region and to swap ideas on this rather ill-defined yet important problem.

D. R. DAVIES

Moorhaven Hospital
Bittaford
Ivybridge
South Devon PL21 0EX

The Pathogenesis of Depersonalisation: A Hypothesis

SIR: Depersonalisation/derealisation are common symptoms of anxiety and of affective disorder, and are not uncommon features of other psychiatric syndromes. They are also common in various organic states (e.g. hypoglycaemia) and are frequently likened by patients to dreaming, which occurs at a time of demonstrable alteration in cerebral activity. Furthermore, these phenomena are disorders of perception, which suggests some organic basis. Is it possible that depersonalisation/derealisation are *always* manifestations of demonstrable organic abnormality in the brain?

Hyperventilation, too, is a common symptom of anxiety, and depersonalisation is common in hyperventilation. Anxiety is frequent in most psychiatric syndromes, and it might be reasonable to suppose that whatever the primary psychiatric diagnosis, it is only the patient who is anxious and hyperventilating who develops depersonalisation. In such patients the depersonalisation would be the result of the changes in metabolism and cerebral blood flow produced by the hyperventilation. The hypothesis would be that only those patients who were over-breathing would be depersonalised, whilst those patients who were not over-breathing would not suffer depersonalisation. At the same time the proviso has to be made that not all patients who are over-breathing would necessarily experience depersonalisation, as there might be some individual variation in the propensity to develop this symptom.

Having made this hypothesis, I set out to test it. As a first step I began to look for patients with depersonalisation who were not over-breathing, with a view to comparing various measures in them with patients who were over-breathing. It has, however, proved to be increasingly difficult, if not impossible, to find such patients who were not over-breathing. I think this may be because, since I have become aware of the hypothesis, I am not overlooking hyperventilation in such patients, whereas previously I might have been. Thus the investigation might not turn out to be as easy as it seemed at first, not an unfamiliar situation. In the meantime, however, the idea appears to have spread in this hospital and I have found over the past few months or more that it has been quoted to me as 'received knowledge' in teaching sessions with undergraduates and with junior doctors. For this reason I seek the hospitality of your columns to go on record as its originator.

SAMUEL I. COHEN

The London Hospital Medical College
Turner Street
London E1 2AD

'Neuroleptic Malignant Syndrome' Without Neuroleptics

SIR: In support of the suggestion by Singh & Maguire (*Journal*, December 1987, **151**, 863) that the term neuroleptic malignant syndrome (NMS) should be revised, we report a fulminating case, exhibiting all of the diagnostic criteria proposed by Levenson (1985) but which occurred when lithium and phenelzine were employed in therapeutic doses. The patient had never taken neuroleptic drugs.

Case report: A 42-year-old woman presented to casualty with a rapid onset of restlessness, sweating, and confusion. She had a history of depression with intermittent agitation and some phobic symptoms of several years duration. Her medication comprised the following: phenelzine (15 mg three times daily), lithium carbonate (800 mg daily), L-tryptophan (1 g daily), diazepam (2 mg three times daily), and triazolam (0.25 mg daily). Phenelzine had been commenced six weeks previously, replacing clomipramine which had proved ineffective over four months. Relatives believed that the patient took her medications only as prescribed.

Within three hours she was comatose. Pupillary and corneal reflexes were lost. Trunk and limbs were hypertonic and held in rigid hyperextension. Tendon reflexes were brisk, but plantar responses were flexor. Temperature rose from 38.5°C on admission to 42.5°C four hours later. She had a tachycardia and became hypotensive.

A diagnosis of NMS was made and she was treated with intravenous dantrolene (60 mg three times daily), commenced within four hours. Body temperature returned to normal within 14 h; blood pressure and heart rate were controlled with dopamine and practolol.

Investigation showed mild leucocytosis and initially normal biochemical parameters of hepatic, renal, and muscle function. Serum creatine phosphokinase became elevated, reaching a peak of 41 355 U/l (normal values 24–175) on the third day. Cerebrospinal fluid was normal. Blood and urine cultures were negative. Intravenous benzylpenicillin and gentamicin were commenced before results of these became available.

Severe disseminated intravascular coagulation occurred after 12 h. Acute renal failure and continuing infusion of blood products necessitated treatment by peritoneal dialysis. Mechanical ventilation was instituted. By day five, elevated transaminases and alkaline phosphatase indicated severe hepatocellular damage. Profound hypoglycaemia