

Correspondence

Panic Attacks

SIR: Gelder's review of panic attacks (*Journal*, September 1986, 149, 346–352) provides opportunity for a further comment on this topic. Gelder does not conclude that hyperventilation is a cause of panic attacks, but he does lean to the view that lowering of arterial CO₂ may be a contributory factor. That hyperventilation is the principle cause is fast becoming a fixed belief, and some arguments in support of this are set forth by Hibbert (1984). Furthermore, a therapeutic technique based on the induction of the symptoms of tetany (listed in the second column of Gelder's table), and cognitive restructuring of the consequent apprehension, supposedly supports the argument that lowering of arterial CO₂ is an essential component in the development of a panic attack. Before this view becomes dogma and is taught as fact to generations of clinicians in training, the contrary evidence should be considered.

Firstly, there is the fact, recognised by Gelder, that not all patients who develop panic attacks hyperventilate. Secondly, Hibbert (1984) states that people who hyperventilate become anxious — but this is contradicted by observation and research (Bass & Gardner, 1985).

The third misleading assumption is that the tetanic symptoms of hyperventilation are the same as those experienced during a panic attack; some of the somatic symptoms, e.g., paraesthesia, may be common to the two states but the 'apprehensive feeling' of hyperventilation is of a different order to the overwhelming anxiety accompanied by fear of dying or becoming insane which is the pathognomonic feature of a panic attack. Fourthly, the relationship of CO₂ to the neuronal systems mediating the experience of anxiety is complex, but other evidence points to the conclusion that raising, not lowering, arterial CO₂ induces anxiety (Gorman *et al*, 1984; Woods *et al*, 1986). Whereas the proponents of the hyperventilation hypothesis induce anxiety by deep breathing in their therapeutic technique, others (e.g., Griez & van den Hout, 1983) use inhalations of CO₂ to induce anxiety.

The categorisation of panic disorder in DSM-III followed the observation that this form of anxiety may respond to antidepressant drugs and this implies a 'biogenic' explanation of the disorder. I have the impression that the hyperventilation theory has

gained ground among those who wish to establish an alternative 'psychogenic' explanation. Much of the problem, as is always the case in psychopathology, rests on the definition of states; the reading of many research reports and some books on the topic leads me to the conclusion that the flexible word 'panic' is used in different senses and to support different views. No doubt debate is in progress as to whether ICD-10 should follow the example of DSM-III and categorise 'panic disorder'. If so, we must all be sure what we mean. It seems to me that the debate is irrelevant: West & Dally (1959) described another form of anxiety disorder responding to antidepressants before the observations of Klein (1964) which were the impetus to the DSM-III category. Of greater importance than categorisation is the establishment of clearer clinical guidelines as to which forms of anxiety disorder may respond to pharmacotherapy and for which forms psychological therapy is the procedure of first choice.

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Childhood Encopresis Extended into Adult Life

SIR: Fraser & Taylor (*Journal*, September 1986, 149, 370–371) wonder how many people have encopresis

extending into adult life. We report a small follow-up study which involved 22 teenage soilers (4 girls and 18 boys) admitted to an in-patient psychiatric adolescent unit who were followed up for 2–11 years (mean = 5 years; s.d. = 2 years) after discharge. They had been treated at High Lands Adolescent Unit, Scalebor Park Hospital, Burley-in-Wharfedale, between 1973 and 1983. Mean age on review was 19 years (s.d. = 4 years, range = 15–25 years); mean age at the time of admission was 13.5 years (s.d. = 3.7 years). All were of normal intelligence, and social class was mostly III (Registrar General's Classification). There was associated nocturnal enuresis in eight of them. Average duration of stay in hospital was 5 months (s.d. = 2 months); two were re-admitted. Twelve of them were considered to be conduct disordered, one had obsessional neurosis, one had a severe anxiety state, and one was depressed. Treatment consisted of toilet training, often with supplementary laxatives, therapy and family counselling.

Family doctors provided information on follow-up in all instances. Five former patients agreed to be interviewed, and another eight agreed to complete a review questionnaire. These 13 completed the 60-item GHQ and the Leeds Scale for Anxiety and Depression. It was found that there were three definite soilers between the ages of 17 and 23.

Case reports: (i) A 23-year-old man had been treated at the age of 16 with severe soiling and conduct disorder. Corroborative information came from both questionnaire and family doctor.

(ii) A 19 year-old man who was employed on a high rise building-site had been treated at the age of 14 years, and had subsequently been re-admitted for a recurrence of soiling. He was interviewed at follow-up and said he had a tendency to soil when out of reach of toilets.

(iii) An 18 year-old man who had been admitted at the age of 14 had been considered as conduct disordered and had nocturnal enuresis. Information in his case came from the family doctor. He was still soiling.

None of these three showed any evidence of psychiatric disorder on the GHQ or Leeds Scales.

The family doctors provided a control group selected randomly from their lists. None showed any evidence of soiling, although one 18 year-old had been treated for faecal impaction without soiling. For the 13 who completed the GHQ the mean score was 3.6 (s.d. = 1.9). One individual had a score of 11, indicating possible psychiatric disturbance. He had been treated by a psychiatrist. The mean anxiety score on the Leeds Scale for the 13 was 4 (s.d. = 2) and for depression was 2.3 (s.d. = 1.5).

We would like to thank Mrs B. Morris, who greatly assisted in the carrying out of this small project.

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Clopenthixol in Aggressive Mentally Handicapped Patients

SIR: We read with interest the report by Mlele & Wiley (*Journal*, September 1986, 149, 373–376). They conclude that "clopenthixol decanoate may be useful in reducing aggressive and disruptive behaviour in mentally handicapped adults". But has this finding any reliability? We do not know whether the effect was specific to depot clopenthixol because "none (of the patients) had been given depot phenothiazines" (presumably meaning depot neuroleptics, because only one depot phenothiazine is available). We cannot be certain that there were beneficial effects, because the ratings of behavioural changes were crude, as the authors observe, and even more importantly, they were not carried out blind to the treatment. A control group having placebo injections would be essential.

The paper is further weakened by the absence of diagnostic criteria for the use of the medication. It is disturbing that potent medication given by injection is being advocated for non-specific behaviour disturbances, which invites accusations of the use of 'chemical strait-jackets'.

We appreciate the diagnostic problems in people with mental handicap and the difficulties in their clinical management, but we think that the administration of drugs for behavioural control, rather than for the treatment of specific diagnosable illness, needs justification. With contemporary multiprofessional interest in behaviour modification techniques and non-institutional care, doctors need to be clearer about the indications for intervention with medication, rather than advocating its use on non-specific grounds.

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Cost of Community Psychiatry

SIR: Hoult (*Journal*, August 1986, 149, 137–144) is to be commended for the enthusiasm and enterprise he