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 metabolic and traumatic conditions.[2] Thus an increased vulnerability due to increased thalamocortical 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 partially after adequate treatment of the hypoparathyroidism.[5] In comparing this report with that of the present patient, differences in the clinical syndrome might well be explained by differing regions of increased vulnerability 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 psychopathological 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 hallucinosis.[2]

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

N WODARZ
T BECKER
J DECKERT
Department of Psychiatry, University of Würzburg, Germany

Correspondence to: Dr N Wodarz, Füchsleinstrasse, 15, 97080 Würzburg, Germany.

1 Förstl H, Krumm B, Eden S, Kohlmeyer K. Neurological disorders in 166 patients with basal ganglia calcification: a statistical evaluation. *J Neurol* 1992;**239**:36–8.
2 Paquier P, van Vugt P, Bal P, *et al.* Transient musical hallucinosis of central origin: a review and clinical study. *J Neurol Neurosurg Psychiatry* 1992;**55**:1069–73.
3 Sugar O. Central nervous system complications of hypoparathyroidism. *Arch Neurol Psychiatry* 1953;**70**:86–92.
4 Alexander GE, Crutcher MD. Functional architecture of basal ganglia circuits: neural substrates of parallel processing. *Trends Neurosci* 1990;**13**:266–71.
5 Kartin P, Zupevc M, Pogacnik T, Cerk M. Calcification of basal ganglia, postoperative hypoparathyroidism and extrapyramidal, cerebellar, pyramidal motor manifestations. *J Neurol* 1982;**227**:171–6.

MATTERS ARISING

Chronic fatigue syndrome

Chronic fatigue syndrome: a follow up study by Bonner *et al*[1] reported that 47 patients initially diagnosed with "chronic fatigue" were contacted for follow up four years later. The authors indicated that "These patients were initially assessed before the current criteria for chronic fatigue syndrome became available, but most would have satisfied the criteria retrospectively" (p 617). At the outset, all patients were offered cognitive behavioural treatment and some were offered antidepressant medications. Each patient then made a decision to either undergo or decline cognitive behavioural treatment. Four years later, those patients who reported functional improvement were more likely to have elected to receive the cognitive behavioural treatment. Additionally, patients in the group that did not report any functional improvement were more likely to score higher on measures of depression.

The US Centers for Disease Control and Prevention (CDC) case definition,[2] the proposed revisions to the CDC case definition,[3] and the guidelines for research set forth by Sharpe *et al*[4] were cited, but the researchers did not make it clear as to which criteria were used to diagnose which patients. Thus it is unknown whether uniform criteria were applied to diagnose all patients at the outset. Moreover, the authors did not specify just how many of the initial 47 patients met any of the cited criteria for chronic fatigue syndrome, as opposed to chronic fatigue. In short, they did not differentiate the exact number of chronic fatigue syndrome *v* chronic fatigue cases.

Only 29 of the original 47 patients (62%) agreed to be interviewed for the follow up. Thus 18 (38%) of the original patients were not included in the outcome data, where 10 subjects reported little or no improvement and 19 subjects reported improvement or recovery. The authors acknowledged that the small patient sample size constituted a methodological shortcoming, but nevertheless concluded "that there is a strong association between successful completion of [cognitive] treatment and the absence of functional disability at the four year follow up" (p 620). They further suggest that costs associated with long term disability could be reduced by the utilisation of cognitive therapy in the treatment of chronic fatigue syndrome. We would like to emphasise that the small patient sample size, together with the lack of availability of almost 40% of the initial patients for interview at follow up, make such conclusions highly inappropriate.

There are other possible explanations for the results found in this study. Because of the ambiguity regarding the diagnostic criteria that was employed, it is uncertain whether the two groups of patients came from the same population. Perhaps those who elected to have cognitive behavioural treatment were at a different stage of their illness and recovery than those who refused this treatment. It is also possible that subjects who opted for cognitive treatment had evidence that they were not coping effectively with life. Additionally, it is possible that subjects who refused cognitive therapies might have had more evidence of a physiological illness so severe that they were not physically well enough to engage in cognitive therapies.

Although the researchers reported that no neurological or physical illnesses developed over four years, their diagnostic methods were not specified, other than references to telephone contact with physicians of non-respondents. This leaves open the question of whether any illnesses that might have developed were missed. Based on the information in the article, it does not appear that comprehensive testing for any physical illness was part of the research protocol.

We find the report of no new neurological or physical illnesses after four years surprising, as two of the writers (AVP, SP), in their own chronic fatigue syndrome related research, found that in the first 75 patients evaluated for fatigue, 50 had chronic fatigue syndrome and 28% had diagnoses of curable illnesses. In this presently ongoing research, also, four patients with chronic fatigue syndrome developed concurrent illnesses such as hypothyroidism, hyperthyroidism, and diabetes.

Bonner *et al*[1] construe their finding of greater psychiatric morbidity at the follow up in the group that did not report any functional improvement as evidence that the "prognosis of severe chronic fatigue syndrome appears to be associated with psychiatric morbidity and in particular depression" (p 620). We, however, view these findings as evidence of reactive depression in the group that did not improve, as there were no significant differences between the two groups on the Beck depression inventory or hospital anxiety and depression questionnaire four years earlier.

The presence of depression in any clinical situation needs to be considered and treated, whether the patient has chronic fatigue syndrome, coronary disease, cancer, or any other illness. The suggestion, however, that symptom reporting by patients with an illness for which there is not yet available any reliable diagnostic laboratory measure will only deflect the attention of their physicians (p 621) is not only highly unfair and unfounded, but it can also potentially hinder the medical support that these patients require.

