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The National Adult Reading Test as a measure of premorbid intelligence: A comparison with estimates derived from demographic variables

PETER BRIGHT,[1,2] ELI JALDOW,[1] AND MICHAEL D. KOPELMAN[1]

[1]University Department of Psychiatry and Psychology, King's College, London, UK
[2]Now at Department of Experimental Psychology, University of Cambridge, Cambridge, UK

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Abstract

Since its publication in 1982, the National Adult Reading Test (NART; Revised Version, NART–R) has become a widely accepted method for estimating premorbid levels of intelligence in neuropsychological research. However, the assumption that NART/NART–R performance is relatively independent of brain damage has been increasingly challenged in recent years. In a number of conditions, including Alzheimer dementia and Korsakoff's syndrome, studies have indicated a deterioration in reading ability, leading to an underestimated premorbid IQ. In a reaction to these studies, some researchers have advocated the use of demographic variables as a more suitable foundation for accurately predicting premorbid intelligence. We addressed this issue by calculating IQ estimates on the basis of NART/NART–R, demographic variables, and a combination of the two approaches and by comparing these with current WAIS/WAIS–R IQ in patients with Korsakoff's syndrome, Alzheimer dementia, frontal or temporal lobe lesions, and in healthy controls. Estimated premorbid IQs did not differ across groups, whether derived from NART/NART–R or demographic variables. Those based on NART/NART–R demonstrated higher correlations with current WAIS/WAIS–R IQ in controls and patients than those derived from demographic variables. An equation combining NART scores with demographic variables did not significantly increase the amount of variance in IQ explained by NART only, either in patients or controls. The data offer reassurance regarding the continued use of NART as a valid estimate of premorbid intelligence in a number of conditions. (*JINS*, 2002, *8*, 847–854.)

Keywords: NART, Demographic variables, Intelligence

INTRODUCTION

Measurement of cognitive deficits in brain-damaged patients usually requires an indirect prediction of premorbid ability. Given that intelligence is correlated with practically all cognitive measures (O'Carroll, 1995), and is therefore likely to deteriorate following brain damage, meaningful comparison of patient and controls requires matching on this function. The National Adult Reading Test (NART; Nelson, 1982; NART–R; Nelson & Willison, 1991) is widely used to estimate a person's premorbid level of intellectual ability. The test requires subjects to read out loud a set of 50 words which are irregular in terms of their grapheme–phoneme correspondences (Coltheart et al., 1987). The responses are individually scored as correct or incorrect, according to their pronunciation. This score can then be used to derive a premorbid IQ estimate.

The validity of NART as a measure of premorbid ability rests upon the assumptions that reading ability (of irregular words) is relatively independent of brain damage, and that it is a strong predictor of intelligence in the normal population. A study by Nelson and McKenna (1975) indicated that performance on the Schonell Graded Word Reading Test (Schonell, 1942) is largely preserved in dementia, while a study by Nelson and O'Connell (1978) showed NART to be a robust predictor of premorbid levels on the Wechsler Adult Intelligence Scale (WAIS, Wechsler, 1955) in 40 patients with evidence of bilateral cortical atrophy. In both studies, patients had lower current IQs than healthy controls, but their reading test scores were closely matched. In

Reprint requests to: Peter Bright, Department of Experimental Psychology, University of Cambridge, Downing Street, Cambridge, CB2 3EB, UK. E-mail: pbright@csl.psychol.cam.ac.uk

Nelson's (1982) original standardization study on patients with extra-cerebral disorders ($N = 120$), NART predicted 55% of the WAIS Full Scale IQ (FSIQ), and, in a subsequent study based on a larger sample ($N = 151$) and wider age range, the NART predicted 66% of the variance in FSIQ (Crawford et al., 1989a). A recent retrospective validity study by Crawford et al. (2001) followed up 177 individuals that had been administered an IQ test at age 11. A comparison of these scores with NART scores at age 77 lent strong support to the claim that NART estimates premorbid, rather than current, intelligence.

Despite such evidence, there have been a number of reports that, in dementia, NART performance remains preserved only in the early stages of the disease. In a longitudinal study of Alzheimer patients, Fromm et al. (1991) found decrements in NART performance across the 3 years sampled, suggesting that a sensitivity to dementia may only occur at later stages of the disease. A more recent longitudinal study by Cockburn et al. (2000) also found evidence of a deterioration in NART scores across four annual assessments in 78 patients with autopsy confirmed or a clinical diagnosis of Alzheimer Dementia. Other reports of a deterioration in NART performance at late stages of dementia have been reported by Hart et al. (1986), Stebbins et al. (1990) and Taylor (1999). Patterson et al. (1994) investigated NART performance in a group of 45 patients with a diagnosis of probable DAT. The patients were placed into subgroups of minimal, mild and moderate dementia, based on their Mini-Mental State Examination (MMSE; Folstein et al., 1975). A significant correlation ($r = .56$) was observed between NART score and severity of dementia across the total patient group. Consequently, the authors questioned the validity of NART as a premorbid measure of functioning in dementia.

