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SIR: I am grateful for the opportunity to respond to the letters from Snaith (*Journal*, November 1986, **149**, 794) and Bourne (*Journal*, February 1986, **150**, 265–266) about my article on panic attack. In his letter in the December issue, Snaith argues that panic attacks are not caused by hyperventilation, and implies that I take the opposite view. He has misunderstood my position, for I did not conclude that hyperventilation is the most important cause of panic. The misunderstanding centres round two statements in Snaith's letter and these require a response.

Firstly, Snaith writes that the effect of cognitive treatment "supposedly supports the argument that lowering arterial CO_2 is an essential component in the development of a panic attack". This is not the case: the effects of cognitive therapy support a different argument, namely that patients who experience panic attacks have a particular tendency to misinterpret bodily sensations, ascribing them to causes that are more serious than the real ones. For example, palpitations may be interpreted as evidence of heart disease. This view of panic attacks is consistent with the idea that on some occasions the misinterpreted sensations have been produced by hyperventilation, but it is not suggested that they always arise in this way. Equally, the cognitive hypothesis does not suggest that all those who hyperventilate will experience panic – only those people who have the particular tendency to misinterpret bodily sensations.

Secondly, Snaith writes that "the evidence points to the conclusion that raising, *not lowering*, arterial CO_2 induces anxiety". In fact, raising as well as lowering CO_2 can induce anxiety in some people (Van den Hout & Griez, 1984). At first this finding seems paradoxical, but it is easily explained by the cognitive hypothesis. Thus, either an increase or a decrease in pCO_2 can – through different mechanisms – cause unpleasant bodily sensations, and people who misinterpret these bodily sensations will be made anxious by either kind of change.

Returning to my own article, I did not single out hyperventilation as a frequent or major cause of panic attacks. Indeed, I concluded that it is unlikely that all panic attacks will turn out to have a single cause. I also suggested that 'biological' factors (including hyperventilation) have been overemphasised in the recent literature, and that more attention should be given to the investigation of psychological factors in these patients.

Bourne criticises me for failing to give a fuller account of Freud's views. However, he writes as though the subject of my paper were the aetiology of anxiety neurosis; in fact, it was the more specific problem of why some patients with anxiety neurosis develop panic attacks while others do not. My selective quotation from Freud was chosen to show how carefully he had described the clinical phenomena of anxiety disorders including panic attacks. I included this quotation because I agree with Bourne that some of Freud's early papers contain penetrating observations about clinical phenomena. I also agree that these observations are of great interest to present day practitioners of cognitive therapy. It is in their interpretation of the phenomena that analysts and cognitive therapists differ and, more importantly, in the kind of treatment that they have developed. Time will decide which approach is more fruitful: cognitive treatment for panic disorder is now being tested in controlled trials, but we still await reports of a comparable evaluation in which psychoanalytic treatment is compared with other methods.

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Use of Paraldehyde

SIR: The generally unfavourable discussion on the modern use of paraldehyde by Linter & Linter (*Journal*, November 1986, **149**, 650–651) cannot go unchallenged. The patient reportedly received 40 ml of paraldehyde intramuscularly over 12 hours. This is more than double the recommended maximum i.m. dose of 30 ml per 24 hour period (McEvoy & McQuarrie, 1986). Paraldehyde is a drug with a disagreeable smell and special storage requirements, but over 100 years of anecdotally safe use is precisely why "Its current use is limited to psychiatric units, particularly those without resident medical cover" (Linter & Linter, 1986): an important factor in those parts of the world that are short of doctors. Intermittent claims that paraldehyde is outmoded usually include a pharmacologically erroneous comparison with modern sedative/anxiolytics such as the benzodiazepines and phenothiazines and their derivatives. Paraldehyde belongs to the class of drugs