between symmetric basal ganglia calcifications and psychiatric or neurological symptoms, as no consistent associations regarding aetiology, localisation, volume, or symptoms have been ascertained.1 Patients with basal ganglia calcifications are, however, reported to be exceptionally vulnerable to psychosocial and traumatic conditions.2 Thus an increased vulnerability due to increased thalamicorrtical drive associated with basal ganglia calcifications and an abnormal excitability of neurons due to the electrolyte imbalance might well have brought forth the acoustic phenomena in our patient. There has been one previous case report describing a patient with bilateral basal ganglia calcifications who developed a chronic progressive neurological syndrome of extrapyramidal and cerebellar symptoms, pyramidal signs, and epilepsy 32 years after thyroidectomy, which improved partly after adequate treatment for hypoparathyroidism.1 In comparing this report with that of the present patient, differences in the clinical syndrome might well be explained by differing regions of involvement due to the distribution of calcifications.

In terms of epidemiology, no association between basal ganglia calcifications and particular neuropsychiatric symptoms could be established.1 Extensive basal ganglia calcifications in these two patients, however, may be able to interact with the effects of non-specific noxious conditions, such as electrolyte imbalances and determine their psychological symptoms.

Although it is uncertain whether basal ganglia calcification progression can be stopped by adequate treatment of hypoparathyroidism, our patient shows that both psychopathological and neurological symptoms can be improved. Therefore, prescribing "antipsychotic" drugs should be avoided due to the increased vulnerability to extrapyramidal side effects in patients with basal ganglia calcifications and the likelihood of neuroleptic non-response in this type of auditory hallucination.2

Dr Feltner, Bad Windsheim, kindly permitted publication of CT scans.

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Announcement from the British Neuro-psychiatry Association

The 1995 Summer meeting—to include joint sessions with the British Association for Psychopharmacology—will be held on 15–17 July in Cambridge

On 16 July BNPA will hold a scientific meeting with the theme of “movement disorders” and its AGM. On 17 July BNPA/BAP will have a joint session on neuroimaging, psychiatry, and psychopharmacology. Short scientific papers and single case videos by members of both associations will also be presented. For further details please contact Ms Sue Garratt, 17 Clocktower Mews, London N1 7BB, UK.

For details of membership of the BNPA, which is open to medical practitioners in psychiatry, neurology, and related clinical neurosciences, please contact Sue Garratt at the address above, or Dr Jonathan Bird, Burden Neurological Hospital, Stoke Lane, Stapleton, Bristol BS16 1QT, UK.

BOOK REVIEWS

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The study of brain function and brain-behaviour relationships is addressed by fields as disparate as neuropsychology, neurophysiology and neuroimaging. This book aims to introduce the newcomer to experimental techniques currently available in these areas, and also to help current researchers keep abreast of recent developments in their field.

A wide range of research areas are covered, each chapter being written by a researcher familiar with both experimental and clinical neuropsychology. Areas covered span the spectrum from simple pen-and-paper neuropsychological tests to high-tech fields such as evoked potentials and cerebral blood flow imaging.

Given the rapid advances in cognitive neuropsychology, it is not surprising that the book, written in 1986, is showing its age in this field. The chapter on memory suffers particularly in this respect. More “neurological” research areas such as evoked physiological research areas. Although the book will be of use in directing researchers to more definitive articles. It is less successful in its second aim of providing researchers with an account of recent advances in the field and in keeping up to date; it only occasionally, libraries in neuropsychological research institutes may find it a useful investment.

JOHN GREENE


Bailliere’s Clinical Neurology series, a recently launched sittership to the well established and excellent Neurologic Clinics, has reached only seven or eight issues, but has already established not only an individual personality, but also a reputation for thoroughness and accuracy. This year’s second monograph, Inflammatory Neuropathies, edited by Professor McLeod (Sydney) is an outstanding edition.

It is little that has remained static over the last few years in clinical neuro-science or consequently neurological practice. The study of peripheral neuropathies, and in particular of inflammatory diseases of the peripheral nerve, is no exception. Progress in our understanding of electrophysiological patterns of neuropathy have marched hand in hand with advances in immunoneurology; new strategies for immunological therapies have very closely followed. A single text straddling and drawing together these areas is timely and welcome.

The layout is clear and the organisation readily mastered. The opening chapters authoritatively review the pathology, neurophysiology, and immunology of the inflammatory neuropathies, and the trial of authors (Prineas, Sumner, and Hughes respectively) would be hard to better. Clinical accounts of the Guillain-Barré syndrome, its variants, of CIDP and of para-proteinaemic neuropathy, plus focused chapters on neuropathies related to infection, inflammatory plexopathies, and vas-