The reliance on NART for predicting premorbid ability in the alcoholic Korsakoff's syndrome (AKS) was first questioned by Crawford et al. (1988). They found NART scores were significantly lower in a group of Korsakoff patients than in controls, matched for sex, age, and education. However, they also noted that the differences were relatively small and that the NART appeared more resistant to decline than a comparison measure (the Vocabulary subtest of the WAIS).

More recently, O'Carroll et al. (1992) compared scores on NART among 20 AKS patients and 40 healthy controls. Their conclusion that pronunciation of irregular words is significantly affected by AKS was supported by a number of observations: (1) the patients produced more NART errors than controls; (2) the patients had lower NART predicted IQ than demographically predicted IQ; (3) NART performance in patients was associated with severity of memory impairment. However, a direct comparison of current IQ scores across the patients and control groups was not presented: if WAIS performance was relatively poor in the patient group, lower NART predicted IQ scores would be expected. The authors suggested that the relatively poor performance in AKS might relate to the frontal lobe dysfunction commonly implicated in this condition, whereby there is a failure to monitor and check initial response tendencies prior to vocal output. They concluded that equations used to predict WAIS IQ that are based entirely on demographic variables (e.g., Barona et al., 1984; Crawford et al., 1989b) might be preferable in AKS, in order to prevent the underestimation of premorbid ability. The use of NART as a measure of premorbid intelligence in other conditions, such as Huntington's disease, schizophrenia, depression, and primary brain tumor irradiation has also been questioned (for reviews, see Crawford, 1992; Franzen et al., 1997; O'Carroll, 1995).

An advantage of using demographic variables in the prediction of premorbid intelligence is that, unlike the NART, they are entirely independent of current cognitive status. Crawford et al. (1989b) developed a method of predicting premorbid IQ in UK populations based on equations that predicted 54% of the variance of WAIS Full Scale IQ (FSIQ), 53% of Verbal IQ (VIQ) and 42% of Performance IQ (PIQ) in a USA sample (Wilson et al., 1978). Information regarding age, sex, occupation and education from 151 normal, healthy subjects was used to predict their WAIS performance. Regression of WAIS scores on these demographic variables in a UK sample generated equations that explained 50%, 50%, and 30% of the variance in FSIQ, VIQ and PIQ, respectively (Crawford et al., 1989b).

Crawford et al. (1990) proposed that a better method of predicting premorbid intelligence than that afforded by either the NART or demographic variables would be a combination of the two approaches. While the NART remained the better predictor in their study, a multiple regression equation that incorporated both NART and demographic variables explained significantly more of the variance in FSIQ than NART only. Subsequent studies have failed to reach agreement as to whether this combined method adds significantly to the power of either the NART or demographic variables alone. O'Carroll (1995) concluded that the combined NART and demographic variables regression equation was the best predictor of premorbid ability in the United Kingdom and Australia, but that the amount of explained variance in North Americans was not significantly increased by the inclusion of demographic data.

The evidence to date raises serious questions concerning the use of NART as a predictor of premorbid level of WAIS IQ. Additionally, NART–R provides a quite different predicted IQ than does NART, particularly for higher error scores (e.g., a difference of 12 IQ points is observed for an error score of 35). The clinical significance of this issue has not been fully explored. Despite these potential criticisms, NART and NART–R remain popular with both clinicians and researchers. Administration is straightforward, takes little time, and performance is characteristically preserved relative to other measures of cognitive function (e.g., Crawford et al., 1988; Isaac & Mayes, 1999; Kopelman et al., 1999; Parkin et al., 1999).

In the present study we have systematically addressed the issue of whether NART, demographic variables, or a

combination of the two approaches represents the best method for predicting WAIS and WAIS–R. In order to do this, we re-examined data from 98 patients and 51 healthy controls gathered in previous investigations (Kopelman, 1985; 1989; Kopelman et al., 1999). The patients included Alzheimer, Korsakoff, and focal lesion patients with severe or moderately severe memory disorder. All the subjects had originally been assessed in terms of NART or NART–R and WAIS or WAIS–R, but both NART and NART–R IQs could be determined from a given reading error rate, and formulae derived from the *WAIS–R Manual* were used to convert WAIS IQs to WAIS–R IQs and *vice versa*. In addition, age, educational, and occupational status (social class) data had been collected, from which we could derive demographic predictions of IQ according to the formulae of Crawford et al. (1989b). These data were employed (1) to investigate whether there were significant differences across the patient groups, relative to healthy controls, in terms of predicted IQ, current IQ, and predicted minus current IQ scores derived from these measures; and (2) to examine the size and statistical significance of correlations between the various measures of both predicted and current IQ.

