CAPGRAS' SYNDROME AND CEREBRAL DYSFUNCTION

DEAR SIR,

Doctors Hayman and Abrams (1) in their article of the above title have suggested that prosopagnosia (face non-recognition) could be due to specific cerebral dysfunction, and MacCallum (1973 (2) has reported a case of 'Capgras' Syndrome' caused by cerebral anoxia due to broncho-pneumonia. In this connection I wish to report the following case:—

An 85-year-old married man was admitted with a history of confusion of a day's onset when he misidentified his wife as a strange woman and thought his true wife had been taken away from him by an outside agency and that this strange woman was acting for that agency. He had a history of being depressed before and was being treated by his general practitioner with amitriptyline 25 mgms t.d.s. for the preceding three weeks. On admission he had a pulse rate of 46 per minute, and apparently had been told when in the Army, that he always had a slow pulse. An ECG showed complete heart block with coupled ventricular ectopics. Amitriptyline was discontinued and his confusion improved. It was thought that this episode of confusion and prosopagnosia was probably ischaemic secondary to an arrhythmia caused by amitriptyline. He was referred to a cardiologist who put in a pacemaker, and has since been physically and mentally well.

S. WAGHRAY

Whitchurch Hospital,
Whitchurch, Cardiff CF4 7XB

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EEG STUDIES IN MALE POLYGONOSOMIC PSYCHOPATHS (47, XYY AND 47, XXY)

DEAR SIR,

Some data have been reported about polygonosomic patients (i.e. patients with supplementary X or Y chromosome), that suggest the idea of a minimal brain dysfunction (1, 2) which causes more vulnerability to the environment (3). Further data can be obtained with cerebral evoked responses (ER) and event-related slow potentials.

We have studied seven 47, XYY and four 47, XXY adult psychopaths (27 ± 6.6 years), from our psychiatric security setting who had had no previous treatment and compared them with a control group of 11 patients of the same age (27.5 ± 6.5 years). We recorded ER (Visual, Auditory, Somato-sensory) and Contingent Negative Variation (CNV) as usual in our laboratory (4).

The table shows there are some significant differences between 47, XYY and controls, and between 47, XYY and 47, XXY (statistical analysis performed according to non-parametric C1 test of Fisher-Yates-Terry (5). Fewer differences are observed between 47, XXY and control patients (see Table p. 288).

In 47, XYY there is a significantly higher amplitude in the latest components of VER, AER and SER (II = secondary complex related to the integration where latencies are more than 100 ms), and in the After Discharge (AD) as in hypovigil levels of consciousness (such as drowsiness) (6). The amplitude of the earlier components (I = primary complex corresponding to the afferents) is not affected, and there is no significant difference in latencies. ER in 47, XXY do not differ from controls. In 5 out of 11 polygonosomics there is an abnormal CNV (less amplitude and abnormal waveform called 'late CNV' with a persistant negativity after the motor response) as described in some psychiatric disorders (7, 8, 9).

Electrophysiological multiparametric outlines in psychiatric or sociopathic disorders could show some correlations with character features: ER discriminates 'hypovigil' 47, XYY and 'hypervigil' 47, XXY sociopaths with a lesser interindividual variability than in controls; the abnormality of CNV found on both polygonosomics can be related to a motivational dysfunction which indicates some particularities of the subject's attitude in expectancy and stressing situations (8).

J. PATY

Service d'Exploration Fonctionnelle
du Système Nerveux,
Hôpital Pellegrin—Tripode,
2, place Amélie Raba-Léon,
33076—Bordeaux, France

M. BENEZECH

Service de Sûreté,
Centre Hospitalier Spécialisé,
33410—Cadillac sur Garonne, France

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INHERITANCE OF ALCOHOLISM

Dear Sir,

We have read Cadoret and Gath's paper, 'Inheritance of Alcoholism in Adoptees' (*Journal*, March 1978, **132**, 252–8) with considerable interest. Nonetheless, we do not feel that their findings support their conclusions.

Leaving aside the fact that only 84 parents of the original 173 adult adoptees and only 45 of the adoptees themselves could be interviewed, we would like to question the separation of primary from secondary alcoholism in this study. This distinction presupposes an absence of predisposing psychologicol factors, but the authors themselves clearly have some doubts on whether a diagnosis of primary alcoholism in patients with a previous history of childhood conduct disorder can be justified. We also note that in one adoptive family, where the adoptee was an alcoholic, alcoholism in the biological parents was known to the adoptive parents. One can only conjecture to what extent this knowledge was relevant to the adoptee's ultimate development, and we feel that in such a case a genetic factor might well be out-weighed by environmental contributions. Hence, if this case is omitted and all alcoholics—primary and secondary—included, one finds that one alcoholic came from six families with alcoholism and seven from families without such a history. The difference using the odds ratio measure of association (Fleiss, 1973) is not statistically significant ($P < .2$). If the two probable alcoholics are added, one compares two such alcoholic adoptees from six alcoholic families with eight from non-alcoholic families. Again the difference is not statistically significant ($P < .1$).

The highest degree of statistical significance claimed in this paper ($P = 0.0006$) is based on the pooling of data on first and second degree relatives of adoptees with primary and secondary diagnoses of alcoholism. However, since secondary alcoholism is stated not to appear to be associated with any particular diagnosis in the biological parents, the validity of this procedure must be open to question. Examination of Table Ib shows that of 6 cases where adoptees had alcoholism as a secondary diagnosis, depression appears as the primary diagnosis in 4 and 'bipolar mood swings' in a fifth, suggesting the presence of a confounding variable which Cadoret and Gath do not consider: a genetic loading for affective illness. But what then is to be learnt from the absence of affective disturbance in the biological parents of the secondary alcoholics?

In their quest for satisfactory data, the authors in some instances had to base the diagnoses of biological parents on 'vague remarks alluding to behavioural problems'. If such uncertainty surrounds the diagnoses of first degree relatives, what reliance can be placed on the diagnoses of second degree relatives? And can we be certain that all of these were known to the authors?

The aetiology of alcoholism is complex and varied depending as it must on a host of environmental factors, not the least of which are the availability of alcoholic beverages, attitudes of relatives and friends to drinking and the cultural determinance of patterns of drinking behaviour. In a study of this kind it is impossible to control the influences which have moulded the drinking behaviour of the adopted out individuals. However, until this is done, we contend on present evidence that cultural and environmental factors have far greater impact than any genetic predisposition, apart, possibly, from certain qualities of personality conducive to abnormal drinking behaviour.

F. A. Whitlock
J. Price
M. J. Weston

Department of Psychiatry,
University of Queensland,
Clinical Sciences Building,
Royal Brisbane Hospital,
Brisbane, Queensland 4029

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