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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References

AMSTERDAM, J. D., WINOKUR, A., LUCKI, I., CAROFF, S., SNYDER, P. & RICKELS, K. S. (1983) A neuroendocrine test battery in bipolar patients and healthy subjects. *Archives of General Psychiatry*, **40**, 515–21.

BERGER, M., PIRKE, K., DOERR, P., KRIEG, C. S. & VON ZERSSEN, D. (1983) Influence of weight loss on the dexamethasone suppression test. *Archives of General Psychiatry*, **40**, 585–68.

CARROLL, B. J., FEINBERG, M., GREDEN, J. F., TARIKA, J., ALBALA, A., HASKETT, R., McI JAMES, N., DRONFOL, Z., LOHR, N., STEINER, M., DE VIGNE, J. & YOUNG, E. (1981) A specific laboratory test for the diagnosis of melancholia. *Archives of General Psychiatry*, **38**, 15–22.

EDELSTEIN, C. K., ROY-BYRNE, P., FAWZY, F. I. & DORNFELD, L. (1983) Effects of weight loss on the dexamethasone suppression test. *American Journal of Psychiatry*, **140**, 338–41.

MELTZER, H. Y., FANG, V. S. TRICOU, B. J., ROBERTSON, A. & PIYAKA, S. K. (1982) Effects of dexamethasone on plasma prolactin and cortisol levels in psychiatric patients. *American Journal of Psychiatry*, **139**, 763–8.

SMITH, S. R., BLEDSOE, T. & CHHETRI, M. K. (1975) Cortisol metabolism and the pituitary adrenal axis in adults with protein caloric malnutritions. *Journal of Clinical Endocrinology and Metabolism*, **40**, 43–52.

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

changes may occur independently of any neuroendocrine process i.e. from starvation although may still be an integral part of a psychiatric disorder. Furthermore it emphasises the importance of blood biochemistry in understanding the clinical picture and may strengthen the argument for routine biochemical investigations in psychiatric patients.

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References

CRAMMER, J. L. (1959) Water and sodium in two psychotics. *Lancet, i,* 1122–6.

GAMBLE, J. L., ROSS, G. S. & TISDALL, F. F. (1923) The metabolism of fixed base during fasting. *Journal of Biological Chemistry,* **57,** 633–95.

SANDEK, C. O. & FELIQ, P. (1976) The metabolic effects of starvation. *American Journal of Medicine,* **60,** 117–26.

ZILVA, J. F. & PANNELL, P. R. (1978) *Clinical Chemistry in Diagnosis Treatment.* 2nd edition. London: Lloyd-Luke.

PATTERNS OF CARE FOR THE DEMENTED

DEAR SIR,

The paper by Christie and Train (*Journal,* January 1984, **144,** 9–15) made several salient points about the provision of in-patient beds for the demented elderly. They also presented data showing that the provision of day care and/or "holiday" admissions prior to the final admission had no significant effect on the patients' ultimate length of stay in hospital. From this they concluded that "treatment, or what might more appropriately be described as support for patients and relatives is shown to have had no effect in reducing the duration of terminal hospitalisation".

While this may be so, it is not a logical conclusion on the basis of the data presented. Day care places and holiday admissions were not allocated on a random basis, and were thus presumably a reflection of perceived greater need. Most commonly the greater need would be that of the demented patient's relatives, rather than of the patient herself. Given that the more stressed and/or "help-seeking" relatives tend to be the recipients of this sort of assistance, one could view the fact that the "treated" patients do not require longer final hospitalisations as evidence that day care and holiday admissions are doing just what they ought to be doing i.e. providing sufficient support to enable stressed relatives to cope with a demented dependant at home for as long as their less stressed counterparts.

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DEXAMETHASONE SUPPRESSION TEST PREDICTS RESPONSE TO NOMIFENSINE OR AMITRIPTYLINE

DEAR SIR,

The dexamethasone suppression test (DST) has recently attracted considerable interest among biological psychiatrists. While its specificity for the diagnosis of endogenous or primary depression has not been confirmed fully it is conceivable that it could have application as a tool for the exploration of neurotransmitter dysfunctions in the limbic–hypothalamic system. A pathological (i.e. positive) DST might be secondary to either a decrease of noradrenergic activity (van Loon *et al,* 1971) or increased cholinergic activity within the central nervous system (Garver & Davis, 1979). Data on the involvement of serotonin on ACTH release are contradictory.

Using the DST as a peripheral indicator we investigated the possibility of a central noradrenergic–cholinergic imbalance in subgroups of depressed patients (Janowsky *et al,* 1972).

In 43 depressed inpatients the DST was performed. Subsequently, a group (n = 23) of DST positive and a group (n = 20) of DST negative depressives were treated for 28 days under double blind conditions with either nomifensine (150–300 mg/day), a noradrenaline (NA) potentiating drug, or amitriptyline (150–300 mg/day) a NA potentiating and potent anticholinergic compound.

DST positive depressives responded favourably to amitriptyline, but not to nomifensine. Conversely, DST negative depressives responded favourably to nomifensine but less well to amitriptyline. (Table).

TABLE

Clinical response in depressed patients with pathological (+) and normal (−) dexamethasone depression test (DST) treated with amitriptyline and nomifensine. Response is defined as decrease of Hamilton Depression Rating scale global score by 50 per cent within 28 days

	DST	Response	
		<50%	>50%
Amitriptyline (n = 20)	+ (n=10)	2 20%	8 80%
	— (n=10)	5 50%	5 50%
Nomifensine (n = 23)	+ (n=13)	8 62%	58 38%
	— (n=10)	3 30%	7 70%

https://doi.org/10.1192/bjp.144.4.439 Published online by Cambridge University Press