to the careful examination of the relation between the limbic system's function and the control of respiration. Indeed, like others, we are currently looking at the changes in the sensitivity setting of the respiratory centre in various mental states.

Our first paper, however, is somewhat defective in not clearly stating that the catheters were 0·5 cm in the nostril in nose-breathing subjects. The paper also fails to indicate how one figure for the end-tidal PCO_2 over 5 minutes was obtained. It was the mean for the expiratory CO_2 peaks over a period in which variation was minimal.

'Of course, there is no substitute for measurement of arterial blood itself', but practical reasons have led those interested in the study of the chronic psychogenic hyperventilation syndrome (see Lewis, 1957) to use methods in fact based upon the principles outlined by Tyndall (1865). This appears to be true of much research in general medicine.

We confess that capillary blood and end-tidal PCO_2 showed limited correlations, but we assume that this arose because respiration altered during blood sampling, and because the arterialization was not always complete. It must be emphasized that the correlation was with the mean end-tidal PCO_2 during the blood sampling when that too was more difficult to evaluate.

The catheter is likely to have had effects 'on the respiratory variables and mental state of a subject who was already in a "nervous state" ' but would this have been minimized by taking arterial blood?

Intra-subject variability also presents a problem, but one covered by the statistics.

Certainly benzodiazepines act for long periods, but the respiratory effect is reported by some to be limited and brief. Furthermore, the literature is contradictory (see Steen *et al*, 1966; Dalén *et al*, 1969). Intravenous chlordiazepoxide in the cat (Florez, 1969) and intramuscular lorazepam in man (Gasser *et al*, 1975) have no significant effect on respiration. The demonstration in a short communication to the Medical Research Society by Guz *et al* (1977) was not available to us. They showed effects of diazepam (not nitrazepam) on PCO_2 and ventilation. It does make us hesitate about possible drug effects on our patient sample, as some had nitrazepam the night before, but this may be similar to effects in other psychiatric studies and is not relevant to our second study of the effects of overbreathing. The matter nevertheless deserves closer scrutiny and more experiments.

Respiratory disease, too, could have been more common in our patients than in our controls. On clinical examination it did not seem to be so.

It may be unkind to state it, but nevertheless it is so, that the referees twisted our arms to write about drug effects on the blood brain barrier due to PCO_2. That speculation was not in our original text. It is impossible to please everyone, though, but we do thank Mitchell-Heggs *et al*, for their helpful remarks on our study.

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CAPGRAS' SYNDROME

Dear Sir,

I was struck by Dr Christodoulou's comment (*Journal*, June 1977, **130**, pp 556–64) that one of his patients demonstrated 'reduplicative paramnesia', a phenomenon I also recently noted in a patient whom I diagnosed as having Capgras' syndrome in 1975, and who presented a year later with reduplicative paramnesia (he was in hospital in Chicago but insisted he was actually in New York and that there were two New York Cities). Hayman and Abrams (1) suggested prosopagnosia as a possible cerebral mechanism for Capgras' syndrome, but these recent observations require consideration of reduplicative paramnesia as an alternative explanation.

In their review and case reports of reduplication, Weinstein and Kahn (2) include the delusion of doubles as a form of reduplication for person, and note that most cases of reduplication of body parts reported in the literature have occurred in association with right cerebral brain damage. Viewing Capgras' syndrome as a reduplication for person would

parsimoniously place it with the other reduplications (place, time, body parts), all of which can be localized to the non-dominant parietal cortex—rather than with the agnosias, which can be associated with various cortical dysfunctions according to the sensory modality affected.

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ELECTROCONVULSIVE THERAPY

DEAR SIR,

I recently treated a 62-year-old professional man with a course of right unilateral ECT. He had made a serious suicidal attempt by overdosage of drugs and had clear-cut signs of endogenous depression and a history of two previous episodes of depression for which psychiatric advice had not been sought.

For about 14 years he has suffered from bilateral nerve deafness and tinnitus. These symptoms followed an illness and were attributed to treatment with streptomycin. Having now maintained a good recovery from his depressive episode for eight weeks, he tells me that his tinnitus is much worse and he associates it with the application of ECT. I have treated with ECT other patients in his age group who have had deafness and tinnitus and have not had this difficulty described to me previously.

I wonder if any other psychiatrists have encountered such a side-effect?

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MOURNING AFTER PETS

DEAR SIR,

I was interested in Dr Keddie's paper (*Journal*, July 1977, pp 21–5). In it he states that 'where such overdependence on a pet does exist there is likely to be a sharp reaction on the part of the owner when the pet dies or has to be "put to sleep" '.

I am at present studying the relationship between children and their dogs. In the results from the 'normal' group, 23 out of 31 parents reported that they had considered the possibility of significant emotional distress occurring (in themselves or the child) if the dog was killed or otherwise permanently lost. This was a factor in their choice of pet, and is statistically significant ($P < 0.05$; binomial test, two tailed, corrected for continuity). This suggests that a certain amount of distress is anticipated by the general public at the loss of a pet dog. The mourning described by Dr Keddie is therefore pathological in severity rather than occurrence, and is presumably related to the degree of overdependence, as he implies in his paper. However, degrees of mourning for dogs may be more widespread in the community than has been thought.

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LITHIUM INGESTION IN THE POPULATION

DEAR SIR,

At the recent first British Lithium Symposium at Lancaster University, Dr A. I. M. Glen reported that in Edinburgh one citizen in every thousand was regularly taking lithium. In the closing address, Dr R. P. Hullin of Leeds reported, as the result of a broad survey, that nationwide about one in two thousand were on lithium treatment.

The biochemistry laboratory at Crichton Royal has the monopoly of measurement of plasma Li levels from patients resident within South West Scotland, a relatively self-contained area with minimal population mobility. As at 25 July 1977 there were 113 residents of the area receiving lithium therapy, comprising 93 out-patients, 18 longer-stay in-patients and psychogeriatric day-patients, and 2 recent admissions. From a population of 150,000 the figures reveal that one in every 1,330 persons is receiving lithium therapy.

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PLATELET MAO IN SCHIZOPHRENICS WITH AND WITHOUT FAMILY HISTORY OF SCHIZOPHRENIA

DEAR SIR,

We recently reported (1) that we were unable to confirm the findings of Murphy and Wyatt (2) of

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