Estimation of Premorbid Intelligence in Schizophrenia

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To determine whether the National Adult Reading Test (NART) would provide a valid estimate of premorbid intelligence in schizophrenia, two schizophrenic samples were recruited, one consisting of 35 patients resident in long-stay wards, the other of 29 patients normally resident in the community. Schizophrenic patients were individually matched for age, sex, and education with a healthy, normal subject. Both schizophrenic samples scored significantly lower on the Wechsler Adult Intelligence Scale (WAIS) than their respective control groups. NART-estimated IQ did not differ significantly between the community-resident schizophrenics and their controls, suggesting that the NART provides a valid means of estimating premorbid intelligence in such a population. NART-estimated IQ was significantly lower in the long-stay sample than in their controls. Although low NART scores in this latter sample could be a valid reflection of low premorbid IQ, the alternative explanation that NART performance was impaired by onset of the disease cannot be ruled out.

A valid means of estimating premorbid intelligence in schizophrenia would be of benefit to both clinicians and researchers. It is clear from a host of studies (see Aylward *et al*, 1984) that schizophrenia can be associated with intellectual impairment. However, because of the wide variation in intellectual abilities in the general population, detection and quantification of this impairment in individual cases requires that current performance on an IQ test be compared with an *individualised* comparison standard (Lezak, 1983).

It might be thought that this comparison standard could easily be determined if premorbid IQ test results were available. However, such information is rarely available and, when it is, usually consists of test scores obtained at primary school. As it would be inappropriate to readminster the same test in adulthood, it is necessary to compare these premorbid scores with those obtained on an adult IQ measure. Even if the two tests adopt the same scaling method (e.g. are designed to have a mean of 100 and a standard deviation of 15), such comparisons are beset with difficulties. Differences in scores between any two tests will reflect not only any potential change in an individual's abilities, but also any differences in the representativeness of the tests' standardisation samples (e.g. a test with an elite standardisation sample will be more difficult than a test without such a bias). Furthermore, this century has witnessed large and continuous gains in performance on IQ tests throughout the Western world (Flynn, 1984, 1987). As a result, tests become easier as the time between their standardisation and their administration increases. Thus, comparisons of an individual's scores on two tests can mislead if the tests differ in the obsolescence of their norms (Flynn, 1984).

Because of the difficulty of obtaining premorbid IQ scores and the problems of interpretation when such scores are available, methods of estimating premorbid IQ from readily available information have been proposed. The most common approach is to use measures of current ability which meet, as fully as possible, the three following criteria. Firstly, they should have high reliability. Secondly, they should be capable of predicting a substantial proportion of IQ variance (in the normal population). Thirdly, performance on the measures should be resistant to cerebral dysfunction.

The most promising test in this regard is the National Adult Reading Test (NART; Nelson, 1982). The NART consists of 50 short, irregular words (e.g. 'ache', 'gauche') which subjects have to read and pronounce. As the words are short, subjects do not have to analyse a complex visual stimulus and, because they are irregular, intelligent guesswork will not provide the correct pronunciation. Therefore, it has been argued that performance depends more on previous knowledge than on current cognitive capacity (Nelson & O'Connell, 1978).

Studies of the NART's split-half reliability (Nelson, 1982; Crawford *et al*, 1988), test–retest reliability (Crawford *et al*, 1989*a*), and inter-rater reliability (O'Carroll, 1987; Crawford *et al*, 1989*a*) indicate that the NART is reliable.

A number of studies have investigated the validity of the NART as a measure of intelligence. In the NART standardisation sample (Nelson, 1982), 120 normal subjects were administered the NART and selected subtests of the Wechsler Adult Intelligence Scale (WAIS; Wechsler, 1955). Performance on the NART predicted 55% of the variance in WAIS IQ. In a recent cross-validation study, using a full-length

WAIS, the NART predicted 66% of IQ variance (Crawford *et al*, 1989*a*). The most widely adopted method of evaluating a test's construct validity as a measure of intelligence is to factor analyse it along with existing measures of intelligence and examine its loading on the first unrotated factor or principal component (commonly termed '*g*', or general intelligence). A combined factor analysis of the NART and WAIS (Crawford *et al*, 1989*b*) revealed that the NART had a high loading on '*g*' (0.85); indeed, this loading was higher than all but 2 of the 11 WAIS subtests.

