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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 CO_2 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 CO_2 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 CO_2 to the neuronal systems mediating the experience of anxiety is complex, but other evidence points to the conclusion that *raising*, not lowering, arterial CO_2 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 CO_2 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