1980), but as yet there is no satisfactory or widely accepted definition of HVS. Until this deficiency is remedied it will not be possible to determine the relation between hyperventilation, i.e. objective evidence of hypocapnia, and symptoms reported during episodes of panic. Secondly, we need to be much more rigorous in excluding organic disorders that can present with anxiety or panic symptoms (Jacob & Rapport, 1984). We have recently demonstrated that organic lung disorders may provide the initial stimulus for breathlessness in patients with symptomatic hyperventilation (Gardner & Bass, 1984). Elaborate investigations may be required to exclude these disorders. We are not told how organic disease was excluded in Kraft and Hoogduin's patients.

Diagnosis may be difficult in patients with acute, intermittent hyperventilation who may be normocapnic between episodes. We have suggested elsewhere (Bass & Gardner, 1983) that ambulatory CO_2 monitoring may provide useful information in such cases, but this technique is not yet widely available. We have devised a protocol in which $PACO_2$ is monitored uninvasively in the laboratory over long periods, including sleep. End-tidal pCO_2 is carefully measured whilst the patient is subjected to a number of standardised stressors, including exercise and forced overbreathing. The technique is reliable and acceptable to patients, and provocation of hypocapnia during the procedure is highly suggestive of HVS.

In view of the heterogeneity of HVS (acute and chronic forms occur in clinical practice), we believe it is essential to establish objective diagnostic criteria before subjecting patients with vague and non-specific symptoms to trials of treatment. Otherwise hyperventilation syndrome (or more correctly "symptomatic hyperventilation") is destined to acquire the status of Briquet's syndrome: a clinical entity of dubious validity characterised by a conspicuous lack of positive diagnostic features.

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KORO IN NON-CHINESE SUBJECTS

DEAR SIR,

I was interested to read the reports concerning koro symptoms in non-Chinese subjects. I have recently encountered a further case, which I report here:

A 25 year-old previously well single black male presented with acute anxiety and the conviction that his penis was retracting into his abdomen. Onset was sudden, after erectile failure during attempted coitus. Fear of impotence had existed for 8 months, since he had been an unwilling participant in a tribal ritual during which he was circumcised. He was of good intelligence, with no hallucinations or other delusions. His condition deteriorated while being treated with clomipramine 75 mg per day, and he eventually became mute and catatonic. Haloperidol 30 mg per day resulted in rapid improvement, but he remained convinced that his penis was shrinking.

The exact nosological status of this patient's condition is unclear, perhaps best fitting Ang and Weller's description (*Journal*, September 1984, **145**, 335) of an acute anxiety state and delusional belief due to an underlying psychotic illness. However, the clear-cut initial sensitising experience and the ultimate precipitating event are in keeping with the psychogenic syndrome described by Yap (*Journal* 1965, **111**, 43–50).

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DEAR SIR,

I have read with interest the reports of koro-like symptoms in non-Chinese subjects in several psychatric conditions. I here report a relevant case:

A 60 year old Scotsman married with 3 children was referred for withdrawal from lorazepam. He had been treated with 4 mg daily 10 years before, after developing cardiac dysrhythmia. Withdrawal after some months precipitated feelings of restlessness, and ringing in his ears and he was replaced on lorazepam 10 mg daily until 3 years ago when he was admitted to hospital to be withdrawn from it. On the 10th day after discontinuation he was agitated, restless, and tearful; he felt numb around his mouth and thought his whole body was shrinking and his penis and testicles were disappearing almost as though he was changing into a woman. He did not believe the

symptom would cause death and no manoeuvres were employed to prevent penile and testicular "loss".

The most likely explanation for these koro-like symptoms is that of depersonalisation focussing on the genital area, occurring in a state of extreme anxiety. The growing number of cases being reported in non-Chinese subjects, and the strong link with anxiety and anxiety-related states in these cases suggests strongly that these symptoms can be regarded simply as an unusual presentation of anxiety.

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RELAXATION AND DEPERSONALISATION

DEAR SIR,

In his letter (*Journal* August 1984, **145,** 217) Dr Fewtrell noted paradoxical anxiety reactions in seven of forty cases treated with relaxation training. He also noted a relationship between liability to depersonalisation and negative reactions to relaxation treatment in these patients, and as a result hypothesised that a tendency toward depersonalisation may predispose a patient to respond poorly to progressive relaxation. This hypothesis raises a number of issues.

It seems questionable whether depersonalisation or any other specific negative reaction necessarily confounds the entire relaxation training process. In fact, in their progressive relaxation training manual Bernstein and Borkovec (1973) note a variety of untoward responses to relaxation that might be eliminated by means of a number of specific treatment measures. Specifically they suggest such procedures as therapist modeling, emphasising the patient's self-control, allowing the patient to practice with eyes open, slowing the pace of treatment and general explanation of the negative reactions themselves. Since it is unclear from Dr Fewtrell's report whether such measures were attempted, it remains open to question whether patients' depersonalisation reactions to relaxation can be effectively addressed. My clinical experience has suggested that depersonalisation can be effectively eliminated in some patients by means of the Bernstein and Borkovec (1973) procedures.

However, I do not intend to suggest that all negative responses to relaxation training can be effectively addressed. A recent survey (Edinger & Jacobsen, 1982) revealed that adverse reactions to relaxation training led to noncompliance or patient-initiated termination of treatment in 3.47% of the cases treated by a group of 116 clinician respondents. Further these clinicians reported discontinuing relaxation training in 3.83% of their cases because adverse reactions seriously confounded the treatment process. Hence significant treatment confounding reactions to relaxation training seem to be reasonably common.

In addition it is important to note that untoward responses other than depersonalisation can at times confound relaxation treatments. Dr R. Jacobsen and I (Jacobsen & Edinger 1982), for example, described at length two cases, one with heightened anxiety without depersonalisation and the other muscle cramping. It was interesting that in both cases underlying psychodynamic issues seemed to explain the observed reactions. Moreover, efforts to counteract these reactions failed to eliminate them. Hence, untoward reactions other than depersonalisation require our attention.

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DEPRESSION AND PHARMACOLOGICAL TREATMENT

DEAR SIR,

The recent report by Garvey *et al* (*Journal,* **145,** October 1984, 363–365) lends support to the notion that stressful life events may precipitate a depression of such severity that pharmacotherapy is warranted. Unfortunately their conclusions would seem to be limited by the patient population's low pre-treatment scores on the Hamilton Rating Scale for Depression (mean−17±6). As they note, their patients might have improved without treatment, in time, or with other, nonsomatic therapies.

An interesting issue not addressed in their data relates to whether the reported stresses were confined to the pre-treatment period or persisted through the treatment period. There has been very little research on the efficacy of pharmacotherapy in Major Depressive Disorder (MDD) in the face of ongoing, stressful life events (Feinberg & Halbreich, 1985). Existing evidence suggests in fact that some stressors do interfere with the somatic treatment of MMD (Lloyd, 1981; Akiskal, 1982).