Thus the NART meets the first two criteria for a measure of premorbid intelligence. Therefore, in order to determine whether the NART will provide a valid means of estimating *premorbid* intelligence in schizophrenia, it only remains to examine whether NART performance is impaired by schizophrenic illness. Examinations of NART performance in other clinical conditions have been encouraging. Crawford *et al* (1987) reported that the NART performance of a sample of depressed patients did not differ significantly from that of matched controls, despite impairment on the WAIS Vocabulary subtest (a test commonly used as an alternative means of estimating premorbid intelligence). Studies of NART performance in organic disorders suggest decline in some conditions but, in general, indicate that the NART is a surprisingly robust test (see Crawford, 1989, for a review). For example, both Nebes *et al* (1984) and Crawford *et al* (1988) have reported that the NART performance of patients with dementia of the Alzheimer type (DAT) did not differ significantly from that of matched controls. Hart *et al* (1986) found some evidence for impairment of NART performance in DAT but reported that this was minimal in comparison with the impairment on other psychometric tests.

The purpose of the present study was to determine whether the NART would provide a valid estimate of premorbid intelligence in schizophrenia. Because of the large differences in the length of hospital stay within schizophrenic populations, it was decided to recruit two samples: one consisting of schizophrenic patients resident in long-stay wards and the other of patients from other areas of the psychiatric services, that is, acute admission wards, day hospitals, hostels, and out-patient clinics. The intention was to compare the performance of these samples on IQ measures and the NART, with that of matched healthy, control samples.

Method

Subjects meeting DSM-III criteria (American Psychiatric Association, 1980) for schizophrenia were recruited from the Grampian Region Psychiatry Service. Potential subjects had to pass a simple test of visual acuity (reading standard type-written letters of the alphabet). A sample of 35 patients (15 men, 20 women) resident in long-stay wards was recruited (the LH sample). A sample of 29 patients (22 men, 7 women) normally resident in the community was also recruited (the CR sample). As the intention was to recruit representative samples, patients who had suffered a cerebral insult following onset of illness were not excluded. As a result, the LH sample included 13 patients who had undergone a frontal leucotomy or insulin coma therapy.

Most patients (90.6%) were receiving some form of neuroleptic medication at time of testing. The majority (58%) were also on anticholinergic medication. Most of these were receiving procyclidine. However, four patients were receiving orphenadrine. In order to summarise anticholinergic medication a conversion factor of 10 was assumed for orphenadrine to procyclidine.

Schizophrenic subjects were individually matched for age (± 3 years), sex, and years of education (± 2 years) with a healthy control subject. Control subjects were screened for the absence of neurological and psychiatric disorder, and were recruited from a variety of sources (community centres, pensioners' clubs, unemployment clubs, etc.).

The social class of each schizophrenic and control subject was derived from the Office of Population Censuses and Surveys (1980) *Classification of Occupations*. Subjects who were currently unemployed or of retirement age were coded by their previous occupation. Subjects who had never worked were coded as social class 5.

Schizophrenic patients and control subjects were administered the NART and a full-length WAIS. The WAIS was used as the IQ measure rather than the WAIS-R (Wechsler, 1981; Lea, 1986) as equations to estimate premorbid WAIS-R performance from the NART have yet to be developed. All tests were administered and scored according to the standardised procedures set out in the relevant test manuals (Wechsler, 1955; Saville, 1977; Nelson, 1982). NART errors were converted to estimated premorbid IQ by the following regression equation, provided by Crawford *et al* (1989*a*, p. 273):

$$\text{estimated premorbid IQ} = 131.36 - (0.95 \times \text{NART errors})$$

This equation was derived from a sample ($n = 151$) free of neurological, psychiatric, or sensory disability. It was used in preference to Nelson's (1982) original equation because: (a) it has a smaller standard error of estimate, (b) it permits estimation over a wider range of IQ and age (up to 88 years), and (c), most importantly, Nelson's equation was built to estimate a short-form IQ rather than an IQ obtained from a full-length WAIS.