Cognitive behavioural treatment might, in the end, be deemed to hold a place in the treatment of chronic fatigue syndrome. It is important, however, to note that cognitive behavioural treatment over an extended period can be extremely expensive, and the benefit of such treatment must be carefully assessed before any generalisations regarding its use are made. We very much hope that future studies on this issue will apply uniform criteria to diagnose patients, include adequately large numbers of patients, and randomly assign patients to various blinded treatment groups.

DAVID M LIPKIN
RUTH ROBIN
LAURA VASQUEZ
Chronic Fatigue Syndrome Society of Illinois Inc
AUDRIUS V PLIOPLYS
SIGITA PLIOPLYS
The Chronic Fatigue Syndrome Center, Mercy Hospital and Medical Center

Correspondence to: Dr David M Lipkin, Chronic Fatigue Syndrome Society of Illinois Inc, PO Box 10139, Chicago, IL 60610, USA.

1 Bonner D, Ron M, Chalder T, Butler S, Wesley S. Chronic fatigue syndrome: a follow up study. *J Neurol Neurosurg Psychiatry* 1994;57:617–21.
2 Holmes G, Kaplan J, Gantz NM, *et al.* Chronic fatigue syndrome: a working case definition. *Ann Intern Med* 1988;**319**: 172+6–28.
3 Schluederberg A, Straus SE, Peterson P, *et al.* Chronic fatigue syndrome research: definition and medical outcome assessment. *Ann Intern Med* 1992;117:325–31.
4 Sharpe MC, Archard LC, Banatavala JE, *et al.* A report-chronic fatigue syndrome: guidelines for research. *J R Soc Med* 1991;84: 118–21.

Bonner et al reply:

We would like to respond to some of the questions that Lipkin *et al* have made in response to our follow up of patients with chronic fatigue syndrome.

As we stated, our study began before the current operational criteria were introduced. Retrospectively, all would have fulfilled Oxford criteria and as far as we can tell nearly all would have fulfilled the 1988 US Centers for Disease Control (CDC) criteria. (We did not routinely record the physical criteria, and these have now been discredited.)

Lipkin *et al* are correct to state that this was a non-randomised trial of cognitive behaviour therapy. A randomised trial has now been completed and will be reported shortly. All we purport to show in the paper by Bonner *et al* is that the benefit of cognitive behavioural therapy in an uncontrolled study does seem to be stable over time and that spontaneous improvement in the non-treated group did not occur. We agree that data from non-randomised studies must be interpreted with extreme caution, but at least we have shown that something can be done. It is for other studies to determine what, when, and how.

Lipkin *et al* point out that patients who refuse cognitive treatment may have had more evidence of physiological illness. All the patients who participated in this study were extensively investigated by neurologists at Queen Square. Most had also been extensively investigated elsewhere and the chances of any other disease process presenting itself must be regarded as slight. We agree that cognitive behaviour therapy is expensive and that it requires skilled personnel. Some 12–16 sessions of treatment, however, in terms of the reported costs to society of chronic fatigue syndrome,[1] do not seem excessive to us. We think that our finding adds to the consistency of published work on outcome in chronic fatigue syndrome. It seems that the best determinant of long term outcome is the strength of adherence to a solely physical model.[2 3] Cognitive behavioural therapy aims to show that disability in chronic fatigue syndrome is more complex and can be best understood, and hence alleviated, by considering physical, social, and psychological factors. We hope that this message will be disseminated to those with chronic fatigue syndrome in Illinois.

D BONNER
M RON
T CHALDER
S BUTLER
S WESSELY

1 Lloyd A, Pender H. The economic impact of chronic fatigue syndrome. *Med J Aust* 1992; 157:599–601.
2 Sharpe M, Hawton K, Seagroatt V, Pasvol G. Follow up of patients with fatigue presenting to an infectious diseases clinic. *BMJ* 1992; 305:347–52.
3 Wilson A, Hickie I, Lloyd A, Hadzi-Pavlovic D, *et al.* Longitudinal study of the outcome of chronic fatigue syndrome. *BMJ* 1994; 308:756–60.

NOTICE

Announcement from the British Neuropsychiatry 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

All titles reviewed here are available from the BMJ Bookshop, PO Box 295, London WC1H 9TE. Prices include postage in the United Kingdom and for members of the British Forces Overseas, but overseas customers should add £2 per item for postage and packing. Payments can be made by cheque in sterling drawn on a United Kingdom bank, or by credit card (Mastercard, Visa or American Express) stating card number, expiratory date, and your full name.

Experimental Techniques in Human Neuropsychology. Edited by H JULIA HANNAY. (Pp 593 £22·95.) Published by Oxford University Press, Oxford 1994. 0–19–505471–7.

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 potentials, electroencephalography and electromyography are covered in standard fashion. Given recent developments, the section on regional cerebral blood flow is least useful. Only xenon studies are covered; there is no mention of SPECT, and PET is mentioned only briefly.

The book fulfils its purpose of providing a broad introduction to current neuropsychological research areas. Although dated, it 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 their area. Although an individual might consult it only occasionally, libraries in neuropsychological research institutes may find it a useful investment.

JOHN GREENE

Bailliere's Clinical Neurology–Inflammatory Neuropathies. Guest Editor J G McLEOD. (Pp 215; Price: £27.50). 1994. Bailliere Tindall, London. ISBN 0-7020-1818-X.

Balliere's Clinical Neurology series, a recently launched sistership 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 authority and accuracy. This year's second monograph, *Inflammatory Neuropathies,* edited by Professor McLeod (Sydney) is an outstanding edition.

There is little that has remained static over the last few years in clinical neuroscience or consequentially 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 immunopathology; 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 authoritively review the pathology, neurophysiology, and immunology of the inflammatory neuropathies, and the triad 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 paraproteinaemic neuropathy are followed by chapters on neuropathies related to infection, inflammatory plexopathies, and vas-