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Meeting

At the Annual Meeting of the Royal College of Psychiatrists, its Research Committee organized a special session for 7 July. The programme was in two sections, and abstracts follow.

DOES IT WORK?

Is ECT Effective? Preliminary results of a controlled trial. JOHN LAMBOURN and DAVID GILL. Knowle Hospital, Fareham, Hants.

ECT has never been adequately tested against placebo, but is applied empirically, in different forms, for the alleviation of several conditions. The efficacy of one waveform and electrode placement was therefore compared with placebo in the treatment of psychotic depressive illness, the major indication for ECT. Patients suffering no other psychiatric or organic state were assigned in a constrained random manner, forming two groups of 16, matched for age and sex. The match was also good for previous admissions, use of ECT, and tricyclic failure.

Psychoactive medication was stopped, and all received a standardized intravenous anaesthetic regime, plus the application of right unilateral electrodes (Lancaster's position) thrice weekly. This constituted the placebo, while the active group also received a brief pulse stimulus, and exhibited a bilateral modified convulsion every time.

Change was rated blind in four ways. Hamilton rating showed only a trend in favour of ECT (improvement of 53 per cent against 43 per cent). This was partly due to physiological effects interfering with the rating measures. Leeds self assessment scores corrected for side effects showed ECT and placebo equally effective on the depression ratings, but ECT was significantly superior on the anxiety ratings. Global improvement was rated only marginally better by the referring doctors, who failed to break the blind.

One month follow-up produced comparable outcomes for days in hospital, Hamilton rating, additional ECT, and antidepressant medication.

The following conclusions were made. Six brief pulse unilateral ECTs, in the treatment of psychotic depressive illness:

1. Had a good effect on the depressive features, but only equal to placebo.
2. Had a superior effect compared with placebo on the anxiety features.
3. Produced an interference from side effects with conventional rating scales.

The study did not demonstrate this particular application of ECT grossly superior to placebo, neither did it show ECT to be worthless. The large placebo effect justifies further such studies, opening a new phase of ECT research.

A Controlled Study of the Effect of the Samaritans on the Suicide Rate of England and Wales: Negative Findings. C. JENNINGS, B. BARRACLOUGH and J. MOSS. MRC Clinical Psychiatry Unit, Graylingwell Hospital, Chichester, Sussex.

The claim has been made that the Samaritan Organization is responsible for the fall in the suicide rate in England and Wales since 1963. Bagley (1968) found that the suicide rate in towns with Samaritan branches fell while the rate in matched control towns rose and that this difference was significant. We replicated his study using better methods of choosing controls. Method I used Bagley's fifteen Samaritan towns and found control towns most similar on the four principal components derived by Moser and Scott (1961). These components represented, in order of importance, 'social class', 'population change, 1931–51', 'population change, 1951–58' and 'overcrowding'. Method II derived twenty-three pairs of Samaritan towns and controls matched on these four components. In comparison, Bagley used only the first two components in his study. Method III matched Samaritan towns and controls on their suicide rate before the opening of the branch. Method IV matched on the proportion of single-person households. The matching variables used in Methods III and IV were more highly correlated with the subsequent suicide rate than were the variables used in Bagley's second method. No significant differences were found when the changes in the suicide rates of the Samaritan towns were

compared with those of the control towns. Our methods produced closer matching between Samaritan and control towns and larger samples than Bagley's study. The results are in line with controlled evaluations of American Suicide Prevention Centres and with Holding's (1975) study of the Samaritans and do not support the hypothesis that the fall in the suicide rate is due to the Samaritans.

References

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Crisis Intervention after Deliberate Self-Poisoning: A Trial of Task Centred Casework. J. S. GIBBONS, J. BUTLER, P. J. URWIN and J. L. GIBBONS. Department of Sociology and Social Administration, and Faculty of Medicine, University of Southampton.

Four hundred patients, aged at least 17 and coming from a defined geographical area, who attended the Casualty Department of Southampton General Hospital between April 1975 and March 1976 after deliberate self-poisoning, were randomly assigned to an experimental, crisis-oriented social work service (E) or to the routine service (C) (referral to a general practitioner, to a psychiatric out-patient clinic or to some other form of support). A further 139 patients were excluded because they needed immediate psychiatric intervention or were already in intensive treatment, usually with a psychiatrist. The experimental service used a contractual approach, setting a maximum time limit of 3 months service.

After a year there was no difference between E and C groups in the proportion of patients who repeated self-poisoning. The excluded patients repeated over twice as much. E patients were significantly more satisfied with the service they had received and showed more changes in some areas of social functioning.

TRANSMITTERS IN DEMENTIA

Cholinergic Mechanisms in Alzheimer's Disease. PETER DAVIES. MRC Brain Metabolism Unit, Department of Pharmacology, 1 George Square, Edinburgh.

We have attempted to evaluate the status of various neurotransmitter systems in clearly defined cases of this condition measuring key enzymes, receptors, and in some cases the transmitter and/or its metabolites in brains obtained at autopsy. Over a period of two years, seven brains have been collected which satisfied our criteria for acceptance as cases of Alzheimer's disease. Clinically they showed profound progressive dementia, and neuropathologically the presence of great numbers of neuritic plaques and neurofibrillary tangles in sections of frontal, temporal and parietal cortex, without evidence of significant cerebro-vascular disease. A further fourteen brains were collected from cases free of obvious neurological or psychiatric disorder, and in which no gross cerebral abnormalities were detected.

By far the most striking neurochemical abnormalities we have found in the brains from cases of Alzheimer's disease are reductions to less than 25 per cent of normal values in the activities of two enzymes, choline acetyltransferase (the enzyme responsible for the synthesis of acetylcholine) and acetylcholinesterase (responsible for its degradation) in hippocampal, temporal, frontal and parietal cortex (Davies and Maloney, 1976). These same brain regions show the most extensive pathological changes which are characteristic of Alzheimer's disease.

The activities of these two enzymes in hippocampus, temporal cortex and parietal cortex are almost without exception below the lower end of the normal range of activities. In other brain regions, whilst the mean activities of these enzymes are lower than in normals, there is considerable overlapping of the data from normals and Alzheimer's disease cases. The data seem to suggest that central cholinergic neurones generally are vulnerable to whatever agent or process produces Alzheimer's disease. In certain brain regions however, these neurones appear to be particularly badly affected, and it seems possible that the extent of the loss of enzymes of the cholinergic system in hippocampal, temporal and parietal cortex indicates a functional deficit in cholinergic transmission in these areas. Whether this apparent deficiency can explain any or all of the clinical manifestations of this condition, such as memory loss and spatial disorientation, is not clear but there are grounds for thinking that this may be so (Drachman and Leavitt, 1974). Whether the smaller losses of these enzymes from other brain regions are indications of functional deficiencies of acetylcholine or not is likewise unclear. The extent of the normal range of values, and of the overlaps between data from normals and cases of Alzheimer's disease suggests that post-mortem measurements of enzyme activities may not be a sensitive indicator of the previous cognitive function of an individual.

To date, there has been no clear indication that neurotransmitter systems other than the cholinergic are significantly affected in Alzheimer's disease, and