

To then applaud a study which demonstrates a high rate of diagnosed schizophrenia in black patients as having "brought transcultural psychiatry research to life" seems a little mysterious. Presumably this is a (covert?) way of arguing that the problems of intercultural psychiatry are simply problems to be located in the presumed psychopathology of the designated patient, and that the procedures of psychiatric practice, our underlying theoretical assumptions and their social context, are above any critique. If this is their message, then it is one I deplore.

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LITTLEWOOD, R. (1986) Ethnic minorities and Mental Health Act. *Bulletin of the Royal College of Psychiatrists*, 10, 306–308.

Professor Max Hamilton: an apology

SIR: During the recent CINP meeting in Japan a promotional item was distributed with a copy of the Hamilton Rating Scale for Depression, including the name of our drug, Tolvon. It was by no means our intention to indicate that the late Professor Hamilton had endorsed this drug or that he had been associated with it. We are sorry to learn that close colleagues and the immediate family of the late Professor Hamilton have been disturbed by this publication, particularly in view of the fact that, in helping with drug trials, Professor Hamilton was always impartial and insisted that his name should not be used to sway a decision one way or another.

If we have inadvertently given any other impression we should like to state that this was certainly not our intention.

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Suicide in Indian women

SIR: We want to put forward our views about some of the aspects related to suicide in Indian women, described by Raleigh *et al* and Veluri & Greene in two recent issues of the *Journal* (January 1990, 156, 46–50, and July 1990, 157, 149–150).

Burning is not the only method commonly chosen by Indian women to commit suicide. In a recent Indian study (Shukla *et al*, 1990) carried out in Jhansi, a small city in North India, burning, drowning, poisoning and hanging were the methods used by women for committing suicide in 36%, 23%, 27% and 16% of cases respectively.

There does not appear to be any relationship between the phenomenon of *Sati* ('Suttee') and suicidal burning in the current context as mentioned by Raleigh *et al* and Veluri & Greene. *Sati* is a custom, which was practised by Hindu women after the death of their husbands, in which they used to burn themselves on the husband's pyre. It was accepted as a devotion to the husband by society rather than suicide as would be described in terms of recent thinking. The practice (malpractice) of *Sati* was quite prevalent in India, until the first few decades of this century, although it started in the medieval period. Sporadic cases are still reported, especially from the rural areas and the state of Rajasthan, where it still has social acceptance. In some cases of *Sati* it is a forced burning rather than a voluntary suicide.

The comment of Veluri & Greene regarding over-dosage of drugs as a method for committing suicide does not appear valid in our opinion. It is not due to ignorance on the part of the suicidee, but due to the difficulty in procurement and expense involved, that Indian women chose other methods for committing suicide. Drowning is another method used by women from rural India. A common method of drowning is by jumping into the village well.

We feel that the availability and accessibility of a particular method to Indian women determines the way of committing suicide.

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SHUKLA, G. D., VERMA, B. L. & MISRA, D. N. (1990) Suicide in Jhansi city. *Indian Journal of Psychiatry*, 32, 44–51.

Carbamazepine and NMS

SIR: Dalkin & Lee (*Journal*, 1990, 157, 437–438) report a case of probable neuroleptic malignant syndrome (NMS) without fever following an overdose of trifluoperazine and carbamazepine. The authors suggest that carbamazepine may modify NMS,

increasing the likelihood of apyrexial presentation. They discussed possible mechanisms but state that carbamazepine is not known to affect dopaminergic systems.

The hypodopaminergic state thought to be responsible for NMS may arise: from direct interference with dopamine (DA) transmission by neuroleptics or withdrawal of anti-parkinsonian drugs (Toru *et al*, 1981); by imbalance of serotonin (5-HT) and DA, as has been suggested for a case induced by fluoxetine (Halman & Goldbloom, 1990); or by acetylcholine:DA imbalance (Corrigan & Coulter, 1987).

