LETTERS TO THE EDITOR

Posterior leukoencephalopathy syndrome

EDITOR,—I read with interest the excellent review on posterior leukoencephalopathy syndrome published in January.[1] The author, however, has omitted an important differential diagnosis in his article—namely, progressive multifocal leukoencephalopathy (PML). This can mimic the appearances of posterior leukoencephalopathy on both computed tomography and magnetic resonance imaging (MRI) scans of the brain and needs to be high on the differential diagnosis especially in patients with AIDS.[2] PML was first described in 1958[3] and is characterised by widespread demyelination in the cerebral hemispheres. Today PML is seen most frequently in patients with AIDS but can also occur in patients with chronic neoplasia and immunosuppressed states. Intellectual changes, hemiparesis, visual field defects, ataxia, aphasia, and dementia are clinical features. Seizures are rare. The cerebrospinal fluid is usually normal. On computed tomography there may be low attenuation areas in the posterior fossae but MRI (T2 weighted) shows characteristic increased signal in the posterior fossae. No enhancement is seen after intravenous contrast medium administration. The condition is associated with JC virus and has a very poor prognosis.

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1 Garg RK. Posterior leukoencephalopathy syndrome. *Postgrad Med J* 2001;**77**:24–8.
2 Banerjee AK. *Radiology of AIDS*. Bristol: Clinical Press, 1993.
3 Astrom KE, Marcall EL, Richardson EP. Progressive multifocal leucoencephalopathy. *Brain* 1958;**81**:93.

The author responds:

I am grateful to Dr Banerjee for his interest in my article. I agree with his comment that PML should be included in the differential diagnosis of posterior leukoencephalopathy. Even in patients with PML computed tomography shows hypodense non-enhancing white matter lesions without associated oedema or mass effect. MRI is more sensitive than computed tomography and reveals similar hyperintense signals in the cerebral white matter on T2 weighted spin-echo images. The white matter lesions of PML also have a predilection for occipital and parietal lobes. Occasionally, white matter lesions may symmetrically involve bilateral occipital lobes and may clinically present with cortical blindness; in such patients the clinical and imaging picture is similar to that of posterior leukoencephalopathy.[1] Moreover, both posterior leukoencephalopathy and PML can occur in HIV infected patients and in patients with various lymphoproliferative and myeloproliferative disorders. Certainly in HIV infected patient PML should always be considered as a diagnostic possibility.

As name implies, PML is a progressive disorder, the presenting symptoms include altered mental status, speech and visual disturbances, gait difficulty, hemiparesis, and limb incoordination. The clinical condition deteriorates progressively and the patient dies within six months.[2] In patients with posterior leukoencephalopathy the symptoms develop rapidly, and after treatment the clinical features and imaging abnormalities resolve completely. The characteristic clinical manifestations of posterior leukoencephalopathy include seizures, headache, vomiting, confusional state, visual abnormalities, and infrequently focal motor and sensory neurological deficits. Dr Banerjee has rightly commented that the seizures are infrequent in patients with PML, while in patients with posterior leukoencephalopathy seizures (especially occipital lobe seizures) are dominant and a universal manifestation. Patients with posterior leukoencephalopathy usually have a predisposing cause, the most common being hypertensive encephalopathy, toxaemia of pregnancy, renal diseases, and treatment with cytotoxic and immunosuppressive drugs.

In my opinion PML in patients with an acquired immunodeficiency state can reliably be differentiated from posterior leukoencephalopathy on clinical grounds even if bilateral symmetrical demyelinating white matter abnormalities of parieto-occipital regions are present on neuroimaging.

1 Rickards C, Shepherd DI. Cortical blindness in a 35 years old man. *Postgrad Med J* 1996; **72**:249–51.
2 Cavert W. Viral infections in human immunodeficiency virus disease. *Med Clin North Am* 1997;**81**:411–25.