METHODS

Research Participants

The patient groups investigated and relevant references are summarized in Table 1, together with the measures used to provide current IQ and estimates of premorbid IQ in the original publications. In addition to the patients, there were 51 controls from the same studies. The controls were originally recruited to match the relevant patient groups on age, education, occupational level and sex. As a consequence of these matching criteria, comparable NART scores were observed across patients and their respective controls. Only a brief description of the patient groups is given here, as they have been reported in detail in the earlier publications. Quantitative MRI findings in the case of the 1999 study are given in two publications (Colchester et al., 2001; Kopelman et al., 2001).

Temporal lobe lesion patients

Fourteen patients with temporal lobe amnesia resulting from probable or definite (antibody confirmed) herpes encephalitis (9), hypoxia (4), or epilepsy associated with medial temporal lobe atrophy (1) were included. In all patients there was evidence of temporal lobe involvement on MRI.

Frontal lesion patients

Nine patients were assessed following bilateral frontal tractotomy for treatment of chronic affective disorders. Testing took place in the second or occasionally the 3rd postoperative week, following the observation that such patients behave most like patients with large frontal lesions at this time (Kartsounis et al., 1991). A further 6 patients with focal frontal lesions were also assessed. Three of these patients had right frontal lesions, 1 had a left frontal lesion, and 2 had bilateral lesions.

Korsakoff patients

Thirty-five patients were selected. They conformed as closely as possible to the clinical features of the acute onset subgroup identified by Cutting (1978) in a retrospective study of patients from the Maudsley Hospital, London. All had a history of disorientation and confusion at admission, and the numbers of other Wernicke features present at diagnosis are described in the original papers. All had a history of heavy and prolonged alcohol abuse (range = 10–48 years) and were severely incapacitated by their memory disorder, either heavily dependent upon a relative or living in institutions. In addition, each patient had had either a CT scan or clinical MRI scan to exclude other pathology and 12 out of the 35 Korsakoff patients had had an FDG PET scan as well.

Alzheimer patients

The data from 32 patients with Alzheimer-type dementia were included in this study. All these patients were diagnosed according to clinical history, psychometric evidence of generalized impairment and CT scan evidence of cortical

Table 1. Age, education and source references for the patient groups

		Age		Education		
Group	*N*	*M*	*SD*	*M*	*SD*	Reference**
Temporal lobe patients	14	45.14	16.28	12.29	2.81	3
Frontal lobe patients	15	45.73	10.17	11.40	1.68	3
Korsakoff patients	35	55.43	8.46	10.80	2.18	1,2,3
Alzheimer patients	32	67.16	6.37	10.31	1.69	1,2
Total patients*	98	56.15	12.84	11.02	2.19	1,2,3
Controls	51	55.39	16.01	11.20	2.59	1,2,3

Note. Age and education are in years.
*Includes 2 pituitary adenoma patients (treated with surgery and irradiation).
**Source references: 1 Kopelman (1985); 2 Kopelman (1989); 3 Kopelman et al. (1999).

atrophy. They were not hypertensive, nor had a history or any evidence of cerebrovascular disease. Mean duration of symptoms was 2.9 years (range 6 months to 6 years).

Procedure

In the original studies, estimates of premorbid intelligence were based either on NART or NART–R. The present study required that each subject receive a NART *and* a NART–R predicted IQ in order for the relative accuracy of these measures to be examined against the appropriate measure of current IQ (WAIS or WAIS–R). This simply entailed matching the original error score against the relevant conversion table, provided by Nelson (1982) and Nelson and Willison (1991).

To calculate premorbid levels of intelligence based on demographic variables we used the formulae provided by Crawford et al. (1989b) and Crawford and Allan (1997):

- Predicted WAIS FSIQ = 104.12 − 4.38 (social class) + 0.23 (age) + 1.36 (education) − 4.7 (sex)
- Predicted WAIS–R FSIQ = 87.14 − 5.21 (social class) + 0.18 (age) + 1.78 (education)

Social class was determined by occupation using the Office of Population, Censuses and Surveys (1980) Classification of Occupations (range = 1–5). Age was recorded in years. Sex was recorded as 1 for males and 2 for females. Years of education were derived from records of peak educational attainment or school leaving age.