All statistical analyses were carried out using repeated measures or paired-samples *t*-tests. When conducting within-group comparisons, two-tailed tests were used. As the hypotheses to be examined through between-groups comparisons were directional (i.e. the hypotheses of superior performance by controls were set against the competing hypotheses of no difference), one-tailed tests were used in these.

Table 1
Background characteristics of two schizophrenic samples
(means (s.d.))

	Long-stay hospital sample	Community-resident sample
Age: years	57.1 (10.6)	34.9 (12.4)
Years of education	9.6 (1.0)	11.3 (2.0)
Age of onset: years	27.8 (10.6)	25.1 (8.9)
Total in-patient stay: years	19.3 (12.9)	2.3 (4.6)
Daily chlorpromazine units (or equivalent) at time of testing	628.7 (809.7)	344.4 (436.5)
Daily procyclidine units (or equivalent) at time of testing	10.7 (11.7)	7.7 (7.4)

Results

Summary statistics for age, years of education, age at onset of schizophrenic illness, in-patient stay (total time spent in hospital since onset of illness, excluding current admission), and medication are presented in Table 1 for both samples.

Mean (s.d.) age in the control group for the LH sample was 57.0 (10.9) and mean years of education was 9.9 (0.9). Mean age in the CR control group was 33.9 (13.4) and mean years of education 11.5 (2.1). Paired-samples t-tests revealed that the two schizophrenic samples did not differ significantly from their respective control samples in terms of age or years of education (>0.1 in all comparisons). None of the schizophrenic or control subjects were coded as social class 1. The numbers in each of the remaining categories of social class were as follows (the figures for the schizophrenics are followed by the figures for controls): social class $2 = 14/16$; $3 = 23/28$; $4 = 18/12$; $5 = 9/8$. The distribution of social class in schizophrenics and controls did not differ significantly ($x^2 = 1.88$, d.f. $= 3$).

Mean WAIS IQ and NART-estimated full-scale IQ in the two schizophrenic samples and in their respective control samples are presented in Table 2. The mean IQs in the control samples could be viewed as indicating that they were of above average intellectual ability. However, the WAIS yields IQs in the contemporary UK population that are inflated by over half a standard deviation; that is, the best available estimate of the UK population mean on the WAIS is 108 (Crawford *et al*, 1990a). Therefore, the control samples were of essentially average intellectual ability.

Paired-samples t-tests (one-tailed) revealed that the CR schizophrenic sample obtained significantly lower scores

than their control group on WAIS full-scale IQ ($t = 3.7$, $P < 0.01$), verbal IQ ($t = 2.35$, $P < 0.05$), and performance IQ ($t = 4.76$, $P < 0.001$). The LH sample also obtained significantly lower scores than their control sample on full-scale IQ ($t = 11.25$, $P < 0.001$), verbal IQ ($t = 8.82$, $P < 0.001$), and performance IQ ($t = 12.56$, $P < 0.001$).

The NART-estimated IQ did not differ significantly between the CR sample and control sample ($t = 0.47$, one-tailed). However, NART-estimated IQ was significantly lower in the LH sample than in their controls ($t = 8.05$, one-tailed, $P < 0.001$).

Repeated-measures t-tests (two-tailed) revealed that, as expected, WAIS full-scale IQ did not differ significantly from NART-estimated full-scale IQ in either control sample. WAIS full-scale IQ was significantly lower than NART-estimated IQ in both the CR schizophrenic sample ($t = 8.83$, $P < 0.001$) and the LH schizophrenic sample ($t = 12.09$, $P < 0.001$). Verbal IQ in the schizophrenic samples was substantially higher than performance IQ. In view of this it was decided to compare verbal IQ directly with the NART score. To permit this within-group comparison, NART errors were converted to estimated verbal IQ using the relevant regression equation (Crawford *et al*, 1989a, p. 273). Mean (s.d.) NART-estimated verbal IQ in the CR schizophrenic sample was 103.9 (9.9), compared with an obtained verbal IQ of 96.9 (15.7) ($t = 3.69$, $P < 0.01$). In the LH sample, NART-estimated verbal IQ was 97.1 (8.2), compared with an actual verbal IQ score of 84.4 (15.9) ($t = 7.01$, $P < 0.001$).

