The patient with true koro also exhibits manifest hypochondriacal features (Yap, 1965*a*,*b*). We propose that somatic awareness and health consciousness may be the common clinical feature in patients who suffer koro and koro-like states. In true koro these concerns are exaggerated and moulded by culture-bound ideas, whereas in koro-like syndromes the final presentation is generated by psychotic or anxiety-related beliefs.

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Sodium Valproate as an Antidepressant

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A patient with chronic depression responded to treatment for her major depressive episodes, but was left with a dysthymia which was eventually relieved by anticonvulsants. Sodium valproate may be of use in a range of affective disorders.
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Resistant depression is "symptomatic non-recovery for a period of two years or more and may be a sequel to one or more episodes of major depression from which the patient does not recover" (Cassano, 1983). The prevalence of such a disorder is estimated as being 12–15% of psychiatric patients (Scott, 1988). Patients are often severely handicapped by their illness, and it is frequently necessary to try various regimes of treatment to find the one to which any particular patient will respond.

The use of sodium valproate in the treatment of bipolar affective and schizoaffective disorders has been documented (Post & Uhde, 1983; Calabrese & Delucchi, 1990). It ranks alongside the more commonly used carbamazepine as a useful alternative or adjunct to mood-stabilising therapy with lithium carbonate (Klosiewicz, 1985). The psychotropic properties of anticonvulsants was first noted by Kubanek & Rowell (1946), who used phenylhydantoin in patients with mania or schizophrenia and observed a higher rate of recovery among the manic patients. Sodium valproate has also been used in the control of episodes of mania (Emrich *et al*, 1985). Its usefulness has been reported in patients who have suffered ongoing affective disorders following closed head injuries, even in the presence of a normal electroencephalogram (Pope *et al*, 1988).

A literature search has failed to show any published data on the effect of sodium valproate in resistant unipolar depression. A patient is reported for whom anticonvulsant medication provided the answer to resistant depression.

Case report

A 38-year-old woman had first been referred for psychiatric assessment at the age of 22. She had been adopted in infancy,

and there is no record of her natural family. However, there is no history of psychiatric illness in her adoptive family. The patient suffered from agoraphobia from the age of 16 to 19, but did not seek help.

At 22 she presented twice in five days, having taken overdoses. She appeared quiet and hesitant, with vague suicidal ideas and a diurnal variation in mood, insomnia with early wakening, impaired concentration, and general loss of interest accompanied by anorexia and some weight loss. She had had fluctuating depressed mood and irritability for as long as she could remember, but they had become more severe in the preceding week. All physical investigations were within normal limits. Following the second overdose she was admitted and treated with dothiepin (75 mg daily) and supportive psychotherapy. There was an uneventful follow-up in the out-patient clinic.

Before the next admission she married a divorcee, also an ex-patient. The marriage was happy and voluntarily childless. She functioned poorly and was never completely well according to her husband. She worked part-time in a shop but found it very stressful.

Over the next 15 years she was admitted informally to hospital on a further six occasions, on three occasions following an overdose of her current medication. The second overdose required resuscitation in the local hospital before her transfer to our care.

Before each admission there had been a three- to six-month history of worsening symptoms. Her admissions ranged in duration from 24 hours, when she discharged herself against medical advice, to five months. The mainstay of treatment were tricyclic antidepressants: always amitriptyline (75 mg daily), sometimes used in combination with a course of eight sessions of electroconvulsive therapy (ECT), or with tranylcypromine (10 mg) and stelezine (1 mg) once daily, or with phenelzine (15 mg twice daily).

The last admission was two years ago, during which the patient was commenced on lithium (800 mg daily), achieving adequate blood levels and resulting in some minor clinical improvement which was not sustained. A month later she had to be detained compulsorily under section 2 of the Mental Health Act 1983, as she had attempted suicide. A further course of ECT was offered and refused. As an adjunct to lithium therapy, carbamazepine (100 mg daily) was added and within a few days her mood lifted to a level previously unknown to her. The biological symptoms disappeared and her whole appearance changed; she became smart, well dressed, confident, spontaneous, and sociable. Her thinking became much more positive in its content.

She developed an itchy erythematous rash, and all medication had to be stopped; she became depressed within 48 hours. Her mood remained low for about four weeks, until the introduction of sodium valproate (100 mg, increasing to 600 mg twice daily over two weeks) which began to restore her mood within 48 hours.

