or loss of selectivity for monoamine oxidase type B resulting in excess plasma tyramine. However, in this study selegiline seemed to cause supine hypertension as well as orthostatic hypotension. This combination would be consistent with either an amphetamine or a tyramine effect.

The failure of patients with severe orthostatic hypotension induced by selegiline and levodopa to develop a compensatory tachycardia indicates impairment of cardiovascular reflexes¹ and as such is consistent with significant, albeit reversible, drug toxicity by whatever mechanism.

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- Churchyard A, Mathias CJ, Lees AJ. Autonomic effects of selegiline possible cardiovascular toxicity in Parkinson's disease. *J Neurol Neurosurg Psychiatry* 1997;63:228-34.
- Turkka J, Suominen K, Tolonen U, et al. Selegiline diminishes cardiovascular autonomic responses in Parkinson's disease. *Neurology* 1997;48:662-7.
- Johnson RH. Aging and the autonomic system. In: Bannister RB, Mathias CJ, eds. *Autonomic failure. A textbook of clinical disorders of the autonomic nervous system*. 3rd ed. Oxford: Oxford University Press, 1992.
- McDaniel KD. Clinical pharmacology of monoamine oxidase inhibitors. *Clin Neuropharmacol* 1986;9:207-34.
- Waldmeier PC, Felner AE. Deprenil: loss of selectivity for inhibition of B-type MAO after repeated treatment. *Biochem Pharmacol* 1978;27:801-2.
- Ekstedt B, Magyar K, Knoll J. Does the B form selective monoamine oxidase inhibitor lose selectivity by long term treatment? *Biochem Pharmacol* 1979;28:919-23.
- Gelowitz DL, Richardson JS, Wishart TB, et al. Chronic 1-deprenyl or 1-amphetamine: equal cognitive enhancement, unequal MAO inhibition. *Pharmacol Biochem Behav* 1994;47:41-5.
- Riederer P, Youdim MBH, Rausch WD, et al. On the mode of action of 1-deprenyl in the human nervous system. *J Neural Transm Gen Sect* 1978;43:17-26.
- Reynolds GP, Elsworth JD, Blau K, et al. Deprenyl is metabolised to metamphetamine and amphetamine in man. *Br J Clin Pharmacol* 1978;6:542-5.
- Gill JR, Mason DT, Bartter F. Effects of hydroxy amphetamine on the function of the sympathetic nervous system in normotensive subjects. *J Pharmacol Exp Ther* 1967;155:288-95.

Idiopathic generalised epilepsy in adults manifested by phantom absences, generalised tonic-clonic seizures, and frequent absence status

I read with interest the report by Panayiotopoulos *et al.*¹ Their series recalled a 58 year old man who presented for evaluation after several episodes presumed to be complex partial status epilepticus. His neurological history was remarkable only for two convulsions in his late teens which led to his discharge from the armed forces. He did not recall whether these seizures were related to sleep deprivation or alcohol withdrawal. He did not recall being informed of seizures, staring spells, or other paroxysmal events as a child. He denied a family history of seizures or other neurological diseases. He was treated briefly with phenytoin, but was free of seizures and took no anticonvulsant drugs for almost 40 years, when he began to experience episodic confusion lasting for hours. Interictal EEGs were reported to show spike wave discharges. Brain MRI was normal. He was treated with various combinations of phenytoin, carbamazepine, phenobarbitone, and primidone, which did not prevent these confusional episodes. Video EEG monitoring

after withdrawal of all antiseizure medications showed frequent bursts of 3-3.5 Hz spike wave activity. Hyperventilation produced clinical and electrographic seizures lasting ≥ 8 minutes at a time, and terminating spontaneously. The patient became confused, was unable to follow simple commands, answer questions, or repeat simple phrases. Electrographically, these seizures consisted of bisynchronous, frontally predominant spike wave discharges which began at 3.5 Hz but slowed to 2.5 Hz over several minutes. At the end of each seizure, the patient was immediately alert and able to follow commands, converse, and repeat. Treatment with divalproex (750 mg/day) abolished the seizures and the interictal EEG abnormalities for four years. The patient did not wish to attempt withdrawal from divalproex, and was returned to the care of his family physician.