The lowest full-scale IQ that can be predicted from the NART is 84; this would be the estimated premorbid IQ for a subject getting none of the NART items correct. The mean obtained full-scale IQ in the LH sample was 80.3 (Table 2). Therefore, if all LH schizophrenics had failed every NART item, the estimated premorbid IQ would still have been higher than obtained IQ. Although none of the LH schizophrenics did perform this poorly on the NART, it can be seen that this feature exaggerates the difference between obtained and estimated IQ. To investigate the effect of this on the significance of the difference between these two measures, the IQs of schizophrenics were recorded to an IQ of 84 if they scored below this level. A t-test carried out on the recoded data revealed that the difference between obtained IQ and estimated premorbid IQ remained highly significant ($t = 11.5$, $P < 0.001$).

Within the LH sample, independent-samples t-tests (one-tailed) were used to compare NART-estimated IQ in the 13 patients who had suffered a cerebral insult with the 22 who had not. Mean NART-estimated IQ in the former

Table 2
Mean (s.d.) scores for the WAIS scales and NART-estimated full-scale IQ in two schizophrenic samples and matched controls

	WAIS: full scale	WAIS: verbal	WAIS: performance	NART-estimated full-scale IQ
Community-resident schizophrenics	92.2 (14.9)	96.9 (15.7)	87.0 (14.9)	105.6 (9.8)
Controls	105.3 (13.6)	104.7 (14.0)	105.7 (12.9)	106.7 (10.1)
Long-stay schizophrenics	80.3 (13.7)	84.4 (15.9)	76.9 (11.4)	98.0 (8.5)
Controls	111.7 (10.6)	112.22 (12.0)	109.9 (10.5)	111.2 (8.1)

group was 97.6 (10.7), which did not differ significantly ($t = 0.03$) from that in the remaining LH subjects (mean 97.7, s.d. 6.9).

Discussion

In the present study, both schizophrenic samples obtained significantly lower scores on the WAIS scales than their respective control groups. Considered in isolation, these results are open to three possible explanations. Firstly, the poorer performance may be entirely due to lower premorbid IQ; in other words the onset of schizophrenia did not impair a below-average intellect. Alternatively, it may be entirely a result of intellectual impairment caused by the onset of schizophrenia. Thirdly, the deficit in current performance may be due to a combination of these factors, that is, the cumulative (or additive) result of low premorbid IQ and acquired impairment.

In both schizophrenic samples, full-scale IQ as judged by the WAIS was significantly lower than the NART-estimated full-scale IQ. Given the substantial proportion of shared variance between these two measures in the general population (see Introduction), this indicates a decline in intellectual functioning from a previously higher level. The alternative interpretation, that development of schizophrenia leads to a substantial and isolated improvement in the ability to read and pronounce irregular words, is scarcely credible.

In studies where actual rather than estimated premorbid IQ scores were available, the performance of schizophrenic patients was examined on intelligence tests originally administered in the premorbid period, either during secondary school (Rappaport & Webb, 1950), or on induction into the army (Lubin *et al*, 1962; Schwartzman & Douglas, 1962*a,b*). Postmorbid IQs were significantly lower than premorbid IQs in all three investigations. To the present authors' knowledge, the only study which failed to find a decline between the premorbid and postmorbid periods was that of Albee *et al* (1963). The design of this study differed from those above in that the test administered in the postmorbid period (the Wechsler–Bellevue) was not the same as that administered in the premorbid period (the Stanford–Binet). There are considerable problems of interpretation in such circumstances.

In the present study, having concluded that some decline in functioning has taken place, the remaining issue is whether poor current performance can be attributed *solely* to impairment or whether it also reflects low premorbid IQ. NART performance in the CR schizophrenic sample was essentially equivalent to that of the matched control sample, indicating that

premorbid IQ was not below average. The same conclusion cannot be drawn for the LH sample. NART performance in the LT schizophrenics was significantly poorer than that of their control sample. This could be viewed as an indication that the premorbid IQs of the LH sample were below average. This would be consistent with studies in which the premorbid IQ scores of people with schizophrenia were compared with those of their peers. Most of these studies indicate that a proportion of schizophrenic subjects have abnormally low premorbid IQs (see Aylward *et al*, 1984, for a review). However, an alternative interpretation must be considered. Although the abilities underlying NART performance appear to be surprisingly resistant to various types of disorder (Crawford, 1989), it may be that the LH schizophrenic sample was impaired on this test. If this were the case, then the NART would systematically underestimate the true premorbid intelligence of the sample.