Smoking and diabetes in Chinese men

EDITOR,—I read with interest the report by Ko *et al* on the association between smoking and diabetes in Chinese men with an odds ratio of 1.7 of smoking on the risk of diabetes.[1]

China is the greatest producer and consumer of cigarettes in the world.[2] The main increase in cigarette consumption in China has taken place only recently: in 1952, 1972, and 1992 the mean consumption among Chinese men was one, four, and 10 cigarettes a day, respectively.[3] Deaths due to smoking will increase from about one million worldwide in 1995 to more than seven million in 2025.[4] In response to comments on their earlier reports on smoking and death in China published in 1998,[3 5] Peto *et al* reported that there are now already a million deaths a year in China alone from smoking.[6] So on present day smoking patterns Chinese tobacco mortality will increase substantially.

Even more alarming is the prevalence of teenage smoking in China. Three of every five Chinese smokers begin smoking at the age of 15–20 years, and cessation is rare.[7] Teenage smoking is increasingly becoming a health problem in modern China. About 200 million children living today in China will become regular smokers. Of these, about 50 million, or one quarter, will die prematurely of smoking related illness.[8] The association of smoking and diabetes reported by Ko *et al*[1] with the attendant complications of diabetes will most likely increase further this number.

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1 Ko GTC, Chan JCN, Tsang LWW, *et al*. Smoking and diabetes in Chinese men. *Postgrad Med J* 2001;**77**:240–3.
2 Office on Smoking and Health. *Smoking, tobacco, and health: a fact book*. Rockville, Maryland: Department of Health and Human Resources, PHS, 1986.
3 Niu S-R, Yang G-H, Chen Z-M, *et al*. Emerging tobacco hazards in China: 2. Early mortality results from a prospective study. *BMJ* 1998;**317**: 1423–4.
4 Yang G, Fan L, Tan J, *et al*. Smoking in China. Findings of the 1996 National Prevalence Survey. *JAMA* 1999;**282**:1247–53.
5 Liu B-Q, Peto R, Chen Z-M, *et al*. Emerging tobacco hazards in China: 1. Retrospective proportional mortality study of one million deaths. *BMJ* 1998;**317**:1411–22.
6 Peto R, Chen Z-M, Boreham J. Emerging tobacco hazards in China: double standards applying with importation of tobacco into developing countries—UK authors' reply. *BMJ* 1999;**318**:1555.
7 Cheng TO. Teenage smoking in China. *J Adolesc* 1999;**22**:607–20.
8 Novotny TE, Peto R. Estimates of future adverse health effects of smoking in China. *Public Health Rep* 1988;**103**:552–3.

BOOK REVIEWS

The reviewers have been asked to rate these books in terms of four items: readability, how up to date they are, accuracy and reliability, and value for money, using simple four point scales. From their opinions we have derived an overall "star" rating: * = poor, ** = reasonable, *** = good, **** = excellent.

The Effectiveness of Continuing Professional Development. J Grant and F Stanton. (Pp 39; £12 non-members, £10 members.) Association for the Study of Medical Education, 2000. ISBN 0-9044-73260.****

This publication saw life initially as a report on continuing professional development (CPD) to the Chief Medical Officer. It gives the background to the nature of CPD, its prevalence, types, aims, the educational approaches, and the factors that influence its provision and the participation in CPD programmes. The next section deals with the methodological issues. These include the design of programmes and the assessment of outcomes. The main part of the report is devoted to a review of the literature up to 1997.

There is recognition of the importance, reflected in its prevalence, of self directed learning and of how individuals will largely initiate, control, and evaluate their own continuous learning. This is a difficulty for professional bodies who award credits for more formal learning experiences. The assessment of outcomes is very difficult and falls far short of being able to measure health care benefits, which is what government wants for its financial investment in CPD. There is also no best learning method. The authors conclude that the effectiveness of CPD is a function of the process and the context in which it occurs. Horses for courses!

The authors do well to avoid the all too frequent opacity of language that makes educational papers so difficult for the general