As the hyperthermia of NMS may be attributable to DA receptor blockade in the hypothalamus, it may be relevant that carbamazepine has been shown to enhance the growth hormone response to apomorphine in humans (Elphick *et al*, 1990). Although studies in rats suggest that carbamazepine has no affinity for DA receptors (Marangos *et al*, 1983), the growth-hormone response to apomorphine in humans may be modified by a direct action on dopamine receptors, perhaps supersensitivity of post-synaptic receptors secondary to reduced DA transmission. A selective increase in dopamine activity in the hypothalamus but not in the nigrostriatal pathways would account for the absence of pyrexia in the case described.

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References

- CORRIGAN, F. M. & COULTER, F. (1987) Neuroleptic malignant syndrome. Amitriptyline and thioridazine. *Biological Psychiatry*, **23**, 320–321.
- ELPHICK, M., YANG, J. D. & COWEN, P. J. (1990) Effects of carbamazepine on dopamine- and serotonin-mediated neuroendocrine responses. *Archives of General Psychiatry*, **47**, 135–140.
- HALMAN, M. & GOLDBLOOM, D. S. (1990) Fluoxetine and neuroleptic malignant syndrome. *Biological Psychiatry*, **28**, 518–521.
- MARANGOS, P. J., POST, R. M., PATEL, J., *et al* (1983) Specific and potent interactions of carbamazepine with brain adenosine receptors. *European Journal of Pharmacology*, **93**, 175–182.
- TORU, M., MATSUDA, O., MAKIGUCHI, K., *et al* (1981) Neuroleptic malignant syndrome-like state following a withdrawal of anti-parkinsonian drugs. *Journal of Nervous and Mental Disease*, **169**, 324–327.

Benzodiazepine withdrawal syndrome

SIR: Ashton and her colleagues (*Journal*, August 1990, **157**, 232–238) showed that buspirone is

unhelpful in the management of the withdrawal syndrome that commonly follows the discontinuation of long-term benzodiazepines given at therapeutic doses. This disorder may persist for months, occasioning great distress and despair. Treatment of these symptoms remains unsatisfactory (Higgitt *et al*, 1985) and relapse with resumption of drug-taking is only too common (Golombok *et al*, 1987).

The biochemical basis for the dependence remains unclear, although alterations in benzodiazepine receptor function have been sought (Miller *et al*, 1988). In animals, administration of the benzodiazepine antagonist, flumazenil (Whitwam, 1988), results in reversal of receptor changes (Gonsalves & Gallager, 1985) and obviation of benzodiazepine withdrawal symptoms (Gallager *et al*, 1986). Based on this research we have attempted to treat protracted benzodiazepine withdrawal symptoms by administration of intravenous flumazenil.

With informed consent, we have so far tested 11 patients who have been benzodiazepine-free for at least three weeks. In the first five patients a total dose of 0.5 mg or less was used with little effect. In most of the other six patients a larger total intravenous dose of 2 mg of flumazenil divided into three doses over a few hours was found to have promising effects. Long-standing symptoms were reported by several patients to be dramatically relieved. These included clouded thinking, tiredness, muscular symptoms such as neck tension, jerks and shaking, and the perceptual symptoms occurring as a characteristic component of benzodiazepine withdrawal: pins and needles, pain and subjective sensations of bodily distortion. Mood disorder, when present, also improved but the changes in anxiety and depression may have been a response to relief of physical symptoms. Some patients reported the maximum response delayed by as much as a day but in most the onset of effect was noted soon after the injections. Side-effects were reported to be either absent or typically described as light-headedness or dizziness lasting only a few minutes and usually well tolerated.

The benefits were reported to last between a few hours and several days which is noteworthy considering flumazenil's half-life is less than one hour. Those patients receiving a second or third dose tended to report subsequent longer relief. However, symptoms did return to varying degrees in most cases, suggesting that a course of treatment may be required. The parameters of such therapy are currently being explored.

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