We are grateful to John Crawford (University of Aberdeen) for providing us with formal equations for comparing WAIS and WAIS–R, based on data included in the test manuals (Wechsler, 1987, Table 17, p. 47). With these equations (reproduced below) we were able to calculate a WAIS and a WAIS–R FSIQ for each subject:

- Predicted WAIS FSIQ = (.757 × WAIS–R FSIQ) + 32.77
- Predicted WAIS–R FSIQ = (1.024 × WAIS FSIQ) − 10.12

We calculated separate estimates of WAIS based on NART and demographic variables (referred to as DEM, for *social demographics*) and of WAIS–R, based on NART–R and the demographic variables (referred to as *DEM–R*).

RESULTS

Predicted IQ scores based on the original (NART, DEM) and revised (NART–R, DEM–R) measures are presented in Table 2, together with current IQ (WAIS, WAIS–R), for controls and individual patient groups. Statistical analyses demonstrated that there were no significant differences between patient groups and controls on any of the premorbid measures. On WAIS and WAIS–R there were highly significant differences. *Post-hoc* analyses (Dunnett's test) indicated that, on both measures, only the Alzheimer patients performed more poorly than controls.

Differences between premorbid estimates and WAIS/WAIS–R FSIQ scores were calculated and compared across the controls and patient groups. The results are shown in Table 3. An analysis of variance was performed on these difference scores across temporal amnesic, frontal amnesic, Korsakoff, Alzheimer, and control groups. As shown in Table 3, there were significant differences across groups on all comparisons.

Post-hoc comparisons (Dunnett's test) indicated that, consistent with their having dementia, the Alzheimer patients showed a wide and significant departure from controls on all comparisons, with current intellectual ability well below that of predicted premorbid level, consistent with the diagnosis of dementia. Premorbid estimates based on demographic variables were more different from current IQ than were those based on NART or NART–R [$t(29) = -4.90$, $p < .001$; $t(29) = -2.09$, $p < .05$], for original and revised measures, respectively. Within the frontal and temporal groups, repeated measures *t* tests found the differences between predicted and actual IQ to be equivalent, whether the predicted measure was based on NART scores or on demographic variables. In the Korsakoff patients, NART and WAIS performance was less discrepant than DEM and WAIS performance [$t(33) = -3.70$, $p < .01$], but NART–R minus WAIS–R did not differ significantly from DEM–R minus WAIS–R. Overall, the findings indicated that only in the

Table 2. Comparison of IQ estimates across groups, with *post-hoc* comparison of patient groups against controls

Measure	Controls	Temporal	Frontal	Korsakoff	Alzheimer	*F*	*p*
NART	108.84	108.77	110.67	107.21	106.03	.76	NS
NART–R	102.49	104.00	105.13	100.03	98.30	.85	NS
DEM	112.31	112.50	108.47	113.03	112.56	1.50	NS
DEM–R	102.13	101.21	99.60	101.09	102.13	.39	NS
WAIS	109.41	105.29	105.00	105.40	80.03***	29.58	<.001
WAIS–R	101.76	95.86	95.47	97.17	71.91***	23.30	<.001

Note. DEM and DEM–R are measures of premorbid IQ based on demographic variables. NS = not significant at $p = .05$. *Post-hoc* comparison of patients and controls indicated by asterisks were significant at or below $p = .05$.
***$p < .001$ (Dunnett's test).

Table 3. Analysis of discrepancies between premorbid estimates of WAIS IQs and obtained WAIS IQs among groups, with *post-hoc* comparison of patient groups against controls

Measure	Controls	Temporal	Frontal	Korsakoff	Alzheimer	$F(4,134)$	p
NART *vs.* WAIS	−.92	5.01	5.65	1.97	24.6***	32.59	<.001
NART–R *vs.* WAIS–R	.37	8.42	9.67	3.06	24.97***	19.44	<.001
DEM *vs.* WAIS	2.67	7.14	3.49	7.75	31.73***	33.24	<.001
DEM–R *vs.* WAIS–R	−.05	5.58	4.13	4.12	29.47***	25.41	<.001

Note. *Post-hoc* comparison of patients and controls indicated by asterisks were significant at or below $p = .05$.
***$p < .001$ (Dunnett's test).

Alzheimer group was present level of intellectual ability consistently lower than estimated levels, relative to other patients and controls.

Table 4 provides correlations between the measures used to predict WAIS performance (NART, NART–R, DEM, and DEM–R). The correlations between NART and NART–R were all unity as the two sets of scores are linear transformations of each other. The correlations between the demographic estimates of IQ were also reassuringly high (these estimates were obtained from different standardization samples and used different predictor variables). The correlations of NART and DEM, and NART–R and DEM–R, were also significant in patients and controls (apart from within the group of 14 temporal patients), although the degree of shared variance between these measures (<25% in both the total patient and control groups) was relatively low. The original (NART, DEM) and revised (NART–R, DEM–R) versions of both measures produced intercorrelations of similar magnitude.