Some weeks after discharge a trial without any medication was undertaken, with consequent relapse of her mental state within a week. Treatment was reinstated after one week, with rapid recovery of her euthymic mood. During out-patient follow-up she has on other occasions stopped her medication, again resulting in the dramatic worsening of mood, rectified within days of restarting at the previous dose. She has now been maintained on sodium valproate alone for two years, the lithium having been discontinued without consequences.

Discussion

This patient fulfils the DSM-III-R criteria for a major depressive episode (American Psychiatric Association, 1987). There are many indicators of biological origins in this case: a history of agoraphobia (possibly an atypical first biological illness), chronic depression/dysthymia with major depressive episodes characterised by loss of weight, poor concentration, and disturbed circadian rhythms.

Lambert *et al* (1966) piloted studies in patients with schizoaffective disorders using valpromide (the amidation product of valproate) and observed improvements in patients with depression as well as those with mania. Since then little work has been published on its use in depression, but a considerable bank of knowledge has built up about its use in mania and rapid-cycling bipolar affective disorder, either on its own or in combination with lithium.

Post & Uhde (1983) postulated that selective anticonvulsants such as carbamazepine and perhaps valproate, if not the entire class of anticonvulsants, might provide an alternative treatment for the primary disorders of affective dysregulation. Jann *et al* (1984), in a literature review on alternative drug therapies for mania, highlighted two double-blind control trials of valproate, which included 12 patients who were followed up for up to three years without relapsing.

Some authors have postulated that mania involves a central dysfunction of the GABAergic system. Compounds with GABAergic activity have been used, including sodium valproate (Emrich *et al*, 1985).

Klosiewicz (1985) discussed the use of valproate in cycling bipolar illness and pointed out that the doses required to stabilise mood disturbance are much smaller than are normally used in anticonvulsant prophylaxis. As valproate is a less potent inhibitor of amygdaloid kindling, using it as a substitute in patients stabilised on carbamazepine can lead to a relapse of their affective illness.

The antidepressant effects of carbamazepine, when evaluated, have shown it to be useful in depression that is resistant to lithium and in stabilising affect thereafter (McElroy *et al*, 1988). Valproate may have a similar effect, the only apparent advantage demonstrated so far being that the onset of action of valproate seems more rapid.

Sodium valproate has a number of side-effects, including disturbances of hepatic function, which can

be fatal if missed, thrombocytopenia, altered platelet aggregation, and minor gastrointestinal intolerance, but for a patient who is unable to function because of chronic illness the benefits may outweigh the risks.

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Chronic Depersonalisation Neurosis *au* Shorvon – A Successful Intervention

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A patient with chronic primary depersonalisation responded well to a combination of psychotherapy and abreaction.
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In ICD-9 (World Health Organization, 1978), depersonalisation neurosis is described as "a neurotic disorder with an unpleasant state of distorted perception in which external objects or parts of one's body are experienced as changed in their quality, unreal, remote or automised. The patient is aware of the subjective nature of the changes he experiences."

Shorvon (1946) has described what he called primary idiopathic depersonalisation, a disorder of primary depersonalisation commencing in late adolescence and which is continuously present for many years, although with some fluctuation in severity. We report a patient with this disorder who improved substantially with a combination of psychotherapeutic techniques and abreaction.

Case report

A 55-year-old lady was referred to a psychiatric out-patient clinic because of worsening of her feelings of unreality, which had been present for 35 years. She complained that her voice felt distant from her, that she felt distant from things, and that she felt as if she were not there. She found these phenomena difficult to explain but said that she felt herself to be unreal. She was able to feel the full range of emotions, and no parts of her body had a 'woolly' or unreal character. She had no altered perception of time, although she complained of distorted perception of space. Her image seemed real in the mirror and other people and external objects retained their real quality, but she described life as sometimes having the quality of a play.

She described occasional dysphoria, usually when she was inactive and when she had time to ponder on the unpleasantness of her subjective experiences. She had never had any biological features of depressive illness or features suggestive of an agoraphobic, obsessional, or other neurotic disorder, apart from primary depersonalisation. She had never suffered from features suggestive of temporal lobe epilepsy, she never abused alcohol or drugs, and there was no history of head injury or intracranial infection.

The subjective depersonalisation first arose when she was 20, at a time when her husband was called up for national service, leaving her at home to look after two children under 18 months of age. She is unable to recall the initial experience, but feels that its onset was insidious. She was first referred to a psychiatrist in 1954 when she was prescribed electroconvulsive therapy; this gave no improvement. She soon became disillusioned with psychiatric