The NART performance of patients who had suffered a cerebral insult (a leucotomy or insulin coma therapy) did not differ significantly from that of patients without such a history. This result largely rules out one of the more likely potential causes of impairment of performance on the NART. However, other factors, common to all patients in the LH sample, could produce such an impairment – for example long-term institutionalisation and, of course, the long history of severe schizophrenic illness itself.

The principal question addressed by the present study was a practical one: does the NART provide a valid estimate of premorbid intelligence in schizophrenic populations? In drawing conclusions on this question it is worth commenting on the comparison between the schizophrenic samples' NART and verbal IQ performance. As noted above, the present results, in common with most other studies of IQ (Aylward *et al*, 1984), indicate that the highly significant difference between schizophrenics and controls on full-scale IQ is largely the result of impairment on the performance scale. The verbal scale of the WAIS, like the NART, draws largely on well consolidated verbal abilities and is relatively spared. This raises the question of whether it is necessary to employ the NART as an estimate of premorbid ability. However, although verbal exceeded performance IQ in both schizophrenic samples, it was nevertheless significantly lower than verbal IQ in the matched control samples. Furthermore, individuals scored significantly higher on the NART than for verbal IQ. This demonstrates that the NART is more resistant to schizophrenic dysfunction than verbal IQ and will, therefore, provide a more valid measure of premorbid intelligence.

Although the NART proved more resistant than verbal IQ in the LH sample, the NART performance of these patients was still significantly poorer than that of their matched controls. As noted, it cannot be determined whether the low NART scores were due to impaired performance or were simply a reflection of low premorbid intelligence. Therefore at present it would be inadvisable to use the NART as a measure of premorbid IQ with such patients.

In contrast, the results and conclusions for the CR sample were unequivocal. As the mean NART scores of the sample were equivalent to those of their matched controls, there was therefore no evidence of impaired NART performance. This suggests that the NART can validly be used as a means of estimating premorbid intelligence in such a population.

The NART can therefore provide clinicians with a simple and rapid means of assessing a patient's premorbid intellectual resources. It could also be used in retrospective studies aimed at delineating the relationship between premorbid variables and prognosis, treatment outcome, or symptom patterns. Thirdly, the NART provides a comparison standard for a patient's current performance on intelligence tests. Crawford *et al* (1990*b*) used hierarchical discriminant function analysis to examine how successfully a measure of *current* intelligence (the WAIS) could discriminate between healthy subjects and samples of patients with either evidence (from computerised tomography) of cortical atrophy or a diagnosis of dementia. The discriminative ability of the WAIS alone was then compared with that achieved by combining the WAIS and the NART. Inclusion of the NART in the analysis, which essentially served to partial out the effects of premorbid intelligence, led to 96% of the healthy and demented subjects being correctly classified. This discrimination was significantly better than that achieved by the WAIS alone.

Finally, the NART has a contribution to make to general research methodology. A common design in psychiatric/psychological research on schizophrenia involves comparing a schizophrenic sample with either healthy subjects or other clinical groups. As group membership is the independent variable in this design, every effort should be made to match the groups on potential confounding variables. In normal practice this would involve matching the groups for age, sex, and education. Such matching could be made more stringent by using the NART to match the groups for premorbid intelligence. This would be especially important in investigating the effects of schizophrenic illness on cognitive functions.

The NART could also be used in many correlational designs, as it would permit the researcher to control for the effects of premorbid intelligence when examining relationships between other variables. For example, with the increased availability of brain-imaging techniques, there are opportunities to investigate the relationship between cognitive performance in schizophrenia and indices of abnormal cerebral metabolism, blood flow, and morphology. In an impaired sample, the variance of any cognitive measure will reflect not only the effects of acquired cerebral dysfunction but also preexisting differences in general intellectual ability. The NART could be used to partial out this latter source of variance, which would otherwise serve to obscure relationships between brain and behaviour. The NART has already been successfully employed for this purpose in a study of the relationship between cognitive function and cerebral morphology in alcoholics (Acker *et al*, 1987).

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