agents, e.g. dopamine agonists such as bromocriptine or dantrolene, may be necessary initially.

KAREL DE PAUW

Towers Hospital
Gipsy Lane
Leicester LE5 0TD

KRYSTYNA SZULECKA

Bassetlaw District General Hospital
Worksop
Notts S81 0BD

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SIR: Wilcox & Nasrallah (*Journal*, December 1986, **149**, 782–784) suggest that the reason why catatonia has become something of a rarity is that brain injury in childhood, which the authors postulate as predisposing to catatonia, has declined in frequency.

I work in the field of mental handicap, in which brain damage is considered, in many instances, to be at the root of the handicap, particularly in profoundly affected patients. I have seen hardly any mentally handicapped individuals suffering from catatonia, although only recently I have been treating someone with this condition. If my experience is shared by other psychiatrists practising mental handicap, this would be an argument against the hypothesis that an organic brain condition is a forerunner of catatonia, unless one were to say that in the mentally handicapped catatonia, for reasons to do with the level of intelligence, is not a feature.

A survey of the incidence of catatonia in the mentally handicapped would throw light on the attractive theory put forward by Wilcox & Nasrallah.

I. FREEDMAN

Monyhull Hospital
Monyhull Hall Road
Birmingham
B30 3QB

A Case of Resistant Schizophrenia

SIR: I wish to comment on the treatment of this unfortunate young man (Mr A) (*Journal*, December 1986, **149**, 789–793). The record of therapeutic failure includes a trial of electroconvulsive therapy: "He was treated with six ECT for a possible affective component to his disorder, but with little benefit." Such a course of electroconvulsive therapy was probably inadequate.

Prior to the introduction of neuroleptic drugs, ECT was commonly used in the treatment of patients with schizophrenia. The comparative studies of the 1960s did not demonstrate a failure of ECT; rather, they demonstrated the equivalence of group results between those patients treated with ECT and those with neuroleptic drugs. The ease of administration, lesser expense, and assumed greater safety of psychotropic drugs, however, led to their replacement of ECT. Recent concerns about tardive dyskinesia, neuroleptic malignant syndrome, and other hazards of neuroleptic drug use (as well as therapeutic failures) have led some clinicians to re-examine the application of ECT in the treatment of schizophrenia.

Friedel (1986) recently reported the successful use of the combination of thiothixene and ECT in eight of nine patients who were non-responders to extended courses of neuroleptic drugs. We have treated nine schizophrenic patients who were neuroleptic and multi-drug treatment failures with the combination of ECT and fluphenazine. Of these, seven have been functioning well in the community for at least one year, and we are encouraged enough to undertake a random assignment trial. However, these patients required an average of 15 ECT in their treatment course, a number greater than is ordinarily given to our depressed patients.

The report by Brandon *et al* (1985) of the results of schizophrenic patients treated in the Leicestershire study and that of Taylor & Fleminger (1980) also encourage the use of ECT in schizophrenic patients. These findings were recently endorsed by the NIH Consensus Conference (1985) and by van Valkenberg & Clayton (1985).

If Mr A is still psychotic, a repeat trial of ECT should be considered with a minimum of 12 induced seizures, preferably with bilateral electrode placement, and with minimum durations of 25 s for each peripheral seizure (30 s central seizure). These treatments should be given while the usual dosages of neuroleptic drug therapy are continued.

MAX FINK

Department of Psychiatry
State University of New York at Stony Brook
USA

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Panic Attacks and Hyperventilation

SIR: Snaith (*Journal*, November 1986, **149**, 794) takes issue with Gelder's suggestion (*Journal*, September 1986, **149**, 346–352) that lowering of arterial pCO_2 may be a contributory factor in the development of a panic attack, and cites four pieces of evidence that fail to support such a view. However, Snaith has misrepresented Gelder, who did *not* state that hyperventilation *per se* causes panic. The psychological model proposed by Gelder is that hyperventilation induces panic *only* when the bodily sensations which it induces are (a) perceived as unpleasant, and (b) interpreted in a catastrophic fashion (Clark, 1986). In this respect hyperventilation is but one of a number of various pharmacological and physiological agents (such as caffeine, yohimbine, and CO_2 inhalation) which have been shown to provoke panic in susceptible individuals (Margraf *et al*, 1986). Thus the "overwhelming fear of dying or of becoming insane", which Snaith regards as the pathognomonic feature of a panic attack, is simply (in the cognitive model) a consequence of the misattribution of hyperventilation-induced somatic sensations to a life-threatening illness. Ley (1985) provided support for this view when he found that panic patients frequently report that the *first* thing they notice during an episode of anxiety is a physical feeling, most commonly breathlessness.

It is important to note that hyperventilation is neither a necessary nor sufficient prerequisite for panic anxiety. However, it is important to identify the sub-group in whom it may be a necessary condition, i.e. those in whom the symptoms of hyperventilation lead to panic, for the following reasons: firstly, the symptoms which cause panic can usually be reproduced by asking the patient to overbreathe; and secondly, the patients' fears and beliefs about the symptoms can be modified using a therapeutic technique that has been shown to be effective in reducing panic attack frequency (Salkovskis *et al*, 1986). We therefore need to know how this sub-group differs from those patients in whom hyperventilation does not play a role in panic.

Snaith remarks that not all patients who hyperventilate become anxious. This is certainly the case, as we have shown, but such a finding is not unexpected, since it is known that correlation between measures of physiological arousal and subjective reports of anxiety is not robust. It is also consistent with the cognitive model of anxiety. Although it has not been demonstrated that non-anxious hyperventilators do not report anxiety precisely because they do not appraise their somatic symptoms as frightening, this proposition is testable.

Snaith also points out that the administration of CO_2 to patients *induces* anxiety rather than the reverse, and argues that such a finding is not consistent with the hyperventilation theory. But whether CO_2 inhalations are anxiolytic or anxiogenic depends on the individual's cognitive set, i.e. on the instructions that are given before the inhalation. This observation emphasises the importance of cognitive factors in determining the affective state produced by manipulation of CO_2 (Clark, 1986).

I agree with Snaith that there is a lack of consensus about the definition of panic. It is not clear whether panic is quantitatively or qualitatively distinct from other forms of morbid anxiety, and the word has been used to describe both a psychological and a physiological experience. Some authors have introduced additional behavioural criteria such as fear of death, fear of losing control, fear of going crazy, or an urgent need to run, in addition to DSM-III symptoms.

It should be noted that the 'hyperventilation theory' has gained ground not because of the ideology of those committed to a psychogenic rather than a biogenic explanatory model, but because of studies that have revealed the importance of not only cognitive processes, but also specific physiological abnormalities such as hypocapnia in patients with anxiety disorders (Bass, 1985). Laboratory studies using phobic imagery and other provocations may help to further clarify the relationships among cognitive, physiological, and behavioural variables in patients with panic anxiety. Finally, it is worth recalling that the impetus for splitting panic disorder from the body of anxiety disorders arose from the observation that tricyclic drugs had a specific anti-panic effect. The evidence for this 'biogenic' theory of panic is not compelling (Margraf *et al*, 1986).

CHRISTOPHER BASS
Academic Department of Psychological Medicine
King's College Hospital
London SE5

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