Premorbid adjustment in schizophrenia: implications for psychosocial and ventricular pathology

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Abstract

Premorbid adjustment in schizophrenia is thought important (1) as a predictor of current pathology and course, and (2) as a psychosocial expression of brain pathology preceding psychosis. Its valid and reliable measurement, however, pose a major challenge. To address this issue we interviewed 12 chronic male schizophrenic veterans and their first degree relatives, plus 12 age and social class of origin matched normal controls and their relatives, using the Cannon-Spoor et al. Premorbid Adjustment Scale (PAS), for which we developed our own semi-structured interview. Objective data from school records were also obtained. Schizophrenic's PAS scores were significantly poorer, irrespective of whether PAS scores were based on information from subjects, first degree relatives or from 'combined sources'. PAS scores were worse at all developmental epochs, with a marked divergence beginning in late adolescence. Worse premorbid adjustment in schizophrenia was also highly correlated with current clinical state, more current negative symptoms, less independent living and longer duration of hospitalization. Additionally, worse premorbid adjustment in schizophrenia was associated with larger Magnetic Resonance (MR) Ventricular Brain Ratio (VBR) in an exploratory analysis using a subset of these patients. Premorbid adjustment, rigorously measured, is poorer in schizophrenics than in normal controls and correlates with psychosocial and ventricular pathology in schizophrenia.

Key words: Premorbid adjustment; Psychosocial outcome; Negative symptom; Ventricular brain ratio; (Schizophrenia)

1. Introduction

Premorbid adjustment in schizophrenia has attracted great interest as a potential predictor of clinical course and outcome (Isele et al., 1985). A review of the literature reveals numerous reports showing that premorbid adjustment in schizophrenia is worse than in normal controls (Cannon-Spoor et al., 1982; Watt et al., 1970; Watt, 1978; Lewine et al., 1978, 1980). Additionally, poor premorbid adjustment in schizophrenia has been associated with poor prognosis and worse outcome as well as with greater severity of negative symptoms, greater overall psychopathology, and worse social and occupational adjustment (Hamilton, 1976; Holmboe and Astrup, 1957; Gross et al., 1986; Strauss and Carpenter, 1974, 1977; Kokes et al., 1977; Keefe et al., 1989). Furthermore, poor premorbid adjustment in schizophrenia has been associated with poorer response to neuroleptic medication (Klein and Rosen, 1973), though con-
trary findings have also been reported (Keeffe et al., 1989; Goldstein, 1970).

Given the hypothesis that patients with schizophrenia suffer from developmental neuropathology which precedes the onset of psychosis (Weinberger, 1987; Lewis, 1989), poor premorbid adjustment could then be conceived of as a psychosocial expression of this developmental brain pathology. A number of lines of evidence support this hypothesis. These include a follow-up study of ventricular enlargement by Illowsky et al. (1988); studies of schizophrenic patients who revealed enlarged ventricles early in the course of their illness (Weinberger, 1982; Schultz et al., 1983; Andreasen et al., 1990b; DeLisi et al., 1991); obstetric/perinatal injury studies (Parnas et al., 1982; Reveley et al., 1982) and postmortem studies suggesting that the absence of typical gliosis in schizophrenic brains is more consistent with neurodevelopmental than neurodegenerative abnormalities (Waddington et al., 1991). Lastly, the association of especially poor premorbid adjustment in schizophrenics with enlarged ventricles is consistent with the idea that enlarged ventricles precede psychosis. Conflicting reports regarding this association have, nonetheless, been reported in the literature with some supporting this association (Weinberger et al., 1980; Jeste et al., 1982; DeLisi et al., 1983; Williams et al., 1985) and others not (Andreasen et al., 1982; Van Kammen et al., 1983; Nasrallah et al., 1983a, 1983b). Given these conflicting reports, we felt it was useful to try to replicate previous findings and in an exploratory way to extend them by carefully assessing premorbid adjustment in a sample for whom, in a subset, we had MRI data.

