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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

including ethinate, clomethiazole, methyprylon, and ethanol (Goodman & Gilman, 1985) that act by membrane transport inhibition due to high lipophilicity and produce dose-related sedation devoid of the autonomic side-effects common with those drugs that act putatively via neurotransmitter mechanisms. While it is probably correct to state that the high potency benzodiazepines such as clonazepam and lorazepam have superceded paraldehyde in the parenteral treatment of status epilepticus, it remains useful as a sedative/hypnotic adjunct in hyperaroused states such as established delirium tremens, psychotomimetic 'street-drug' toxicity, and extreme mania, complementing the actions of neuroleptics of high anti-psychotic potency (but low sedative rating) that are usually administered concurrently. In these cases paraldehyde in normal doses does not produce the marked hypotension common with equi-sedative doses of the low potency sedating phenothiazines such as chlorpromazine which have marked anticholinergic and alpha-adrenergic blocking actions (Hollister, 1977). The patient reported by Linter & Linter arguably received an overdose of a drug no longer indicated for the condition concerned. These two errors should not be extrapolated to adversely influence the continued use of a drug still regarded by many as the standard against which other agents must be measured (McEvoy & McQuarrie, 1986), which has limited but highly specific indications and which currently has no exact pharmacological equivalent.

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Autocastration in Ontario Federal Penitentiary Inmates

SIR: With regard to the report by Waugh (*Journal*, November 1986, **149**, 656–658), the summary asserts that autocastration usually occurs in men with chronic paranoid schizophrenia. The evidence for this is not clear in the text, and it seems to contradict our experience, with an admittedly selected population, at the Regional Treatment Centre (Ontario). The Treatment Centre provides in-patient psychiatric care for the approximately 3000 federal penitentiary inmates of the region. The following brief case studies cover seven cases of attempted or successful autocastration on record since 1980.

Case reports: (1) A 23-year-old inmate was incarcerated for life for the second degree murder of his mother, which occurred during an argument about his transsexual behaviour. In 1980 he inserted foreign bodies into his genitalia as an attempt to force surgical castration. During reparative surgery his physique caught the attention of the urologist, and subsequent tests confirmed an XYY karyotype. In 1983 he lacerated his scrotum, intending castration, as part of a suicide attempt in protest at prison authorities' refusal to finance sex reassignment surgery. Final psychiatric diagnoses were of transsexualism and schizoid personality disorder.

(2) A 50-year-old Hungarian immigrant, also serving life for murder, in 1981 cut his scrotum, removed his testicles, and flushed them down the toilet, then swallowed the razor blade. He revealed that he did so to pre-empt a Mafia inspired scheme for other inmates to crush his testicles in punishment. Final diagnosis was of paranoid schizophrenia.

(3) A 38-year-old inmate, serving two years for theft, with earlier convictions for prostitution, in 1984 lacerated his scrotum, removed the right testicle, and by stuffing the cavity with cigarette ends, forced a complete surgical orchidectomy. Diagnoses were of transsexualism and antisocial personality disorder.

(4) A 41-year-old man lacerated his scrotum as part of a suicide attempt after revocation of his parole. He had served ten years of a composite 24 year sentence for a series of rapes. While out on parole and because of impotence he had refused continued provera treatment, which in turn lead to the revocation. Diagnoses were of adjustment disorder with depressive features, and antisocial personality disorder.

(5) A 40-year-old transsexual, who had castrated himself in 1979, slashed at his penis in 1980 and again in 1983 in protest at being placed in a male, rather than female, institution after his conviction for a series of armed robberies. Diagnoses were of gender dysphoria and personality disorder, unspecified.

(6) A 34-year-old serving ten years for six counts of robbery in 1984 almost completely severed his genitalia and slashed his wrists, intending suicide. He did it, he said, to punish himself. His sexual orientation was normal; diagnoses were of substance abuse and borderline personality disorder.

(7) A 27-year-old transsexual serving a 15-year composite sentence for assault and attempted murder in 1983, under the influence of "brew" and illicit drugs, attempted castration to force sex reassignment surgery. Diagnoses were of transsexualism, substance abuse, and personality disorder, unspecified.

Of these seven patients, three of them successful, only one was diagnosed as having a psychosis. The