I would like to ask the authors whether their patients manifested an EEG or clinical response to hyperventilation.

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- C P Panayiotopoulos, M Koutroumanidis, S Giannakodimos, et al. Idiopathic generalised epilepsy in adults manifested by phantom absences, generalised tonic-clonic seizures, and frequent absence status. *J Neurol Neurosurg Psychiatry* 1997;63:622-7.

Panayiotopoulos *et al* reply:

We thank Bleck for vividly illustrating the challenging diagnostic and management problems of these patients that were among the main purposes of our report. His patient, like ours, had infrequent generalised tonic clonic seizures with onset in the late teens and frequent episodes of absence status that was misdiagnosed as complex partial status epilepticus. Also, he probably had phantom absences during interictal generalised 3-3.5 Hz spike and slow wave discharges but this needs documentation with breath counting or other means of testing for mild impairment of cognition. Similarly to our cases, he was also erroneously treated with phenytoin, carbamazepine, and barbiturates for many years and seizures stopped only when appropriate medication of divalproex was prescribed. He was fortunate not to receive vigabatrin, which exacerbates absences and absence status.

The only difference from our cases is that Bleck's patient had prolonged, 8 minute, severe absence seizures provoked by hyperventilation after withdrawal of all antiseizure medication. Although briefer, this was similar to the absence status in our patients; this severe electroclinical state was documented only during spontaneous absence status, not during hyperventilation. However, in all patients over-breathing increased the generalised 3-4 Hz spike or polyspike and slow wave discharges that were often associated with mild impairment of cognition, shown with errors of breath counting. Hyperventilation was the only means of inducing generalised 3-4 Hz spike and slow wave discharges in some of the patients whose resting EEG could be normal. This is in accordance with the provoking effect of hyperventilation on absences of the idiopathic generalised epilepsies.

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BOOK REVIEWS

The Neuropathology of Dementia.

Edited by MARGARET M ESIRI and JAMES H MORRIS. (Pp 440; £75.00.) Published by Cambridge University Press, Cambridge. 1997. ISBN 0-521-43311-8.

This distinguished pair of Oxford neuropathologists offer a new book to the "pathologist faced with the task of determining the cause of dementia in cases requiring post-mortem diagnosis". They are credited with editorship; actually, they have written 11 chapters and two appendices, among them those chapters which deal with the pathological processes which account for 90% of adult dementia (Alzheimer's disease, vascular disease, and Lewy body disease). For the remaining eight chapters and appendix, Professor Esiri and Dr Morris recruited acknowledged experts.

After an introductory chapter on dementia, which describes the clinical syndrome and describes its neuroanatomical basis, there are two chapters in which Esiri and Morris define the practical approach to the pathological diagnosis of dementia. The first of these chapters describes (and illustrates diagrammatically) the neuroanatomical structures which are particularly pertinent to cases of dementia. The diagrams of neural connectivity in the hippocampus and neocortex may daunt the general pathologist, but the chapter provides a useful summary of pathological processes which affect each anatomical region of the brain; that is to say, it offers a regional approach to the differential diagnosis of dementia. The second of these chapters gives instruction on the conduct of the post-mortem in a case of dementia, details the observations which should be made on the unfixed brain at the time of postmortem, and on the fixed brain, and provides a scheme for the selection of tissue blocks and histological stains.

The rest of the book is composed of chapters which are dedicated to individual causes of dementia, starting with the commonest diseases: Alzheimer's disease, vascular disease, and Lewy body disease. The very numerous rare causes of dementia are all discussed in subsequent chapters, which include adequately comprehensive and up-to-date accounts of aetiology and pathogenic mechanisms, but manage to retain the practical perspective; a mark of successful editorship. I should emphasise that this book is not simply a practical manual although it largely succeeds in that respect. It succeeds also in taking a common sense approach to issues which are controversial among neuropathologists. The general pathologist who turns to this book will receive sound advice on such matters as the pathological diagnostic criteria of Alzheimer's disease and its distinction from age related changes, on the relation of head injury to the pathogenesis of Alzheimer's disease, and on the range of clinical and pathological phenotypes which are included