Table 5 lists the correlations of the predictors with WAIS and WAIS–R. Neither the reading test (NART, NART–R) nor demographic variable (DEM, DEM–R) measures was significantly correlated with WAIS or WAIS–R scores in the Alzheimer group, consistent with the effect of dementia in these patients. In the other patient groups and controls, the correlations were all statistically significant. To determine whether the size of the correlations differed between controls and individual patient groups, the values were transformed to Fisher's z statistics and compared in the appropriate manner. In all comparisons of the control and Alzheimer groups, the size of the correlations were significantly different. However, no differences in the size of correlations were observed between controls and other patient groups.

In order to examine whether the size of the reading test (NART/NART–R) correlations with current IQ (WAIS/WAIS–R) were significantly greater than were the DEM/DEM–R correlations with current IQ, William's test for differences between dependent correlations was applied to provide t statistics for each comparison. In the combined controls and total patient groups, NART was more closely associated with WAIS than was DEM [controls: $t(46) = 2.59$, $p < .05$; patients: $t(90) = 2.40$, $p < .05$], and NART–R was more closely associated with WAIS–R performance than was DEM–R [controls: $t(46) = 2.50$, $p < .05$; patients: $t(89) = 2.82$, $p < .01$]. In the individual patient group analyses, there were no significant differences in the size of correlations in temporal, frontal or Alzheimer groups, but in the Korsakoff patients, both NART and NART–R were again more predictive of the respective FSIQ scores than were DEM or DEM–R [$t(30) = 2.72$, $p < .05$; $t(30) = 2.89$, $p < .01$, respectively].

To test the theory put forward by Crawford et al. (1989a), that a combination of NART and demographic variables could produce a more accurate estimate of intellectual function than either independently applied approach, we calculated new predicted FSIQ scores using their supplied equation: Predicted FSIQ = (135.96 − 0.79 × NART errors − 4.6 × sex) − (2.15 × class) + (0.112 × age).

Strong, significant correlations between the combined measure and WAIS were observed in controls and across the entire patient group (see bottom row in Table 5). Within the individual patient groups, clear associations were also demonstrated in temporal, frontal, and Korsakoff groups. However, as found with the individually applied predictors, the combined measure was not significantly correlated with WAIS performance in Alzheimer patients. A comparison of NART and the combined measure (in terms of the size of

Table 4. Correlations between estimates of premorbid full-scale intelligence

Correlations	Controls	Temporal	Frontal	Korsakoff	Alzheimer	All patients
NART & NART–R	1.0***	1.0***	1.0***	1.0***	1.0***	1.0***
DEM & DEM–R	.93***	.96***	.96***	.92***	.95***	.93***
NART & DEM	.41**	.51	.68**	.56**	.49**	.48***
NART–R & DEM–R	.47**	.47	.63*	.61***	.46*	.49***

***$p < .001$; **$p < .01$; *$p < .05$.

Table 5. Correlations between predicted and actual full-scale IQ scores

Correlations	Controls	Temporal	Frontal	Korsakoff	Alzheimer	All patients
NART & WAIS	.75***	.74**	.83***	.77***	.26	.50***
NART–R & WAIS–R	.73***	.69*	.83***	.72***	.25	.51***
DEM & WAIS	.50**	.70**	.73**	.46**	.23	.27**
DEM–R & WAIS–R	.46**	.68*	.61*	.38*	.16	.25*
Combined measure & WAIS	.76***	.80**	.88***	.78***	.28	.49***

***$p < .001$; **$p < .01$; *$p < .05$.

the correlations with WAIS) failed to produce significant differences in controls, the total patient group or in the individual patient groups. These results indicate that the combined approach, based on NART scores and demographic variables, did not provide a significantly more accurate estimate of intellectual ability than NART alone.

DISCUSSION

The present study provides little evidence for the superiority of an estimate of premorbid intellectual ability that is based on demographic variables rather than current NART performance. The NART, both in its original and revised formats, was correlated with WAIS and WAIS–R scores to a significantly greater extent than were the demographically based equations (DEM, DEM–R) in controls and across the total patient group. Furthermore, within individual patient groups, this observation also held true for the Korsakoff patients, and there was a trend in the same direction for the frontal group. An estimate of FSIQ based on a combination of NART and demographic variables failed to add significantly to the variance in actual WAIS FSIQ scores afforded by the NART on its own. This latter finding is consistent with previous reports by Blair and Spreen (1989) and Grober and Sliwinski (1991), but inconsistent with the observations by Crawford et al. (1990) and Willshire et al. (1991).

