeine may reduce Parkinson's risk only among women who do not use hormone replacement therapy. In contrast, among hormone users heavy coffee drinkers had a four-fold higher risk of Parkinson's than nondrinkers.

**see page 790**

## Heterozygosity for *parkin* gene mutations and age of onset of PD

Foroud et al. found that Parkinson's disease patients with *parkin* gene mutations on both alleles have an earlier age of onset than those patients with only one mutated *parkin* allele. Patients with no *parkin* mutations have the latest age of onset.

**see page 796**

## White matter hyperintensities and cognition: Effect of education

*Cerebral white matter lesions.*

Dufouil et al. found that cerebral white matter lesions were associated with diminished cognitive performances in lower educated persons, whereas in those with higher education no relationship was observed. Thus education may delay the cognitive consequences of vascular insults to the brain.

**see page 831**

*continued on page 735*

## Neurologic complications after allogeneic bone marrow transplantation

Sostak et al. prospectively followed patients after BMT. Neurologic signs developed in 65% over the year after transplantation. Peripheral neuropathy, generally benign, was frequent. A subset of patients developed unexplained cognitive deficits and associated with white matter lesions. Risk factors for this unexplained complication were chronic graft-versus-host disease and immunosuppression.

**see page 842**

## *Aprataxin* mutations: Recessive ataxia without ocular motor apraxia

*Aprataxin* gene mutations have been associated with ataxia and ocular motor apraxia in Portuguese and Japanese patients. Tranchant et al. report three new cases that demonstrates such mutations can be found in other countries and in patients with recessive ataxia without ocular motor apraxia.

**see page 868**

## Peri-ictal water drinking in temporal lobe epilepsy

Trinka et al. evaluated seven patients with peri-ictal water drinking behavior in temporal lobe epilepsy. All had right temporal seizure onset; all became seizure free postoperatively. Peri-ictal water drinking lateralizes seizure onset to the nondominant hemisphere in temporal lobe epilepsy.

**see page 873**