British Journal of Psychiatry (1984), **145,** 555–559

Correspondence

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ECT, DOPAMINE AND DELUSIONS

DEAR SIR,

We were interested to read the comments of Dr Eagles (*Journal,* June 1984, **144,** 670) as these are similar to some raised previously by ourselves (Cooper & King, 1982). We would like to support the view that ECT may have its effects specifically on what might be termed "positive" psychotic symptoms, such as delusions etc., irrespective of whether they occur within the context of schizophrenia, mania or endogenous depression and suggest that this is because ECT reduces dopaminergic transmission.

We have studied the effects of ECT on amine metabolite concentrations measured in lumbar cerebrospinal fluid (CSF) samples collected from 9 schizophrenic patients during a course of 6 ECT treatments (paper in preparation). The patients conformed to standard diagnostic criteria and ECT was given twice weekly using an Ectron Mark 4 apparatus. Lumbar CSF was collected by a standardised technique 24 hours before the first ECT treatment and 24 hours after the first and sixth treatments. A significant elevation (P 0.01, Wilcoxon Test) of HVA concentrations was found after the first treatment but not after the sixth. This previously unreported elevation of dopamine turnover followed by the development of biochemical tolerance was accompanied by clinical improvement and is similar to the time dependent effect of neuroleptic drugs on dopamine turnover (Post & Goodwin, 1975). It can be interpreted as indicating a decrease in dopaminergic transmission with an initial increase in dopamine turnover, mediated through negative feedback mechanisms, to which tolerance then develops, as it does with neuroleptic drugs.

Previous attempts to examine the effect of ECT on dopaminergic transmission through neuroendocrinological investigations in patients have produced conflicting results (Cooper & King, 1982), though some would support our contention, such as the demonstration that serum prolactin concentrations may rise following ECT. Evidence from previous clinical studies also supports our view. ECT is effective in the treatment of schizophrenia and mania, conditions in which increased dopaminergic transmission is important. More intriguing, however, are the results of recent studies of the effects of ECT in endogenous depression, which suggest benefit only (Clinical Research Centre, 1984) or mainly (Brandon *et al,* 1984) in patients with delusions. It has previously been noted that the addition of neuroleptic drugs to other antidepressant treatment aids the recovery of deluded depressed patients (Kaskey *et al,* 1980) implying that increased dopaminergic transmission might have a role in some of their symptoms.

Thus clinical evidence and our present results support the hypothesis that ECT may have a general antipsychotic effect mediated through dopaminergic mechanisms.

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DST IN MANIA: PROLONGED SUPPRESSION?

DEAR SIR,

The study of cortisol secretion in mania has produced inconsistent results. However, Bunney *et al*

(1965) investigated a patient with 48 hour manic depressive cycles and found low urine 17 hydroxy-corticosteroids during the manic days and high levels during the depressed days. Longitudinal studies in patients with manic depressive psychosis have shown lower cortisol secretion in the manic state and hypersecretion during depression (Ettigi & Brown, 1977). As far as the dexamethasone suppression test is concerned, patients in the manic or hypomanic phase do not usually exhibit non-suppression or early escape from suppression (Carroll, 1982). We wondered if manic patients would show a prolonged suppression of cortisol in response to dexamethasone.

Eleven manic patients and 12 schizophrenic patients without prominent affective features formed the subjects of the study. All patients satisfied the appropriate DSM III criteria. Conditions likely to interfere with DST were excluded. Blood was drawn for baseline plasma cortisol estimation at 11.30 p.m. prior to the administration of 0.5 mg of dexamethasone orally. It was decided to use this smaller dose of dexamethasone to accentuate any difference between the two groups. Blood samples were drawn at 11.30 p.m. on day II and 8.00 a.m. and 11.30 p.m. on day III. Plasma cortisol was estimated using the radio immuno assay technique. Two of the manic patients were given barbiturates during the period of DST and had to be excluded from the study. All the other patients were on various anti-psychotic drugs and two patients (one manic and one schizophrenic) were on small doses of orphenadrine hydrochloride. All the manic patients remained in a manic state for at least one week after the DST was completed. A baseline cortisol level of less than 2 μgm/DL was considered to be below normal and patients with post-dexamethasone cortisol levels of less than 6 μgm/DL were considered to show suppression.

Two out of nine manic patients and five out of twelve schizophrenic patients showed baseline cortisol value. There was no significant difference in the proportion of patients showing suppression between the two groups on any of the three post-dexamethasone cortisol assays ($P = 0.714$; $P = 0.429$; $P = 0.414$ respectively—Fischer's Exact Test). In view of the problems involved in the interpretation of changes of cortisol values following the administration of dexamethasone in patients who prior to the administration showed low cortisol values, the analysis was repeated using only those cases whose baseline values were normal or high (seven manics and seven schizophrenics). No significant differences were found between the two groups.

Amongst the difficulties in interpreting the data are the small sample size and the absence of normal controls. It is interesting to note that the only patient who showed a suppressed cortisol level (following dexamethasone administration) on day III at 8.00 a.m., a time when a cortisol spurt would normally be expected, was a manic who showed a higher than normal midnight baseline cortisol level. This makes us wonder if any prolonged post-dexamethasone cortisol suppression might exist only in the manics who show an elevated (above normal) midnight baseline cortisol and whether this might be best studied by measuring the 8.00 a.m. post-dexamethasone cortisol levels.

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NEUROLEPTIC MALIGNANT SYNDROME AND HEART STROKE

Dear Sir,

Dr Singh (*Journal*, July 1984, **145**, 98), in his discussion of neuroleptic malignant syndrome, makes the erroneous assertion that loss of consciousness and absence of sweating are classic signs of this disorder. Though confusion and altered consciousness are frequently mentioned in reported cases (Caroff, 1980), actual loss of consciousness is not commonly found. Rather than an absence of sweating, a profuse diaphoresis is usually seen (Caroff, 1980; Smego & Durack, 1982; Ayd, 1983; Szabadi, 1984). In the case presented by Singh, the abrupt loss of consciousness coupled with an elevated temperature and an absence of sweating on an "unusually hot" day, suggest that the patient suffered from heat stroke, not NMS. Neuroleptics may contribute to heat stroke by inhibiting the sweating mechanism (Smego & Durack, 1982). Though rigidity, which was present in this case, is not a common feature of heat stroke, it is a common side effect of neuroleptic use and therefore should not deter the clinician from entertaining this diagnosis when the appropriate signs are present. Also, a more comprehensive presentation of data such as CPK, WBC count,