Premorbid adjustment is a difficult concept both to define (Isele et al., 1985) and measure. Self-report by chronic schizophrenics may be subject to the usual retrospective distortions of any subject plus additional distortions secondary to the patients' presumed cognitive deficits. Other potentially helpful informant sources include previous clinical records and first degree relatives such as parents and/or siblings. Clinical records, however, have the disadvantage of lack of uniformity and often do not provide information on specific, important questions. Multiple informants, including relatives, have been used by methodologically more rigorous studies of premorbid adjustment, although not in a uniform manner (Cannon-Spoor et al., 1982; Keeffe et al., 1989). We concluded that the multiple informant approach applied uniformly would yield the highest likelihood of 'validity', but we concluded further that it would also be important to compare empirically informant sources analyzed separately and also combined in an overall score. A further way in which we attempted to validate our measurement of premorbid adjustment was to obtain objective premorbid measures, i.e., school records and premorbid I.Q. scores.

2. Methods

2.1. Subjects

Patients were recruited from the Brockton VA Medical Center (BVAMC). Twelve chronic male schizophrenic veterans, inpatients and outpatients, and 12 age- and social class of origin-matched normal controls comprised our research sample. Normal controls were obtained through placing recruiting notices in local newspapers and in hospitals for staff. All patients were interviewed using the Schedule for Affective Disorders and Schizophrenia (SADS) (Endicott and Spitzer, 1978). In addition, charts were reviewed and a clinical interview was conducted (R.W.M.)blind to the other diagnostic measures. Chart reviews and the clinical interviews were used to make DSM-III-R diagnoses (APA, 1987).

The inclusion criteria for both the patient and control subjects were: (1) age 20–60; (2) male; (3) right-handed; (4) no history of ECT; (5) no history of neurological illness; (6) no history of alcohol or drug abuse (as defined by DSM-III-R criteria); (7) verbal IQ not less than 75; and, (8) an ability and desire to cooperate with the experimental procedure as evidenced by giving informed consent. The normal control sample in this study satisfied all of the above criteria and, in addition, were selected for having no history of mental illness in themselves or in their first degree relatives. On screening by a psychiatrist (J.J.L.), none of the normal controls revealed evidence of schizo-
Table 1
Descriptive data (mean ± standard deviation) for schizophrenic and normal control subjects

<table>
<thead>
<tr>
<th></th>
<th>Schizophrenics</th>
<th>Normal controls</th>
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</thead>
<tbody>
<tr>
<td><strong>n</strong></td>
<td>12</td>
<td>12</td>
</tr>
<tr>
<td><strong>Age</strong></td>
<td>44.1 ± 10.0</td>
<td>40.7 ± 8.8</td>
</tr>
<tr>
<td><strong>SES</strong>(^a)</td>
<td>4.8 ± 0.4</td>
<td>1.8 ± 1.0</td>
</tr>
<tr>
<td><strong>PSES</strong>(^a)</td>
<td>3.3 ± 0.75</td>
<td>3.6 ± 0.8</td>
</tr>
<tr>
<td><strong>Age of onset</strong></td>
<td>22.0 ± 4.7</td>
<td>—</td>
</tr>
<tr>
<td><strong>% Time in hospital</strong></td>
<td>0.54 ± 0.22</td>
<td>—</td>
</tr>
<tr>
<td><strong>Premorbid IQ</strong></td>
<td>88.2 ± 13.0</td>
<td>104.8 ± 12.1</td>
</tr>
<tr>
<td>((n = 5))</td>
<td>((n = 6))</td>
<td>(1-tailed)</td>
</tr>
<tr>
<td><strong>No. of grades completed</strong></td>
<td>11.1 ± 1.3</td>
<td>11.9 ± 0.3</td>
</tr>
<tr>
<td>((n = 11))</td>
<td>((n = 11))</td>
<td>(p = 0.056)</td>
</tr>
<tr>
<td><strong>No. of years of records obtained</strong></td>
<td>8.2 ± 3.9</td>
<td>6.5 ± 3.4</td>
</tr>
<tr>
<td>((n = 11))</td>
<td>((n = 11))</td>
<td>n.s.</td>
</tr>
<tr>
<td><strong>Records obtained: No. of grades completed</strong></td>
<td>0.73 ± 0.3</td>
<td>0.54 ± 0.3</td>
</tr>
<tr>
<td>((n = 11))</td>
<td>((n = 11))</td>
<td>n.s.</td>
</tr>
</tbody>
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\(^a\) SES: based on Hollingshead, 1965, where 1 = business/professional, 2 = medium business, 3 = skilled, 4 = semiskilled, 5 = unskilled.

phrenia spectrum conditions such as schizotypal personality disorder, or other DSM-III-R cluster A personality disorders. Handedness was assessed by a modified Oldfield inventory (Oldfield, 1971), and education and social class for subject and subject’s parents was assessed using the Hollingshead Two-Factor Index of Social and Economic Status (Hollingshead, 1965).

