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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