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Meeting

BIOLOGICAL PSYCHIATRY GROUP

Brief summaries of a selection of the papers read at the last two meetings of the Biological Psychiatry Group of the Royal College of Psychiatrists on 21 November 1980 and 11 February 1981 follow below.

Value of routine chest radiography of psychiatric patients. J. HUGHES, *Department of Psychiatry, University of Southampton*

Chest X-rays of 231 consecutive patients in an acute psychiatric ward yielded 21 with clinical abnormalities and two young alcoholics with active TB. However, all already had clinical symptoms or signs of physical disease. A chest X-ray seems unnecessary therefore, except in patients with clinical evidence of physical disease or alcoholism.

Adult psychiatric health of children who had experienced chronic temporal lobe epilepsy. C. OUNSTED, *The Park Hospital for Children, Oxford*

One hundred children with temporal lobe epilepsy were followed into adult life: 85 per cent had had psychiatric problems in childhood, but overt psychiatric disorder in adult life was low. Ten per cent of survivors showed a schizophreniform psychosis, one-third of the males with continuing epilepsy and left-sided foci becoming psychotic. No patient coded as having a right-sided focus in 1964 had become psychotic by 1977. Only five survivors received treatment for neurotic or depressive illness. Twelve showed anti-social conduct (males, unremitted epilepsy, focus contralateral to preferred hand). Though 26 patients had been coded as children as having a grossly disordered family life, this had no relationship to adult psychiatric disorder.

Prolonged ambulatory monitoring in neuropsychiatric patients. GREGORY STORES, *Park Hospital for Children and University of Oxford, Department of Psychiatry*

Technical advances have enabled EEG recordings to be carried out over days or weeks in everyday situations, in the investigation of patterns of occurrence of seizure activity and the quantification of response to anti-epileptic treatment, in non-convulsive forms of epilepsy for instance. Prolonged EEG monitoring by unobtrusive means in the home environment during sleep is now possible.

Plasma drug and prolactin levels during maintenance treatment with depot neuroleptics. T. KOLAKOWSKA and D. WILES, *University of Oxford, Department of Psychiatry, Littlemore Hospital Research Unit, Oxford*

Plasma prolactin (PRL) levels were similar in men receiving comparable doses of fluphenazine (FPZ) or flupenthixol decanoates whether the duration of treatment was 1–2 months (n = 8) or several years (n = 25). When drug injections were stopped in four patients, the decline in both plasma drug and PRL levels was very slow: FPZ levels were down by only 40–60 per cent after two months, and in two patients who had been receiving FPZ 25 mg every two weeks, an FPZ level of about 1 ng/ml was found five months after withdrawal, the kind of plasma level achieved with 12.5 mg FPZ per 1–2 weeks. Plasma PRL, above normal in three patients, was still elevated after two months, and remained so for over four months in two women observed.

Age and mortality in dementia. ANTONIA WHITEHEAD, *Kingston and Richmond Area Psychologist, Long Grove Hospital, Epsom*

Data were presented from a five-year prospective study of elderly patients admitted to psychiatric beds. About three-quarters of those with an admission diagnosis of chronic brain syndrome had died within the five years. For the less elderly (i.e. 60–74 year old) this mortality risk was far in excess of that to be expected for their age; for the more elderly, however, the risk was no higher than that for very elderly patients with functional disorders. Thus, from the point of admission, dementia runs a time-limited course irrespective of age. However is 'senile dementia' a single condition or a number of processes?

Unipolar affective disorder, 'stress' and total concentrations of tryptophan in plasma. D. M. SHAW, S. F. TIDMARSH and B. M. KARAJGI, *Department of Psychological Medicine, Welsh National School of Medicine*

Mean total tryptophan concentrations in plasma ([T] in μM) have been studied in fasting subjects in

two investigations. In one, the patients were inadvertently stressed by anticipation of a prolonged kinetic study, and in the second gave just a single blood sample, and were presumably without significant stress.