As shown in Table 1, the samples were well-matched for age and parental socio-economic status (PSES). SES differed significantly in the expected direction with schizophrenics lower than normal controls which we attributed to social drift. Patients were chronic, spending more than 50% of their time in the hospital.

2.2. Clinical scales

To assess premorbid functioning in the two groups we employed the Premorbid Adjustment Scale (PAS) (Cannon-Spoor et al., 1982) which assesses premorbid adjustment from a developmental perspective. We generated a standardized semi-structured interview for the PAS and systematically administered it to schizophrenics and normal controls plus their first degree relatives. As described in Table 2, the PAS assesses the premorbid level of functioning in four major domains at each of several periods during a subject’s life. The cut-off used for the premorbid period for our normal control subjects was 21 years which corresponded to the mean age of onset of our schizophrenics at the time the cut-off was selected. There

Table 2
Premorbid adjustment scale (Cannon-Spoor et al., 1982)

<table>
<thead>
<tr>
<th><strong>Purpose:</strong></th>
<th>Assess premorbid level of functioning in 4 major domains:</th>
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<tr>
<td></td>
<td>Social accessibility vs. isolation</td>
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<td></td>
<td>Peer relationships (quality, quantity)</td>
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<td></td>
<td>Ability to function outside of nuclear family</td>
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<td></td>
<td>Capacity to form intimate socio-sexual ties</td>
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<tr>
<th><strong>Advantages:</strong></th>
<th>Adaptability to variety of information sources</th>
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<tr>
<td></td>
<td>Developmental perspective with:</td>
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<tr>
<td></td>
<td>Overall score</td>
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<tr>
<td></td>
<td>Childhood score</td>
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<td></td>
<td>Early adolescence score</td>
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<td></td>
<td>Late adolescence score</td>
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<tr>
<td></td>
<td>Adulthood score</td>
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<tr>
<td></td>
<td>General score</td>
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<tr>
<th><strong>Methods:</strong></th>
<th>Semi-structured interview</th>
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<tr>
<td></td>
<td>Systematically applied to subjects and 1st</td>
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<tr>
<td></td>
<td>degree relatives (SZs and NCLs)</td>
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<tr>
<td></td>
<td>3 PAS scores per subject</td>
</tr>
<tr>
<td></td>
<td>Subject as informant</td>
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<tr>
<td></td>
<td>Relative as informant</td>
</tr>
<tr>
<td></td>
<td>‘Combined sources’ as informant</td>
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</tbody>
</table>

| **Scoring:**  | from 0 to 1; higher scores = worse adjustment |

*IEL*: Based on Hollingshead, 1965, where 1 = business/professional, 2 = medium business, 3 = skilled, 4 = semiskilled, 5 = unskilled.
were six subscales used altogether: the four life periods, the General section and then an average score for the five previous subscales which we used as our overall score. In each case, the score was derived by dividing the total score by the possible score for all rated items in a given subscale. Higher scores, potentially ranging from 0 to 1, represented poorer premorbid adjustment.

We felt it was particularly important to obtain collateral informant corroboration of patient’s premorbid histories. To empirically assess the relative validity of informant sources, we generated 3 PAS scores per subject: (1) using the subject as informant, (2) using the first degree relative as informant, and (3) using an overall ‘combined sources’ which included information from the subject, his relative, plus detailed chart synopses and school records. The final PAS score, used for correlative purposes, was based on a final best estimate score based on the two independent raters combining their two independently derived ‘combined sources’ scores which had high reliability (see below).

The PAS scale has been shown to have acceptable inter-rater reliability, particularly for trained interviewers (Cannon-Spoor et al., 1982). We determined inter-rater reliability for two raters for the Premorbid Adjustment Scale for several informant conditions: (1) the informant: schizophrenic or normal control; (2) the first degree relatives as informant; and (3) the ‘combined sources’ scores based upon all available informant sources. The inter-rater reliability for assessing schizophrenics ranged from a rho of 0.87 to 0.93 ($p<0.001$, 1-tailed). The inter-rater reliability for assessing normal controls ranged from a rho of 0.76 to 0.81 ($p<0.01$, 1-tailed). We believe the semi-structured interview that we used improved our reliability which uniformly was good.

