

PATTERNS OF CHILDHOOD SOCIAL DEVELOPMENT IN SCHIZOPHRENIA, BIPOLAR DISORDER AND NORMAL CONTROLS

M. Cannon, P.B. Jones, C.M. Gilvarry, A. Foerster, K. McKenzie, L. Rifkin, R.M. Murray. *Department of Psychological Medicine, Institute of Psychiatry, De Crespigny Park, Denmark Hill, London SE5 8AF, UK*

Premorbid adjustment was assessed retrospectively in 100 patients with schizophrenia, (DSM-III-R criteria), 49 patients with bipolar disorder and 100 control patients. Mothers were interviewed using the Premorbid Social Adjustment scale. Principal components analysis on the premorbid rating scores revealed two distinct premorbid factors: (1) 'sociability' and (2) 'school performance'. Schizophrenic patients performed significantly more poorly than control subjects ($p < 0.0001$) in both areas. Patients with bipolar disorder differed from controls only for the 'sociability' factor, (factor 1), with a mean score intermediate between schizophrenic subjects and controls. There was little overlap between the distributions of factor scores in the schizophrenic patients and controls. On factor 1, 82% of the schizophrenic patients scored worse than the 75th centile for control subjects. There was no association between premorbid performance and family history of psychosis, obstetric complications or measures of disease severity such as number of admissions, weeks spent as inpatient or age of onset. An association between low birth weight and poor performance on factor 2, (school performance), was specific for schizophrenia. In conclusion, patients with schizophrenia exhibit abnormalities in all areas of social adjustment in childhood and adolescence. This pattern of social maladjustment appears to be an intrinsic part of the schizophrenic process and is independent of disease outcome.

A TWO YEAR FOLLOW UP STUDY OF THE FIRST 50 PATIENTS TO START CLOZAPINE IN RAMPTON HOSPITAL

B.M. Dalal, E.P. Larkin, P.J. Taylor. *Rampton Hospital, Retford, Nottinghamshire, DN22 0PD, England*

Clozapine is the only antipsychotic drug that has been shown to be consistently superior to other antipsychotics in the treatment of patients with severe chronic schizophrenia. Despite the potential importance of this new treatment, there have been relatively few studies of the effectiveness of Clozapine in offender patient samples.

Case notes of the first 50 patients to start Clozapine in Rampton hospital (a psychiatric hospital for the treatment of patients in conditions of maximum security) were examined retrospectively. The severity of positive symptoms, negative symptoms, violent behaviour and self-harming behaviour were rated according to four-point scales (none, mild, moderate, severe) derived from established instruments. Clinical ratings were made at the start of treatment and after 6, 12 and 24 months.

The mean age (SD) at first contact with psychiatric services was 21 years (4.2).

The mean age (SD) on admission to Rampton Hospital was 30 years (8.2).

The mean age (SD) on starting Clozapine was 39 years (8.8).

Prior to starting Clozapine, the ratings of positive symptoms were severe 88%, moderate 12%. The ratings of violent behaviour were severe 19%, moderate 29%, mild 17%, none 35%.

At one year follow up, 60% had less severe positive symptoms and 50% showed less severe violent behaviour.

At two years follow up; 42% had been discharged (or referred and subsequently accepted for transfer to a less secure hospital), 31% continued Clozapine in Rampton Hospital and 27% had stopped Clozapine.

The rate of response to Clozapine in this sample was similar to that seen in other reported series and confirms that Clozapine represents a major advance in the management of treatment-resistant schizophrenia.

DERMATOGLYPHIC ABNORMALITIES IN SCHIZOPHRENIA

P. Fearon, A. Lane, M. Airey, J. Scannell, A. McGowan, C. Larkin, E. O'Callaghan. *Cluain Mhuire Family Centre, Newtownpark Avenue, Blackrock, Co. Dublin, Ireland*

Background: Fingerprints and palmar creases are formed during the late first/early second trimester of foetal development.

Method: We examined the finger and palm prints of 148 patients (100 M, 48 F) with DSM III-R schizophrenia and 89 (41 M, 48 F) healthy controls of the same ethnic group. Quantitative variables measured included: individual finger ridge counts, total and absolute finger ridge counts and the AB ridge count for each hand. Qualitative variables measured included: fingertip patterns (arch, loop or whorl), interdigital patterns, palmar (thenar and hypothenar) patterns, palmar creases and distal finger creases.

Results: Patients had a significantly lower mean left AB ridge count (39.0 vs 42.0, $p = 0.006$), lower mean right AB ridge count (38.8 vs 41.0, $p = 0.04$), significantly ($p = 0.01$) fewer patterns in the 4th right interdigital, fewer single distal creases on the right index finger (45% vs 55%, $p = 0.01$) and a different proportion of fingertip patterns on the same finger (patients having more arches and less whorls, $p = 0.06$) than controls.

Logistic regression revealed left AB ridge count ($p = 0.007$) and fourth right interdigital pattern reading ($p = 0.03$) to be the best predictors of patient/control status.

Conclusion: These findings support the view that intrauterine development is abnormal for some people who later develop schizophrenia.

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P300: A MARKER FOR GENETIC VULNERABILITY TO SCHIZOPHRENIA

S. Frangou, G. Alarcon, T. Sharma, T. Sigmudsson, C. Binnie, R.M. Murray. *Institute of Psychiatry, De Crespigny Park, London, SE5 8AF, UK*

It has been proposed that schizophrenia can be classified into familial and sporadic based on the presence or absence of family history of psychosis. Previous studies have shown that there is a reduction in P300 amplitude and an increase in P300 latency in schizophrenics and a subgroup of their first degree relatives. We examined 33 schizophrenic patients and 67 of their first degree relatives from families with at least two affected members as well as 29 schizophrenics and 50 of their first degree relatives without any family history of psychiatric disorder and compared them to 35 normal controls. Our objective was to investigate whether P300 amplitude and latency would contribute in differentiating between these groups. Family history was obtained from the patients, their relatives and normal controls by personal interview. Auditory P300 responses were obtained by using the standard two-tone discrimination paradigm. The following results are from P300 responses to target stimuli at the Pz site. 1. There was no difference between P300 latency or amplitude between the controls and the relatives of the non-familial cases. 2. Relatives of the familial cases showed increased latency ($p = 0.012$) and decreased amplitude ($p = 0.021$) as compared to controls. Furthermore, there was a bimodal distribution for the P300 latency in this population. 3. In familial schizophrenics, P300 latency was