[T] in the unstressed group was 72.4 ± 2 (mean and SEM, n = 19) and 76.2 ± 2 (n = 24) in controls and depressives respectively (difference not significant). [T] in the stressed group was significantly reduced comparing the two sets of controls, and the two sets of depressives ($P < 0.001$ in each case). [T] in stressed depressives, 60.8 ± 1.8, n = 28, was slightly lower than in comparable controls; 65.8 ± 1.4, n = 46, $P < 0.05$. Ten stressed depressed patients on tricyclic antidepressants had significantly lower values than the similar drug-free group ($P < 0.001$).

In 10 unipolar patients, [T] 1½ hours before the first of an ECT course was 54.9 (1 male, 9 females).

Males tended to have higher values than females and to have smaller differences between groups. Age and tryptophan levels showed no correlation.

Effects of amitriptyline and desipramine on cholinergic and adrenergic responses in the human iris. E. SZABADI, P. GASZNER and C. M. BRADSHAW, *Department of Psychiatry, University of Manchester*

The effects of amitriptyline (25, 50, 100 mg) and desipramine (25, 50, 100 mg) were compared on pilocarpine evoked miosis (8 subjects), noradrenaline-evoked mydriasis (5 subjects), and methoxamine-evoked mydriasis (4 subjects), in healthy volunteers. Both antidepressants reduced the pupillary effects of pilocarpine and methoxamine, and increased the effect of noradrenaline. Amitriptyline was more potent in antagonizing the effects of pilocarpine and methoxamine, and desipramine was more potent in potentiating the effect of noradrenaline. These results are consistent with the greater potency of amitriptyline in blocking muscarinic and α-adrenoceptors, and with the greater potency of desipramine in blocking noradrenaline uptake.

Prediction of response to lithium prophylaxis in manic depressive illness. M. T. ABOU-SALEH, *West Park Hospital, Epsom*

In a series of bipolar cases clinical variables did not discriminate between good and poor responders, nor did calcium binding or platelet MAO, but on the Eysenck and Foulds scales poor responders were more neurotic, less extraverted and had higher scores on psychoticism, lie scale, and intropunitiveness. Patients who responded well had significantly lower percentages of HLA-BW16 and HLA-A9 and higher percentages of HLA-B5 and HLA-B8. It was suggested that HLA-BW16 might be a marker for poor response to lithium prophylaxis.

The time course of the anti-psychotic effect in schizophrenia. T. J. CROW, C. D. FRITH, EVE C. JOHNSTONE and D. G. C. OWENS, *Division of Psychiatry, Clinical Research Centre, Northwick Park Hospital, Watford Road, Harrow HA1 3UJ*

It was previously suggested that the time course of the anti-psychotic effect of neuroleptic drugs in schizophrenia was rather slow. In a clinical trial of the efficacy of the isomers of flupenthixol, clinical improvement appeared to follow two weeks after the rise in prolactin. This suggested that dopamine receptor blockade was perhaps necessary for some other process with a slow time course to take place. New evidence casts doubt on this conclusion. In a recent trial of the effects of anti-cholinergic medication added to maximal doses of flupenthixol, the addition of the anti-cholinergic was found to reverse some of the therapeutic benefits of the neuroleptic. Moreover, the rate of improvement was much faster in patients who did not receive anti-cholinergic medication and closely paralleled that of the development of extra-pyramidal side effects. The simplest explanation appears to be that both are due to dopamine receptor blockade.

Some clinical and metabolic aspects of propranolol in chronic schizophrenia. DAVID J. KING, *Department of Therapeutics and Pharmacology, Queen's University of Belfast, and Holywell Hospital, Antrim*

Five chronic schizophrenic men, four weeks off all drugs, were given propranolol alone (1000 mg/day) for six weeks without improvement. There was some improvement when trifluoperazine (10 mg/day) was added. Mean basal prolactin levels were non-significantly reduced by propranolol, but there was no change in response to metoclopramide, indicating no change in DA receptor function. CSF HVA was significantly elevated after one week on propranolol and returned to normal at the end of the trial. Thus there was an increase in DA turnover without any alteration in central DA receptor activity. CSF 5HIAA was also elevated but MHPG was slightly decreased. The changes in HVA and 5HIAA but not in MHPG were confirmed in a second study. The HVA changes could be secondary to central 5HT blockade.