The Scale for the Assessment of Positive Symptoms (SAPS) (Andreasen, 1984) was used to assess positive symptoms, and the Scale for the Assessment of Negative Symptoms (SANS) (Andreasen, 1981) was used to assess negative symptoms. In addition, as a measure of current clinical state, the Global Assessment Scale (GAS) (Endicott et al., 1976) was employed. Lastly, measures of current functional status (i.e., degree of independent living, and duration of hospitalization) were collected.

2.3. School records

As shown in Table 1, the mean number of grades completed by schizophrenics, for whom we obtained school records ($n=11$), was $11.1 \pm 1.3$; for normal controls, for whom we obtained school records ($n=11$), it was $11.9 \pm 0.3$, a difference which approached significance ($t(10) = -2.03, p = 0.056$, 2-tailed). There were no significant differences in the mean number of years of records obtained, or in the fraction of years of school records obtained divided by grades completed for schizophrenics or for normal controls. In addition, we obtained premorbid IQ scores, based upon standardized group-administered tests from the school records, for 5 out of 12 schizophrenics and 6 out of 12 normal controls. There was a significant difference between the two groups, with schizophrenics having a significantly lower premorbid IQ than normals ($88.2 \pm 13.0 \text{ v. } 104.8 \pm 12.1$ ($p<0.03$) respectively).

2.4. Neuroimaging measure:

MRI acquisition and image processing. Magnetic resonance (MR) scans were acquired in the axial plane using conventional long repetition rate double echo spin echo sequences on a GE Signa 1.5 Tesla MRI System (General Electric Co., Milwaukee, WI) with a standard 30 cm head coil. Multi-axial, long TR, double echo series were performed with TR = 2800 TE 30/80 ms, slice thickness 5 mm, and 2.5 mm gap (50%). In addition, image processing techniques were used, as previously described (Shenton et al., 1991), to segment the brain into different tissue classes and to derive volumetric information for the regions of interest. Here we focused on the ventricular spaces.

2.5. Statistical analyses

Group differences between mean PAS scores were tested first with a repeated measures ANOVA followed by planned comparisons with univariate
t-tests. We used a non-parametric measure for the correlations, using Spearman’s Rho rank order correlation (Siegel, 1956). Although we performed a number of correlations, those reported here were all hypothesis-driven with directions predicted. We hypothesized PAS scores would be worse in schizophrenics than in normal controls, and we hypothesized, within schizophrenics, worse PAS scores would correlate positively with larger measures of ventricular volume. Also, it was our hypothesis that worse PAS scores would correlate positively with worse psychosocial pathology.

3. Results

3.1. Premorbid adjustment in schizophrenics vs. in normal controls

Fig. 1 shows that premorbid adjustment in schizophrenics was significantly poorer than in normal controls (matched for age and social class of origin) when examined from multiple perspectives. This held true independent of informant source. That is, whether the measurement of premorbid adjustment was based upon subjects, first degree relatives or ‘combined sources’ as informants, overall PAS scores were significantly poorer for schizophrenics than for normal controls. A repeated measures ANOVA was performed with groups as the ‘between-subjects’ factor and informant sources (subject, relative, ‘combined sources’) as the repeated measure. Overall, schizophrenics were found to be statistically different than normal controls on the PAS scores ($F=18.44$, $df=1.20$, $p<0.001$). Moreover, overall PAS scores (informant sources) were found to be statistically different ($F=13.55$, $df=2.40$, $p<0.001$). A group by informant sources (subject, relative, ‘combined sources’ PAS scores) interaction was found ($F=6.81$, $df=2.40$, $p<0.003$). Planned comparisons were then performed using protected $t$-tests. As shown in Fig. 1 and Table 3, mean scores for schizophrenics versus for normal controls, with subjects, relatives, or ‘combined sources’ as informants were all significantly worse for schizophrenics.

Moreover, ANOVA of overall PAS scores, as shown in Fig. 2 and in Table 3, revealed that schizophrenic premorbid adjustment, based upon ‘combined sources’, differed significantly from

![Fig. 1. Overall premorbid adjustment scale (PAS) scores for schizophrenics and normal control subjects; planned comparisons using protected $t$-tests revealed significantly worse mean scores for SZs than NCLs whether scores based upon subjects ($t(20)=4.51$, $p<0.001$, 2-tailed), relatives ($t(22)=3.43$, $p=0.002$, 2-tailed), or ‘combined sources’ ($t(22)=5.30$, $p<0.001$, 2-tailed) as informants.](image1)

