I have recently seen a patient who developed rapid tremor of the eyelids after therapeutic doses of haloperidol, given for an acute relapse of a schizophrenic illness. The patient is a 43-year-old woman with an 11 year history of schizhophrenia, treated with a variety of drugs including chlorpromazine, trifluoperazine, fluphenazine and flupenthixol. She had often experienced tremor in her limbs as a side effect of this medication, but had never had any ocular side effects.

She was readmitted in December 1983 with a relapse of her schizophrenic symptoms and commenced on haloperidol 5 mg b.d. and procyclidine 5 mg b.d. Eighteen days after haloperidol was commenced, the patient complained of "flickering lights" and was noticed to have severe rapid twitching of both eyelids accompanied by a tremor of both arms and legs. Oral procyclidine did not seem to improve the eyelid tremor to any appreciable extent, but it stopped spontaneously a week later when haloperidol was changed to Thioridazine 100 mg od.

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THE DST—A DIAGNOSTIC MIRAGE?

DEAR SIR,

The dexamethasone suppression test (DST) has been under investigation for nearly 15 years, yet neither its reliability nor its validity are clear.

Reputable and scientifically rigorous workers in many centres have tested the claims of Carroll *et al* (1981). Notwithstanding the allowances which have to be made for varying diagnostic concepts, there is still a marked and disturbing lack of consistency in the research findings. These reflect on both the test specificity and sensitivity. For example high rates of non-suppression have been reported for mania, neurotic depression, alcoholism, dementia, anorexia nervosa and even in 'healthy' control subjects. These generally widely diverse and discrepant findings must raise the haunting spectre of psychiatry once more embarking upon the false pursuit of a Holy Grail.

We therefore set out to document the range of serum cortisol values associated with depression and other selected, DSM III based diagnostic groups and to assess the response of serum cortisol to the administration of a standard DST.

One hundred adult patients receiving diagnoses (per DSM III criteria) of major depression (38), dysthymia (19), mania (13), schizophreniform disorder or schizophrenia (30) were accepted into the study provided that they had none of the contraindications to a valid DST.

A 4.00 p.m. baseline serum cortisol (Diagnostic Products Corporation, RIA) determination was performed a minimum of 48 hours after admission. That evening 1 mg of dexamethasone was given at 11.00 p.m. and blood samples for cortisol analyses were taken the next day at 1600 and 2300 hours.

Results: The mean base-line blood cortisol levels did not differ significantly between the groups major depression, dysthymia, mania and schizophrenia. Most groups have followed Carroll's lead and adopted the 138 nmol/l (5 μg/dl) criteria for non-suppression. At that level our rates of non-suppression were, major depression 38 per cent, schizophrenia 20 per cent, mania 46 per cent and dysthymia 32 per cent. However, inspection of our data for major depression indicated that a cut off at 210 nmol/l gave the best compromise between specificity (83 per cent) and sensitivity (38 per cent). The rates of non-suppression were then markedly lower in the non-depressed (schizophrenia 10 per cent, mania 31 per cent, dysthmia 16 per cent).

The fact that the rate of non-suppression for major depressive illness was very much lower than the rate found by Carroll *et al* (1981) could reflect the broader group subsumed under that DSM III label. The relatively high rates of non-suppression in the other diagnostic groups, consistent with the work of many others, needs explaining.

For test specificity the base population definition is important. Specificity in this case is the rate of suppressors in persons who do not have the disorder. Is that latter category (the non-disordered) to be the general, normal population, which is not very relevant in the clinical situation; or is it the non-depressed psychiatric patient population; or is it the non-melancholic, but depressed population?

Our results, and those of a number of other workers, do not support the use of the test as a (specific) pointer towards the diagnostic label of depression, in a general psychiatric population. There are positives in too many patients appropriately classified elsewhere. The high rate of non-suppression in other disorders also militates against placing any reliance on this test in those particular clinical situations where our current phenomonologically based diagnostic criteria are most vulnerable. For example the high rate of non-suppression in the demented renders the test useless in distinguishing the pseudo-demented. It would appear that the dysthymic can not be clearly separated from those with major depression.

The evidence concerning the value of the dexamethasone suppression test is consistent with the idea that non-suppression may simply be a measure of the severity of the clinical state. Relative non-suppression of cortisol could reflect the degree of that person's

deviation from his normal state, independent of the direction of that move. The severely anorectic, the demented, the most dysphoric, dysthymic or schizophrenic patients and the most depressed, suicidal people may be those who tend not to suppress their cortisol. This view needs further investigation and would be consistent with the association of non-suppression with weight loss (Edelstein *et al*, 1983; Berger *et al*, 1983), starvation (Smith *et al*, 1975) and with other, allegedly less specific dynamic hormonal changes in depression (Meltzer *et al*, 1982; Amsterdam *et al*, 1983).

The enthusiasm with which the dexamethasone suppression test has been hailed is probably more a reflection of psychiatry's desire for diagnostic advancement and greater medical acceptance that it is of the value of the test.

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ELECTROLYTE CHANGES IN PSYCHOSIS

DEAR SIR,

I read with interest the report by Drs Lever and Stansfeld (*Journal*, 1983, **143**, 406–410), discussing the relationship between Addison's disease, psychosis and inappropriate ADH secretion. I would like to report a case with interesting electrolyte changes which may add another dimension to the discussion.

A seventy-five year old lady was admitted with a three year history of deteriorating memory and flattening of affect. More recently she had begun to lose her appetite, had lost weight and had been wandering about the streets inappropriately. She had been investigated in a general hospital for weight loss, with negative results. On admission she was retarded, withdrawn and speaking in a depressed way, e.g. "I'm so worried I could cry my eyes out" and had defects in cognitive function. She refused to eat or drink, and for several days her fluid intake was less than 500 millilitres per 24 hours.

Serum urea and electrolytes were: (normal ranges in brackets) sodium 130 mmol/L (137–145 mmol/L), potassium 2.7 mmol/L (3.6–4.9 mmol/L) chloride 86 mmol/L (95–105 mmol/L), urea 7.6 mmol/L (3.3–6.6 mmol/L) and bicarbonate 26 mmol/L (22–27 mmol/L). Urine potassium and sodium levels were low and serum osmolality was 281 mosm/L (280–295 mosm/L).

She was persuaded to take anti-depressants (amitriptyline 100 mg per day) and within a fortnight had lost her depressive features and was eating and drinking normally, although her cognitive deficits remained. Serum electrolytes returned slowly to normal over four weeks and her weight increased from 40 kg on admission to 49 kg.

A possible explanation of the results is that the primary factor was a depressive illness, causing a decrease in the intake of food and fluid. This in turn led to sodium and water depletion, a reduced renal blood flow and secondary aldosteronism (low urinary sodium). ADH secretion would then be stimulated, accounting for the deranged electrolyte levels. The high bicarbonate level may be explained by a mild metabolic alkalosis consequent on hypokalaemia. As food and fluid intake re-established themselves the electrolyte levels returned to normal. The fact that full remission occurred with dietary correction only and that we know her serum electrolytes were normal prior to the loss of appetite (from a previous admission) suggests that this was the only pathological process.

That salt and water depletion occur with starvation is well recognised (Gamble *et al*, 1923; Sandek and Feliq, 1976) and the sequential biochemical processes have been documented (Zilva and Pannall, 1978). Crammer (1959) found that electrolyte changes in psychosis were cyclical and independent of food and fluid intake and postulated a more complex relationship between electrolyte levels and psychosis. The temporal sequence of events in the above patient cannot, however, be denied.

This case raises the possibility that electrolyte