An essential consideration in the choice of measure used to estimate premorbid levels of intelligence is the accuracy with which it predicts intelligence in the normal population. NART, in both its original and revised formats, produced a mean FSIQ that was within a single point of actual mean WAIS and WAIS–R FSIQ in healthy controls. This was also true of DEM–R but not DEM. However, NART and NART–R were better predictors of individual WAIS performance than DEM and DEM–R. In controls, over 50% of the variability in WAIS and WAIS–R was directly predictable from the variability in the respective NART measures compared with approximately 25% predicted from the variability in DEM and DEM–R. On these grounds, NART/NART–R must be seen as a more reliable measure of general intelligence in unimpaired individuals.

Despite the relatively high correlations of NART/NART–R estimates with actual FSIQ scores in the Korsakoff group, the reading test produced predicted mean IQs that were lower than those based on demographic variables. In this respect, our findings are in line with those of O'Carroll et al. (1992), which were used as evidence for NART underestimating level premorbid IQ in Korsakoff patients. In the present study, Korsakoff patients produced a NART mean estimate that approached 2 IQ points above actual (current) WAIS score. The equation based on demographic variables (DEM) resulted in an IQ mean estimate that was nearly 8 IQ points above the WAIS score. However, the revised measures (NART–R and DEM–R) were less discrepant in their estimates of WAIS–R FSIQ, which differed only by a single point (Table 3).

In the context of previous reports of NART underestimating premorbid IQ in Korsakoff patients (e.g., Crawford et al., 1988; O'Carroll et al., 1992), the present results indicate that the revised IQ conversion data (NART–R) may offer a more accurate estimate than the original. It is also likely that differences in recruitment and/or diagnostic criteria may have contributed to some extent to the discrepancies among the studies, in particular the acuteness of onset (Cutting, 1978) and the strenuousness with which patients with concomitant pathology had been excluded.

Equivalent NART/NART–R scores were observed in frontal patients and controls. Contrary to the postulation that a failure in cognitive error-checking in frontal patients may lead to depressed NART performance (O'Carroll, 1995), equivalent NART scores were observed in frontal patients and controls. There are surprisingly few studies that have specifically investigated the validity of the NART in frontal lesion patients. However, Crawford and Warrington (2002) have reported that the NART performance of an anterior lesion sample ($N = 36$) did not differ significantly from controls ($N = 170$).

The findings clearly indicate that large NART/WAIS and NART–R/WAIS–R discrepancies and low intercorrelations are characteristic of Alzheimer patients but not of the other patient groups. In the Alzheimer patients, estimates based on NART were closer to WAIS scores than were those produced by the demographic equations. This might possibly have been caused by a very early deterioration in reading ability associated with disease progression although the lack of any significant differences in NART/NART–R scores across the subject groups mitigates against any weight being placed on this. However, all these patients were in relatively early (but definite) stages of dementia when tested.

Had there been more variability in the length of time between the onset of dementia and assessment, it would have been possible to address the issue of deterioration in reading ability with disease progression through a comparison of patients' NART scores at different stages.

Overall, the present results appear reassuring, given the extent to which the NART is applied in clinical, medicolegal and research settings (e.g., Cools et al., 2000; Isaac & Mayes, 1999; Langdon & Thomas, 1999; Lorch et al., 1999; Mavaddat et al., 2000; McKetin & Mattick, 1997; Mehta et al., 1999; Pantelis et al., 1999; Parkin et al., 1999). While demographic variables did produce significant correlations with intelligence, these were significantly lower than those based on NART performance in patients and healthy controls, and a combined reading test/demographic measure did not add significantly to the total variance in WAIS/WAIS–R accounted for, a finding consistent with earlier studies (Blair & Spreen, 1989; Grober & Sliwinski, 1991). We are not advocating the "bandwagon" approach, warned against by O'Connell (1995), of automatically choosing NART to the exclusion of other estimates of premorbid functioning in all clinical conditions. However, given its superiority to the demographically based approach in terms of its correlations with current intelligence levels in controls and patients, and high levels of interrater and test/retest reliability (Crawford et al., 1989a; O'Carroll, 1987), its continued use as a pragmatic estimate of premorbid ability in a number of conditions appears warranted. These conditions include early Alzheimer dementia and the Korsakoff syndrome, although NART scores are likely to deteriorate in more advanced Alzheimer dementia, semantic dementia, or other disorders that produce a surface dyslexia.

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REFERENCES

Barona, A., Reynolds, C.R., & Chastain, R. (1984). A demographically based index of premorbid intelligence for the WAIS–R. *Journal of Consulting and Clinical Psychology, 52*, 885–887

Blair, J.R. & Spreen, O. (1989). A revision of the National Adult Reading Test. *Clinical Neuropsychologist, 3*, 129–136.

Cockburn, J., Keene, J., Hope, T., & Smith, P. (2000). Progressive decline in NART score with increasing dementia severity. *Journal of Clinical and Experimental Neuropsychology, 22*, 508–517.