![Fig. 2. Overall premorbid adjustment scale (PAS) mean scores for schizophrenics and normal controls based upon ‘combined sources’ as informants. SZs differed significantly from NCLs using Student’s $t$-tests when viewed developmentally over the course of childhood (up to 11: $t(22)=3.60$, $p=0.002$, 2-tailed); early adolescence (12–15: $t(22)=2.97$, $p=0.007$, 2-tailed); late adolescence (16–18: $t(22)=4.86$, $p<0.001$, 2-tailed); and adulthood ($t(20)=5.36$, $p<0.001$, 2-tailed).](image2)
normal controls when viewed developmentally over the course of childhood (up to 11), early adolescence (12–15), late adolescence (16–18) and adulthood periods ($F = 18.36, df = 1.20, p < 0.001$). Also shown in Fig. 2 is an especially marked divergence between schizophrenics and normal controls over late adolescence and adulthood which is statistically confirmed by a significant group by developmental period interaction ($F = 7.80, df = 3.60, p < 0.001$).

3.2. The correlation of schizophrenic premorbid adjustment with psychosocial-clinical course measures

Results showed overall schizophrenic PAS scores based upon 'combined sources' were highly and significantly correlated with: (1) current clinical state (GAS, $n = 12$, Spearman's rho = $-0.50$, $p = 0.05$, 1-tailed); (2) specific current clinical symptoms (SANS, $n = 12$, rho = $0.585$, $p = 0.02$, 1-tailed), but not SAPS ($n = 12$, rho = $0.34$, $p = n.s.$); and, (3) current functional status (degree of independent living, $n = 12$, rho = $0.653$, $p = 0.01$, 1-tailed; duration of hospitalization, $n = 12$, rho = $0.607$, $p < 0.02$, 1-tailed).

As shown in Fig. 3, all predictions were in the expected directions with worse schizophrenic PAS scores predicting worse (lower) GAS scores, more pathological scores on the SANS and on the SAPS, a lesser degree of independent living, and a longer duration of hospitalization.

We also found that specific developmental age period scores correlated in a similar manner with clinical course variables as did the overall PAS score. Generally, these correlations yielded similar trends, but fewer significant findings, suggesting that the comprehensiveness of the overall score lent it greater predictive accuracy (data available on request).

3.3. The association between schizophrenic premorbid adjustment and objective school measures

Results showed that, as predicted, worse childhood school performance and lower premorbid IQ, based upon premorbid school records alone, was highly and statistically correlated with worse overall PAS scores ($n = 9$, rho = $0.68$, $p < 0.03$, 1-tailed; $n = 5$, rho = $-0.82$, $p < 0.05$, 1-tailed) (see Fig. 3). Again, when we looked at these correlations, taking into account specific developmental age period scores, we found similar trends overall but significant findings only for Childhood and Early Adolescent periods (data available on request). The correlations between these objective data and our data based on retrospective information collected at the time of the interview, in our view, enhanced the validity of our assessment of premorbid adjustment based on the interview data.
Fig. 3. Magnitude of Spearman's rank order correlations between overall premorbid adjustment score and psychosocial-clinical variables. Overall schizophrenic premorbid adjustment predicted (p < 0.05) (1) current clinical state (GAS) (2) specific clinical symptomatology (SANS; but not SAPS) (3) current level of functioning (degree of independent living: duration of hospitalization) (4) worse premorbid adjustment was associated (p < 0.05) with lower premorbid IQ and worse childhood school performance.

3.4. The correlation of schizophrenic premorbid adjustment and ventricular enlargement

As information from brain MR was available for only a subset of the patient sample (n = 7), we performed analyses of the relationship between premorbid data and neuroanatomical data in an exploratory manner. We caution, however, that the data are preliminary since the subject sample was small. Clinical and MRI measures on patients were completed within the same 1-year period.

We correlated schizophrenic PAS scores with schizophrenic cerebral MRI measures and found that worse overall schizophrenic premorbid adjustment scores, based upon 'combined sources', correlated positively with larger ventricular brain ratio (VBR) when controlled for age (n = 7, rho = 0.72, p < 0.04, 1-tailed).

