

Correspondence

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DEXAMETHASONE SUPPRESSION TEST AND THYROID STIMULATION TEST IN DEPRESSION DEAR SIR,

Both the dexamethasone suppression test (DST) and the thyroid stimulation test (TST) have been claimed to be laboratory aids in the diagnosis of depression (Carroll, 1982; Loosen & Prange, 1982). However, there are indications that both tests may be abnormal in patients with other psychiatric disorders (Dewan *et al*, 1982; Ferrier *et al*, 1983). Some authors measure more than one endocrine variable in the same patients (Winokur *et al*, 1982; Amsterdam *et al*, 1983), but to our knowledge no direct attempt has been made to compare the relationship between DST and TST in various psychiatric disorders.

We investigated 65 female psychiatric inpatients (age: 21–67 years) who had been free from major psychotropic or hormonal medication for at least 2 weeks before admission. The diagnoses were made using DSM-III criteria: major depression ($n = 21$), schizophrenic disorder ($n = 21$), adjustment disorder ($n = 13$) and alcohol dependence in the 7th–22nd withdrawal day ($n = 10$). All patients were physically healthy without extreme weight loss or dehydration.

TST was performed by injecting 0.2 mg synthetic TRH intravenously at 8.30 after an overnight fast and bedrest; blood samples for TSH assay were taken at 0,

30, and 60 min. Patients with abnormal T_3 or T_4 values in the pre-drug serum were excluded. TSH, T_3 and T_4 were measured by radio-immunoassay, and blunted TSH-response was defined as less than 5 mU/l elevation from baseline after TRH.

DST followed 4–5 days later, with 1 mg dexamethasone by mouth at 22.00 and plasma sampling at 08.00 and 15.00 next day. Cortisol was measured by competitive protein binding, and non-suppression was defined by more than 140 nmol/l cortisol in either sample.

In the total population a significant association was found between the DST and the TST (Table). The two tests showed strikingly similar distribution in patients with major depression, but in the remaining non-depressed group this relationship was no longer demonstrable. Separate analysis of each of the three diagnostic subgroups yielded uniform results: in none of the latter was any apparent association between the test results detectable.

There was quite a high rate of abnormality on both DST and TST in non-depressed patients, in agreement with other results (Banki *et al*, 1984; Arato *et al*, 1983); however, there was a remarkable difference between depressed and nondepressed patients with regard to both tests (67% vs. 39%, respectively).

At present only highly speculative explanations might be proposed for the observation that DST and

TABLE
Dexamethasone suppression test (DST) and thyroid stimulation test (TST) results in female psychiatric inpatients

	Depressed		Not depressed		All patients	
	DST		DST		DST	
	+	–	+	–	+	–
TST (+)	12	2	7	10	19	12
TST (–)	2	5	10	17	12	22

DST (+) means nonsuppression (serum cortisol exceeding 140 nmol/l in either sample), and TST (+) means blunted (less than 5 mU/l) TSH response to 0.2 mg intravenous TRH.

The two tests are significantly associated in the total group ($P < .025$); TST follows DST distribution in depressed ($P < .017$) but not in nondepressive patients ($P = .24$). Fisher's exact probability test.

TST results seem to be closely related in depression but not in other psychiatric disorders. Both endocrine tests are dependent on intact limbic-hypothalamic function involving several neurotransmitter systems (Carroll, 1982; Loosen & Prange, 1982), so it is tempting to suggest that major depression might imply a specific limbic-hypothalamic dysfunction, common to the regulation of both the adrenal and the thyroid axis. Further investigations are needed to explore this phenomenon.

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DEXAMETHASONE SUPPRESSION TEST

DEAR SIR,

Dr Saleem (*Journal*, February 1984, **144**, 181–4) reports that 48% of 59 depressed patients had abnormal suppression on the dexamethasone suppression test (DST), a finding in line with previous studies. However, the conclusions he draws about the relationship between abnormal suppression and anxiety may not be justified for the following two reasons:

- (1) The study was carried out on patients who were receiving medication which affects cortisol output and, consequently, the DST. It is stated that 'The great majority were taking therapeutic doses of psychotropic drugs including tricyclic antidepressants, phenothiazines, lithium carbonate and benzodiazepines . . .' Lithium has been shown to increase cortisol levels (Platman & Fieve, 1968). Benzodiazepines, on the other hand, lower them (Beary *et al*, 1983) and can normalize the DST (Nuller & Ostroumova, 1980). There is, however, a more fundamental criticism of studies which include medicated patients. Psychotropic drugs, by definition, alter mental state (and thus the classification of patients into sub-groups of depression such as 'neurotic' or 'endogenous'). In medicated patients, therefore, it is not really possible to draw conclusions about the relationship between the results of the DST and mental state—even if the medication does not actually affect the DST. This drawback applies to many studies in this field (e.g. Carroll & Davis, 1970; Brown & Shuey, 1980; Carroll *et al*, 1981; Asnis *et al*, 1982).
- (2) There must be doubt about the reliability of the self-administered Leeds General Scale (Snaith *et al*, 1976) in severely depressed patients, especially those with retardation or psychotic features. The other scale which was used, the MADRS (Montgomery & Asberg, 1979) has only one subscale, 'Inner Tension', which relates to anxiety, and other features of anxiety, notably the somatic symptoms, are not measured.

Fuller understanding of the relationship between anxiety and the DST must await studies on drug-free patients, using reliable instruments for measuring mental state.

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