in problematic situations. Figuiredo (1976) attempts to overcome some of these difficulties by providing his translators with specific guidelines, broadly designated as Wording, Redundacy, Context and Decentering.

The general aim of the process of translation should be to arrive at a consensus translation, rather than one that focuses principally on the source language. The technical translator, bilingual psychiatrist and English-speaking psychiatrist should initially examine individual items independently and then confer, in order to arrive at an acceptable translation. I believe that the PSE translation into several languages is a step towards developing a standardised instrument to collect validly comparable data across cultures, but I suggest that the method of translation may have to be reviewed.

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Transcultural Psychiatry

DEAR SIR,

If cultural psychiatry is to move beyond the collection of exotic phenomena which escape our supposedly culture-free Western classifications, it will have to examine more closely the actual phenomena which colonial psychiatry has bequeathed to us.

The papers of Swartz *et al* and Farmer *et al* (*Journal*, April 1985, **146**, 391–394, 446–448) refer to *Witiko* and 'pointing the bone'. *Witiko* (*Windigo*) is a 'near mythical syndrome' (Neutra *et al*, 1977) with perhaps three actual instances, and one which has never been observed by outsiders (Shore & Manson, 1981; Marano, 1982). Similar doubts have been cast on 'pointing the bone', popularly known as 'voodoo death' (Lewis, 1977; Eastwell, 1982), although Gomez (1982), maintains the notion has some validity.

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Is Mania Incompatible with Down's Syndrome

DEAR SIR,

I was interested to read the paper by Drs Sovner, Hurley and Labrie (*Journal*, March 1985, **146**, 319–320) and note, as I have done with some other work from the United States, an apparent unfamiliarity with recent British research.

My colleagues and I investigated a series of 40 mentally disordered patients with mental handicap, 16 of whom suffered from an affective psychosis diagnosed using strict criteria. The other 24 cases suffered from schizophrenia, again diagnosed using rigorous criteria similar to those of DSM III.

In investigating possible aetiological factors we karyotyped all the index patients plus 40 controls from the same hospital for the mentally retarded who did not have mental illness. None of the cases with affective disorders were associated with chromosome abnormalities, whereas one of the schizophrenics and five of the controls were found to have trisomy 21 (Down's syndrome), some of them showing various degrees of mosaicism. However, there was no statistical significance in the differences between these groups.

In my review of the literature on this subject I found no cases of Down's syndrome in association with affective disorder. The absence of such an association is certainly worth pursuing as there is no question from our own work and that of others that affective disorders can occur amongst the mentally handicapped and can be identified even in those whose intellectual capacities are very low indeed (Hucker *et al*, 1979).

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A Malignant Corpus Callosum Tumour in an 85-year-old Demented Woman

DEAR SIR,

An 85-year-old widow with progressive dementia was admitted to hospital for two weeks to provide relief to her family. On admission her grossly defective cognitive state was confirmed but she was mobile, had spontaneous speech, dressed and fed herself and co-operated with the staff. On discharge she went into a residential home and, almost immediately, there appeared a dramatic change in her behaviour. She had become incontinent of urine and faeces, had slowed up considerably and had to be fed, taking two hours to eat a small meal. Over the next 10 days she continued to withdraw, totally lacked any spontaneous movement and had to be re-admitted to hospital. There she was mute but appeared to be aware of things and responded to sounds. She was immobile and continued to be doubly incontinent. A CT head scan revealed a "large malignant tumour in the anterior portion of the corpus callosum, which (was) displacing the lateral ventricles posteriorly". It was deemed inoperable. She rapidly became comatose and died without much pain four weeks later. Apart from some vomiting in the terminal stages of her illness, there was no evidence of any raised intracranial pressure. No autopsy was done.

The point has been made (Mahendra, 1981) that stupor in psychiatric practice was no longer to be simply attributed to "functional" causes and nowadays required neurological investigation.

Tumours of the corpus callosum have long held a fascination for neuropsychiatrists. Mental changes are more frequently observed in these tumours than with tumours anywhere else in the brain (Walton, 1977). Selecki (1964) pointed to the rapid mental deterioration before the appearance of neurological features. These changes consisted of progressive loss of recent memory, slowing down of mental and physical reactions, and poverty of movement and thought, progressing to dullness, apathy and depression.

I would like to thank Dr. R. M. Paxton, Consultant Neuroradiologist, Freedom Fields Hospital, Plymouth, for reporting on the CT head scan of this patient.

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DST, Endocrines and Loss of Weight

DEAR SIR,

We enjoyed reading the article by Berger *et al* (*Journal*, October 1984, **145**, 372–382) on the limited utility of the dexamethasone suppression test (DST) in psychiatry. Especially in North America the DST is widely used as a routine laboratory test for (endogenous) depression. However, some important confounding variables such as reduced caloric intake and catabolic state have been largely ignored in a one-track search for a biological marker. According to most diagnostic classification systems in psychiatry (including the DSM III) weight loss is a common symptom of (major) depression.

We have assessed the effects of weight loss (average 8 kg) in five healthy female subjects of normal weight in a starvation experiment during an initial baseline phase (A), a three week phase of complete food abstinence (B), a phase of weight gain (C) to the original level and a final baseline phase (D). Fasting resulted in a significant impairment of the HPA-function (elevation of 24-hour-plasma cortisol, increase of the time in secretory activity and increase of plasma cortisol half-life). Insufficient suppression of plasma-cortisol was observed after application of 1.5 mg dexamethasone at 11 pm, as shown in the Table. Whilst all DST's in the initial baseline phase were normal, seven out of 14 DST's showed insufficient suppression during the fasting phase (B) and all 11 DST's in the weight gain phase (C) were normal again. During the fasting phase there was also a clear reduction in the plasma dexamethasone levels at 9 am which may have caused insufficient cortisol suppression.

During fasting, basal TSH-values were significantly lowered and the TSH-response to TRH was blunted. Other hormonal axes showed disturbances as well: The growth hormone response to the alpha 2-adrenergic receptor agonist clonidine was blunted