Colchester, A., Kingsley, D., Lasserson, D., Kendall, B., Bello, F., Rush, C., Stevens, T., Goodman, G., Heilpern, G., Stanhope, N., & Kopelman, M.D. (2001). Structural MRI volumetric analysis in patients with organic amnesia, 1: Methods and comparative findings across diagnostic groups. *Journal of Neurology, Neurosurgery, and Psychiatry*, *71*, 13–22.

Cools, R., Brouwer, W.H., de Jong, R., & Slooff, C. (2000). Flexibility, inhibition, and planning: Frontal dysfunctioning in schizophrenia. *Brain and Cognition, 43*, 108–112.

Coltheart, M., Patterson, K., & Marshall, J.C. (Eds.). (1987). *Deep dyslexia* (2nd ed.). New York: Routledge & Kegan Paul.

Crawford, J.R. (1992). Current and premorbid intelligence measures in neuropsychological assessment. In J.R. Crawford & W.W. McKinlay (Eds.), *A handbook of neuropsychological assessment* (pp. 21–49). London: Erlbaum.

Crawford, J.R. & Allan, K.M. (1997). Estimating premorbid WAIS–R IQ with demographic variables: Regression equations derived from a UK sample. *Clinical Neuropsychologist*, *11*, 192–197.

Crawford, J.R., Nelson, H.E., Blackmore, L., Cochrane, R.H.B., & Allan, K.M. (1990). Estimating premorbid intelligence by combining the NART and demographic variables: An examination of the NART standardization sample and supplementary equations. *Personality and Individual Differences*, *11*, 1153–1157.

Crawford, J.R., Parker, D.M., & Besson, J.A.O. (1988). Estimation of premorbid intelligence in organic conditions. *British Journal of Psychiatry*, *153*, 178–181.

Crawford, J.R., Parker, D.M., Stewart, S.E., Besson, J.A.O., & De Lacey, G. (1989a). Prediction of WAIS IQ with the National Adult Reading Test: Cross-validation and extension. *British Journal of Clinical Psychology*, *28*, 267–273.

Crawford, J.R., Stewart, L.E., Cochrane, R.H.B., Foulds, J.A., Besson, J.A.O., & Parker, D.M. (1989b). Estimating premorbid IQ from demographic variables: Regression equations derived from a UK sample. *British Journal of Clinical Psychology*, *28*, 275–278.

Crawford, J.R. & Warrington, E.K. (2002). The Homophone Meaning Generation Test: Psychometric Properties and a Method for Estimating Premorbid Performance. *Journal of International Neuropsychological Society*, *8*, 547–554.

Cutting, J. (1978). The relationship between Korsakoff's syndrome and "alcoholic dementia." *British Journal of Psychiatry*, *132*, 240–251.

Folstein, M.F., Folstein, S.E., & McHugh, P.R. (1975). 'Mini-mental' state: A practical method for grading the cognitive state of patients for the clinician. *Journal of Psychiatric Research*, *12*, 189–198.

Franzen, M.D., Burgess, E.J., & Smith-Seemiller, L. (1997). Methods of estimating premorbid functioning. *Archives of Clinical Neuropsychology*, *12*, 711–738.

Fromm, D., Holland, A.L., Nebes, R.D., & Oakley, M.A. (1991). A longitudinal study of word-reading ability in Alzheimer's disease: Evidence from the National Adult Reading Test. *Cortex*, *27*, 367–376.

Grober, E. & Sliwinski, M. (1991). Development and validation of a model for estimating premorbid verbal intelligence in the elderly. *Journal of Clinical and Experimental Neuropsychology*, *13*, 933–949.

Hart, S., Smith, C.M., & Swash, M. (1986). Assessing intellectual deterioration. *British Journal of Clinical Psychology*, *25*, 119–124.

Isaac, C.L. & Mayes, A.R. (1999). Rates of forgetting in amnesia: II. Recall and recognition of word lists at different levels of organisation. *Journal of Experimental Psychology: Learning, Memory and Cognition*, *25*, 963–977.

Kartsounis, L.D., Poynton, A., Bridges, P.K., & Bartlett, F.R. (1991). Neuropsychological correlates of stereotactic subcaudate surgery. *Brain*, *114*, 2657–2673.

Kopelman, M.D. (1985). Rates of forgetting in Alzheimer-type dementia and Korsakoff's syndrome. *Neuropsychologia*, *23*, 623–638.

Kopelman, M.D. (1989). Remote and autobiographical memory, temporal context memory and frontal atrophy in Korsakoff and Alzheimer patients. *Neuropsychologia*, *27*, 437–460.

