Neuro-ophthalmology Update Edited by J. Lawton Smith. (Pp. 396; illustrated; £37.75.) Masson Publishing: New York, Abacus Press: Tunbridge Wells. 1978.

This book is the record of a symposium organised by the University of Miami in 1977. Fifty-eight authors have contributed 40 papers, each with a list of references. The contributors include ophthalmologists, radiologists, neurologists, and neurosurgeons, of whom only three are not Americans.

The book is directed at clinicians and offers a useful refresher course in neuro-ophthalmology. Certain topics are covered in a quite memorable way—for example, pathological lid retraction; the six syndromes of the sixth nerve; tumours of the optic nerve, and especially the impossible meningioma syndrome (which quotes Walsh's dictum that "progressive unilateral loss of vision without apparent ophthalmoscopic cause is due to a compressive lesion of the optic nerve"); and the ocular symptomatology of sellar aneurysms.

It is rather disappointing to the neurologist, however, to find no mention of the clinical importance of visual evoked responses, but there is an attractively brief chapter (the last) on commonsense in retinal function testing, pleading that "best results come from simple underinterpreted tests performed by the primary physician." It is an even greater disappointment to have no less than six of the 40 chapters devoted to CAT scanning, with inevitable redundancy and overkill. No one now doubts the value of this method of investigation, especially the high resolution cuts for the orbits.

Dr J. Lawton-Smith, an editor of commendable style who insists on clarity and brevity, has produced an interesting and stimulating book which can be recommended immediately to all neurologists and neurosurgeons who have a special interest in neuro-ophthalmological work.

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Letter

Epileptic seizures precipitated by atenolol

SIR,—The central nervous system side effects of beta-adrenoceptor antagonists have included vivid dreaming, insomnia, depression, hallucinations, and the acute organic brain syndrome (Prichard and Gillam, 1964; Stephan, 1966; Waal, 1967; Hinshelwood, 1969; Voltolina *et al.*, 1971; Tyrer and Lader, 1973). We report the occurrence of convulsions precipitated by the oral administration of atenolol (Tenormin).

The patient is a 28 year old man who was briefly unconscious after a head injury at age 16 years. At age 25 years he was admitted to hospital for encephalitis, after which he suffered from attacks of diffuse pulsating headache, reduced memory and concentration, and persistent clumsiness of his right hand. On examination in September 1977 he had reduced memory and concentration, a 4–5 Hz tremor of his right hand, cogwheel rigidity and bradykinesia of the right upper extremity. There was no history of epileptic attacks. Renal and liver function tests, skull radiographs, EEG, and cerebral CAT scan were normal. The rigidity, tremor, and bradykinesia were improved by L-dopa. Frequent, severe headache attacks continued to be his chief complaint, the only preparation which had given slight improvement being propranolol, in a dosage of 40 mg thrice daily. On this medication, however, he became hypotensive and experienced nightmares. We, therefore, decided to attempt treatment with another beta-blocking drug, atenolol. At this time his only medication was L-dopa in a dosage of 3 g daily. On the first day he received 100 mg of atenolol orally. That evening he complained of drowsiness and an odd feeling similar to alcoholic intoxication. Pulse and blood pressure were normal. On the second day he received 50 mg at 1100. At noon he complained of nausea, vertigo, and twitching in both legs. By 1215 he was drowsy with involuntary movements in all extremities. At 1230 he began to have attacks of tonic/clonic seizures which lasted approximately three minutes. During the next two hours he had 10–15 such attacks, and was stuporous. Pulse rate, blood pressure, and blood sugar level were normal. An EEG during the seizures showed bilateral slow waves with sharp components. Attempts to control the convulsions with diazepam were unsuccessful. The attacks subsided by 1530, and the level of consciousness improved. Control EEGs and CAT scan were normal. After discontinuation of atenolol, all symptoms which occurred during its use disappeared.

The close temporal relationship between the occurrence of several symptoms, including convulsions, and the administration of atenolol strongly suggests that atenolol played a role in the development of these symptoms in our patient with preceding encephalopathy. Until more is known about atenolol caution should be exercised in the use of this drug when there is a risk of precipitating epileptic attacks.

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Notice

The Seventh Annual Meeting of the International Society for Paediatric Neurosurgery will be held in Chicago from 16–19 September 1979. The meeting is open to readers interested in attending or in presenting a paper. Further details may be obtained from Dr Francisco A. Gutierrez, Children's Memorial Hospital, 2300 Children's Plaza, Chicago, Illinois 60614, USA.

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