4. Discussion

Results revealed that premorbid adjustment in schizophrenia was statistically significantly poorer than in normal controls (matched for age and social class of origin) when examined from multiple perspectives. This held true whether its measurement was based upon subjects, first degree relatives or 'combined sources' as informants. Moreover, schizophrenic premorbid adjustment, based upon 'combined sources', differed significantly from normal controls when viewed developmentally over the course of childhood (up to 11), early adolescence (12–15), late adolescence (16–18) and adulthood, with an especially marked divergence in late adolescence and adulthood. To our knowledge this marked divergence beginning in late adolescence has not been previously reported.

Given our finding that premorbid adjustment in schizophrenia was worse than in normal controls, we next sought to determine whether or not an association with subsequent clinical course and biological variables would emerge. Results, indeed, revealed that schizophrenic premorbid adjustment correlated, in expected directions, with schizophrenic psychosocial course and outcome measures. Specifically, worse premorbid adjustment was highly correlated with (1) worse current overall clinical state (GAS), (2) specific clinical symptoms, more current negative symptoms but not positive symptoms; and, (3) worse current level of functioning with less independent living and longer duration of hospitalization. Additionally, for a subset of our cases for whom we had neuroimaging data (n = 7), worse schizophrenic premorbid adjustment was associated with larger magnetic resonance (MR) ventricular brain ratio (VBR).

Prior to addressing the importance of these findings in the context of schizophrenia research, it is important to address some of the methodological issues. First, and foremost, data regarding premorbid adjustment in schizophrenia and normal controls were collected retrospectively. We attempted to deal with this in a number of ways: (1) we systematically interviewed collateral first degree relative informants for all subjects; (2) we generated a semi-structured interview to facilitate scoring of the PAS which resulted in generally excellent reliability ratings; (3) we supplemented our retrospective interview data with prior hospital records and follow-back school records (see Garmezy and Streitman (1974) for discussion of this methodology) including, in roughly half the
cases, premorbid IQ scores. This information adds greater objectivity because school grades are quantitative, and teachers were obviously ‘blind’ as to the adult mental health outcome. Premorbid IQ scores are also both non-retrospective and based upon standardized scoring systems. (4) Lastly, the psychosocial and biological measures were independently collected by different raters from the premorbid data, with raters for premorbid adjustment blind to psychosocial and biological measures and vice versa. In addition, our results showed that schizophrenic premorbid adjustment scale scores based upon ‘combined sources’ correlated significantly, in the expected direction, with ‘objective’ school measures (childhood school performance and lower premorbid IQ) which enhanced the validity, in our view, of our retrospective interview measurement of premorbid adjustment.

A second limiting factor about our data is that the ‘n’ in our MRI correlation was small (n = 7). This suggests that our finding should be regarded as tentative and requires replication with a larger sample size.

A methodologic strength of our study was our developmental perspective. For example, of interest to us, viewed developmentally, schizophrenic premorbid scores revealed a pattern of gradually worsening as subjects grew older (see Fig. 2). Conversely, normal controls’ scores remained stable with improvement in the adulthood period. This suggests that growth and development was perhaps characteristic of normal development whereas, even premorbidly, schizophrenics tended to deteriorate. A further strength of our study was our systematic use of multiple informants permitting direct comparisons. Of potential interest to clinicians, we found that, using structured interviews, the patient’s report was as good as that from their relatives.

The results of our study supported our original hypotheses that schizophrenics, when examined rigorously, would prove to have worse premorbid adjustment than matched normal controls, and that those schizophrenics with poorer premorbid adjustment would have poorer prognoses and cerebral ventricular enlargement. The fact that our findings were as strong as they were might in part reflect that we studied exclusively male subjects. As pointed out by Focerster et al. (1991), schizophrenic males, in their study, had greater premorbid impairment than schizophrenic females. They also alluded to the finding of Andreasen et al. (1990a), that non-progressive abnormalities are found predominantly in schizophrenic males. Hence, the association we found between premorbid adjustment and cerebral ventricular enlargement might well have been diminished had we included female subjects.

If our findings regarding worse schizophrenic premorbid adjustment are true, they offer further support for a neurodevelopmental view of schizophrenia (in male subjects) where an early non-progressive cerebral anatomical abnormality finds expression premorbidly with overall poorer social and academic adjustment.

In summary, we conclude that it is possible to evaluate premorbid adjustment reliably and objectively through the use of a semi-structured interview of subjects and first degree relatives. Moreover, these measures differentiate schizophrenics and normal controls and correlate with subsequent schizophrenic clinical course and cerebral pathology. In a future study, combining our current method of examining premorbid adjustment with a larger sample, with representation of females, would strengthen our argument.

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