Kopelman, M.D., Lasserson, D., Kingsley, D., Bello, F., Rush, C., Stanhope, N., Stevens, T., Goodman, G., Heilpern, G., Kendall, B., & Colchester, A. (2001). Structural MRI volumetric analysis in patients with organic amnesia, 2: Correlations with anterograde memory and executive tests in 40 patients. *Journal of Neurology, Neurosurgery, and Psychiatry*, *71*, 23–28.

Kopelman, M.D., Stanhope, N., & Kingsley, D. (1999). Retrograde amnesia in patients with diencephalic, temporal lobe or frontal lesions. *Neuropsychologia*, *37*, 939–958.

Langdon, D.W. & Thompson, A.J. (1999). Multiple sclerosis: A preliminary study of selected variables affecting rehabilitation outcome. *Multiple Sclerosis*, *5*, 94–100.

Lorch, M., Ferner, R., Golding, J., & Whurr, R. (1999). The nature of speech and language impairment in adults with neurofibromatosis 1. *Journal of Neurolinguistics*, *12*, 157–165.

McKetin, R. & Mattick, R.P. (1997). Attention and memory in illicit amphetamine users. *Drug and Alcohol Dependence*, *48*, 235–242.

Mavaddat, N., Kirkpatrick, P.J., Rogers, R.D., & Sahakian, B.J. (2000). Deficits in decision-making in patients with aneurysms of the anterior communicating artery. *Brain*, *123*, 2109–2117.

Mehta, M.A., Sahakian, B.J., McKenna, P.J., & Robbins, T.W. (1999). Systemic sulpiride in young adult volunteers simulates the profile of cognitive deficits in Parkinson's disease. *Psychopharmacology*, *146*, 162–174.

Nelson, H.E. (1982). *National Adult Reading Test*. Windsor, UK: NFER-Nelson.

Nelson, H.E. & McKenna, P. (1975). The use of current reading ability in the assessment of demetia. *British Journal of Social and Clinical Psychology*, *14*, 259–267.

Nelson, H.E. & O'Connell, A. (1978). Dementia: The estimation of pre-morbid intelligence levels using the new adult reading test. *Cortex*, *14*, 234–244.

Nelson, H.E. & Willison, J.R. (1991). *The Revised National Adult Reading Test–Test manual*. Windsor, UK: NFER-Nelson.

O'Carroll, R.E. (1987). Does the National Adult Reading Test hold true in dementia? *British Journal of Clinical Psychology*, *26*, 315–316.

O'Carroll, R.E. (1995). The assessment of premorbid ability: A critical review. *Neurocase*, *1*, 83–89.

O'Carroll, R.E., Moffoot, A., Ebmeier, K.P., & Goodwin, G.M. (1992). Estimating pre-morbid intellectual ability in the Alcoholic Korsakoff's syndrome. *Psychological Medicine*, *22*, 903–909.

Office of Population, Censuses and Surveys. (1980). *Classification of occupations*. London: HMSO.

Pantelis, C., Barber, F.Z., Barnes, T.R.E., Nelson, H.E., Owen, A.M., & Robbins, T.W. (1999). Comparison of set-shifting ability in patients with chronic schizophrenia and frontal lobe damage. *Schizophrenia Research*, *37*, 251–270.

Parkin, A.J., Ward, J., Bindschaedler, C., Squires, E.J., & Powell, G. (1999). False recognition following frontal lobe damage: The role of encoding factors. *Cognitive Neuropsychology*, *16*, 243–265.

Patterson, K., Graham, N., & Hodges, J.R. (1994). Reading in Dementia of the Alzheimer Type: A preserved ability? *Neuropsychology*, *8*, 395–407.

Schonell, F. (1942). *Backwardness in the basic subjects*. London: Oliver & Boyd.

Stebbins, G.T., Wilson, R.S., Gilley, D.W., Bernard, B.A., & Fox, J.H. (1990). Use of the National Adult Reading Test to estimate premorbid IQ in dementia. *Clinical Neuropsychologist*, *4*, 18–24.

Taylor, R. (1999). National Adult Reading Test performance in established dementia. *Archives of Gerontology and Geriatrics*, *29*, 291–296.

Wechsler, D. (1955). *Wechsler Adult Intelligence Scale*. New York: The Psychological Corporation.

Wechsler, D. (1987). *Wechsler Memory Scale–Revised*. New York: The Psychological Corporation.

Willshire, D., Kinsella, G., & Prior, M. (1991). Estimating WAIS–R IQ from the National Adult Reading Test: A cross-validation. *Journal of Clinical and Experimental Neuropsychology*, *13*, 204–216.

Wilson, R.S., Rosenbaum, G., Brown, G., Rourke, D., Whitman, D., & Grisell, J. (1978). An index of premorbid intelligence. *Journal of Consulting and Clinical Psychology*, *46*, 1554–1555.