have an urgent EEG, they recognised that the clinical features were not an alternative to EEG in the diagnosis of NCSE [7]. Furthermore, in patients with prolonged confusion after convulsions, an EEG is the unique test able to distinguish between postictal encephalopathy and generalised NCSE [8].

Although I agree with Cooper and Feely that an urgent EEG may not be readily available, this obstacle does not mean that we have to forget that an EEG is the method of choice in the diagnosis and classification of NCSE. Frequently, the episodes of NCSE are prolonged and recurrent, and on many occasions the diagnosis may be obtained in hours where the clinical neurophysiologists are available. The essential message is the need to keep this diagnostic possibility (NCSE) in mind when evaluating confused older patients. In our hospital, neurophysiologists are on call for only two situations: the diagnoses of brain death and NCSE. Although this intermediate solution is not ideal, it may contribute to the early identification and treatment of those patients presenting symptoms of NCSE.

Obviously, the interpretation of the electroencephalographic abnormalities in NCSE is complex even for trained experts. Nevertheless, this does not mean that we have to doubt the role of EEG in the diagnostic confirmation of NCSE. Thus, a precise diagnosis should be based on a high level of clinical suspicion and an accurate EEG interpretation. Although intravenous benzodiazepines were acutely administered during the EEG recording, all our patients were treated with classical antiepileptic drugs (valproate, phenobarbital and phenytoin). It is routine practice to use intravenous benzodiazepines when requiring a rapid effect in the diagnostic confirmation and treatment of NCSE [4, 9]. Thus, a recent review confirmed that benzodiazepines are the first-line drugs in the treatment of NCSE [10]. In addition, a full knowledge of the metabolic status and previous therapy of the patients and the possibility of a delayed or tardy benzodiazepine response should also be kept in mind [11].

**Clinical experience with high success rate of antiretroviral therapy in elderly HIV-infected patients**

SIR—Before the highly active antiretroviral therapy (HAART) era, advanced age itself was described as a risk factor for progressive HIV disease and increased morbidity and mortality [1]. As HAART has become available, morbidity and mortality have markedly decreased [2]. However, limited data are available on antiretroviral therapy in the elderly, and rare controlled studies included patients aged 265 years [3]. We conducted a retrospective cohort study to evaluate the safety/tolerability and efficacy of HAART in HIV-infected patients aged ≥65 years in 2001 and followed up for 2 years. There were 10 patients with a mean age of 68.0 ± 3.9 years and 60% male. Heterosexual contact was the means of acquisition of HIV infection in all patients. Of these, four presented with tuberculosis and one presented with cryptococcal meningitis. The others were diagnosed HIV infection after the diagnosis in their spouses. Diabetes mellitus was co-morbidity in two patients. At diagnosis, mean CD4 cell count was 165.4 ± 115.1 cells/µl and eight patients had HIV RNA >100,000 copies/ml. All patients had received HAART within 3 months of the diagnosis of HIV infection. The majority of the HAART regimens (70%) were non-nucleoside analogue reverse transcriptase (NNRTI) based. Mean CD4 cell counts at 3, 6, 9, 12 and 24 months of treatment were 259.9, 233.4, 255.2, 259.4, and 273.4 cells/µl, respectively. The percentage of patients who achieved undetectable HIV RNA (<50 copies/mL) at the corresponding time were 80%, 90%, 90%, 100% and 100%, respectively. One patient developed anemia due to zidovudine and another patient could not tolerate indinavir; both patients needed one-drug substitution and could continue their new regimens. No patient needed to discontinue HAART.

We have shown that the majority of patients were diagnosed in the stage of advanced disease, with 70% having a CD4 cell count <200 cells/µl and half had been diagnosed after the occurrence of major opportunistic infections. Our findings are similar to previous studies in that the diagnosis in elderly patients is made at the late stage [3, 4]. Elderly patients...
Adequately powered prospective studies in populations at risk, such as the elderly, are needed. (iii) The dissemination of (at least) misleading information is one of the important mistakes made by the pharmaceutical industry.

We disagree with the authors that (i) ‘In the development of the coxibs, potential problems with the cardiovascular, renal and other systems affected by prostaglandins were ignored’. In contrast, renal side-effects, small bowel and colon toxicity, cardiovascular effects, interactions with aspirin and NSAID-induced asthma were neglected for decades and ‘discovered’ in the development of the selective COX-2 inhibitors [1–4]. In particular, the cardiovascular risk was and currently is intensely discussed, despite no further evidence for a higher rate of cardiovascular thrombotic events by specific COX-2 inhibitors so far [1]. (ii) ‘Additional concerns include worries about renal toxicity’. There is no evidence for a special renal risk by coxibs. Coxibs cause sodium and water retention and inhibit renal renin release to the same extent as COX-unselective NSAIDs [2]. With regard to renal perfusion they offer a small, yet clinically irrelevant, advantage [2]. The real problem is the marketing-driven perception of coxibs as generally safer than conventional NSAIDs, leading to prescription of coxibs to patients with a risk for NSAID-induced renal failure, which should not be treated with NSAIDs at all [2]. (iii) ‘Coxibs are relatively new. We still do not know the full extent of their potential for benefit or harm’. Despite many open questions selective COX-2 inhibitors are better studied than any of the other NSAIDs previously [4]. Significantly, most of the data on conventional NSAIDs, in particular ibuprofen, naproxen and diclofenac, stem from trials with selective COX-2 inhibitors where those drugs served as active comparators.

In summary, the conclusion that coxibs should be avoided in the elderly is not justified. In accordance with the NICE guidance [5] we recommend a sensible use of coxibs in elderly and non-elderly patients.

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