breakdown of acetylcholine, a neurotransmitter in the brain that is important in memory function. On the 18th hospital day, follow-up MMSE and CDR score showed 9 and 2 (sum of box: 11), respectively. Ultimately, the patient discharged on 20th hospital day without improvement of cognition. On 15th day after discharge, the dose of donepezil hydrochloride was increased 10mg/day. About three months later, the patient's cognitive impairment gradually recovered, and the MMSE and CDR score reached 26 and 0.5 (sum of box: 3.5), respectively. But neurological examination showed the persistence of moderate impairment of gait function.

Discussion

The initial acute CO intoxication was followed by a persistent vegetative state[3]. However, in the majority, there was a period of more or less full recovery, followed by an abrupt relapse. During the initial recovery, intellectual and neurological function often was normal, and there was no hint that potentially fatal sequelae were subsequently to occur[3]. After recovery period of usually two to three weeks, the neurological and psychiatric symptoms of delayed encephalopathy may occur, but the exact mechanism of this phenomenon is unclear[2]. Since cerebral cortex, basal ganglia, and hippocampus are very sensitive to hypoxia, the cerebral hypoxia caused by CO intoxication can only account for lesion sites by CO intoxication[2]. However, Gilmer et al reported in their animal study that hyperbaric oxygen treatment is not effective in preventing neurological sequelae and no benefit of hyperbaric oxygen over normobaric oxygen following severe CO neurotoxicity[4].

Nevertheless, Wang et al reported effectiveness of acetylcholinesterase inhibitor for cognitive impairment in delayed encephalopathy, which was unique report about successful management for cognitive symptoms in delayed encephalopathy, to our knowledge[2]. We also experienced that administration of donepezil hydrochloride appeared to be effective in our patient. However, benefit of donepezil hydrochloride for cognitive impairment could be not explained because pathogenesis of delayed encephalopathy was unclear. Recent studies have suggested that donepezil has neuroprotective effects through up-regulation of the anti-apoptotic protein Bcl-2, stimulation of nicotinic acetylcholine receptors, and activation of the phosphoinositide- 3-kinase /Akt pathway and inhibition of glycogen synthase kinase-3, and inhibition of acetylcholinesterase in the cortex and hippocampus of the brain, although the protective mechanisms of donepezil have not yet been clearly identified[5]. Therefore, further studies are necessary to clarify pathogenesis and management of cognitive impairment due to delayed encephalopathy.

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To the Editor

Re: Stereopsis in Drug Naïve Parkinson's Disease Patients. Can J Neurol Sci. 2011; 38:299-302.

I read with interest this report describing impaired stereopsis among drug-naïve parkinson's disease (PD) patients[1]. The authors found an 87.5% prevalence of abnormal stereopsis (as measured by the Titmus stereoacuity test) in PD patients compared to a 10% prevalence among age-matched controls. This difference was both large and statistically significant, and the authors further observed that those PD patients who had abnormal stereopsis had significantly more advanced disease, as measured by the UPDRS (motor) score and Hoehn and Yahr stage, than those PD patients with normal stereopsis. The authors theorize that impaired stereopsis in PD patients may result from dysfunction in the visual association cortex responsible for the binocular representation of 3D surfaces, possibly as a result of dopamine depletion in extrastriate cortical pathways.

I believe there may be a simpler explanation for the authors' findings of impaired stereopsis in PD: convergence insufficiency (CI) .

Convergence insufficiency refers to the impaired ability of the two eyes to adduct simultaneously to center each fovea on a near target. Decreased convergence amplitudes and a distant near point of convergence are the hallmarks of CI[2,3]. Insufficient convergence of the eyes, with consequent decentration of one

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fovea, results in loss of stereopsis and sometimes frank diplopia at near; the two eyes cannot fixate on the same near target to achieve binocular single vision.

The adequate performance of a near visual task, such as the reading of a book or the threading of a needle, requires intact convergence. Convergence insufficiency interferes with the performance of such tasks through the disruption of binocular single vision at near. The Titmus stereoacuity test – a book of plates viewed at 40cm distance with polarized glasses – is also a near visual task, allowing quantification of near stereopsis but not distance stereopsis. Because convergence of the eyes is necessary for viewing the Titmus test and other tests of near stereoacuity, CI may interfere significantly with such measurements of stereopsis.

These observations are relevant to the study by Kim et al[1], because CI is significantly more common among PD patients than age-matched controls and correlates with increasing Hoehn and Yahr disease severity[4,5]. In one study, the prevalence of CI among PD patients was 31%, compared to 0% among controls ($P<0.001$)[5]. Therefore, in the study by Kim et al, it would have been critically important to exclude subjects with CI before comparing the stereoacuity of PD patients to that of controls, especially when studying drug naïve PD patients[1]. It is not clear whether this was done, and therefore the authors' interpretation of their results may be confounded by CI. Because CI is, by definition, a phenomenon that emerges only when viewing a near target, it can easily be overlooked unless specifically sought by: a) examining ocular alignment while the patient views a near target; and b) by measuring the near point of convergence. In fact, ocular alignment and stereopsis may be completely normal when a patient with CI is asked to view a distant target (e.g., a Snellen eye chart or distant fixation light). Although "strabismus" and "ocular motility disturbance" were set as exclusion criteria by Kim et al, none of the seven patients excluded from the study were actually eliminated on these grounds, despite the reported 30% prevalence of CI in PD patients[1].

It would be interesting to repeat the study using a test of distance stereoacuity, which would eliminate altogether the need for intact convergence during stereopsis testing. More robust conclusions about the role of central dopaminergic pathways in stereopsis could then be drawn.

Convergence insufficiency is an underrecognized cause of diplopia and asthenopia in PD patients, often presenting as "difficulty reading" or "tired eyes" when performing near tasks. Symptomatic treatment is easy and generally appreciated by PD patients[5], and it is therefore worthwhile maintaining a high index of suspicion for CI in the PD population. Convergence insufficiency in some PD patients may respond to levodopa[6], but usually CI must be corrected optically using base-in prisms, which are typically either affixed onto or ground into the patient's reading glasses by an optometrist or orthoptist.

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To the Editor

Isolated Recurrent Monocular Vision Loss as a Presentation of Temporal Arteritis

A 73-year-old gentleman was referred to emergency department by his family physician because of a one week history of recurring episodes of monocular vision loss. The episodes were painless and involved the entire visual field of the left eye. Although the episodes had only begun a week ago, they were increasing in frequency and duration. At the time of initial assessment in the emergency department, he estimated approximately eight to ten similar episodes over five to seven days, each lasting anywhere from 10 to 60 seconds in duration before resolving completely without any residual deficits. He denied any other unusual signs, symptoms or focal neurological deficits during the episodes or in between the episodes. He also denied any associated headache, neck pain, jaw pain or jaw claudication.

He denied any obvious precipitating factors for the onset of episodes. He had no significant medical concerns and was on no medications other than aspirin, which was started earlier that week by his family physician when the episodes started. At that time, a referral to stroke neurology was also made, but the accelerating pattern of the episodes necessitated more urgent assessment and investigation. A full functional inquiry revealed no other symptoms and no systemic symptoms apart from some non-specific and diffuse joint aches for several years.

On examination, the patient was afebrile and blood pressure and heart rate were within normal limits. Head and neck exam was normal and there was no scalp tenderness and no

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