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Parietal Signs and Sinister Prognosis in Dementia

A Four-Year Follow-Up Study

RONAN O'CARROLL, JANICE WHITTICK and ELIZABETH BAIKIE

Thirty elderly demented subjects were assessed in 1985 using a neuropsychological test battery which included tests of parietal lobe function that are allegedly predictive of outcome. Four years later, 29 out of the 30 subjects were followed up. Twelve had died. There were no differences between survivors and deceased in terms of age, pre-morbid intelligence, years of full-time education, or scores on parietal tests. However, proportionally more of the women had died, and those subjects with more global cognitive impairment in 1985 were significantly more likely to have died by 1989. Those who scored lower on an aphasia measure in 1985 were more likely to have died. None of the variables differentiated between survivors and deceased Alzheimer subjects.

Roth & Morrisey (1952) defined dementia (senile psychosis) as "an illness of insidious onset pursuing a uniformly progressive course with steadily augmenting mental decay". Since then attempts have been made at defining subgroups of dementing patients who differ in terms of memory dysfunction and life expectancy. Kral (1962) described two types of memory dysfunction, one with a slow progression and the other with a quick and malignant progression. Patients who could remember an event without remembering the details were regarded as suffering from benign memory dysfunction; patients completely unable to recall events of the recent past were regarded as suffering from malignant memory dysfunction. Kral (1962) reported that the incidence of death over four years was higher in the 'malignant' as opposed to the 'benign' memory group.

McDonald (1969) attempted to follow on from Kral, and isolated two subgroups of patients with senile dementia who had differing natural histories. He recruited 51 women who were given a simple test of memory, a neuropsychological test of parietal lobe dysfunction, and a test of dysphasic disturbance. In addition, the patients completed the Weigl–Goldstein–Scheerer colour form sorting test. (This battery was later named the "Kew Cognitive Map".) McDonald found that he could best separate the subjects into two groups on the basis of scores on the parietal test. If they scored more than one error they were assigned to group A. If they scored zero or one error (out of six) they were assigned to group B. Group A was found to be significantly younger than group B, and at six-month follow-up, 26% of group A were dead compared with only 4% of group B. McDonald therefore concluded that patients who had a diagnosis of senile dementia and exhibited signs of parietal lobe dysfunction were (a) younger and (b) had a poorer prognosis in terms of life expectancy. This proved to be an influential and frequently cited study.

Whitehead (1976) tested a mixed diagnostic sample of elderly psychiatric patients and found that the Kew 'parietal test' had some degree of predictive value in terms of outcome. However, in a five-year follow-up study, Whitehead & Hunt (1982) failed to demonstrate differential outcome according to performance on the parietal scale. Hare (1978) looked at indicators of 'true dementia' in an elderly population and found that errors in the three Kew tests (memory, aphasia and parietal lobe functioning) could help predict a poor six-month outcome.

Naguib & Levy (1982) followed up 40 patients with senile dementia who had been assessed neuropsychologically and who had had computerised tomography (CT). Within two years 27 patients had died, and survivors had performed better than deceased on tests designed to assess parietotemporal lobe dysfunction. In addition, CT estimation of radiological density of the right parietal lobe was lower in those who died. Colgan (1985) reported results of a similar study. He followed up 48 patients who had senile dementia. The sample were all given a neuropsychological test battery and were assessed using CT. At six-month follow-up, ten subjects had died. Survivors were compared with the deceased on initial measures, and the only neuropsychological variable on which the two groups significantly differed was the general mental test score. On radiological indices, the deceased were found to have significantly lower mean attenuation densities in the parietal, occipital, and left thalamic regions.

Grady *et al* (1987) also presented data failing to support the hypothesis of different subgroups of

Alzheimer patients, based on age at onset, and failed to observe a faster rate of cognitive decline in younger patients.

Gilleard *et al* (1987) attempted to test the first of McDonald's propositions, namely that those elderly subjects who had senile dementia and who made several errors on the Kew parietal test tended to be younger than those who made one or no errors. They observed no such age difference (79.0 v. 79.3 years respectively). In the present study we attempted to test empirically the second of McDonald's influential propositions, that elderly demented subjects who make parietal test errors have a poorer prognosis in terms of life expectancy.

Method

Thirty psychogeriatric attenders at a day hospital who fulfilled DSM-III criteria (American Psychiatric Association, 1980) for dementia were recruited in 1985. The sample comprised 22 women and 8 men aged 65–86 years (mean 77.3, s.d. 5.6 years), and represented all psychogeriatric day-hospital attenders who fulfilled the recruitment criteria. All subjects had at least a six-month history of mental deterioration with no previous psychiatric history. All could repeat at least three digits forward and were capable of reading aloud five letters and five digits in standard lower-case typescript, to screen subjects with visual difficulties.

Each subject was assessed using the Clifton Assessment Procedure for the Elderly (CAPE) Survey Version (Pattie, 1981), which consists of an information/orientation (I/O) scale (a 12-item mental status questionnaire) and a physical disability (Pd) scale, completed by the day-hospital nursing sister. Subjects were also assessed on two of the subscales from the Kew Cognitive Map (McDonald, 1969) – the parietal scale and the aphasia scale. Subjects also completed the National Adult Reading Test (NART; Nelson, 1982) to provide an estimate of pre-morbid intellectual level, and a half-length version of the Mill Hill Vocabulary Scale synonym section (MHVS; Raven, 1982). Social class was determined by occupation, and number of years of full-time education was recorded for each subject.

The sample was divided into Alzheimer's subjects ($n = 18$), multi-infarct subjects ($n = 9$) and a mixed-aetiology group ($n = 3$) using the criteria specified by the Hachinski Index (Hachinski *et al*, 1975). Details regarding the intercorrelations of measures at this initial sampling point are reported by O'Carroll & Gilleard (1986).

One year later all surviving subjects (two had died) were retested using an abbreviated battery which comprised of the CAPE Survey Version, NART and MHVS. Details of this first follow-up are provided by O'Carroll *et al* (1987). Four years after the initial assessments the entire sample was followed up. A four-year follow-up was chosen as it was predicted that a significant proportion of this elderly demented sample would have died within this time. This length of follow-up is in line with the previously cited studies of Kral (1962) and Whitehead & Hunt (1982).

Results

One subject was lost to follow-up. One man and 11 women had died. Comparative analyses were carried out on all the initial assessment variables, survivors versus deceased, to see whether any of the measures were predictive of mortality. The results are summarised in Table 1. Proportionally more of the women had died, and those subjects more cognitively impaired in 1985 (as assessed by the CAPE I/O and CAPE total score) were more likely to have died over the next four years. In addition the Kew aphasia score differentiated the two groups, survivors having recorded a significantly higher score four years earlier. None of the other measures differentiated the two groups.

A separate set of analyses were carried out using the same variables, but restricted to the 18 subjects (12 survivors, 6 deceased) whose Hachinski Index was less than or equal to 4, thus classifying them as likely Alzheimer cases. None of the variables differentiated between deceased and surviving Alzheimer subjects.

The simple comparison of survivors versus deceased can potentially mask significant effects that predictor variables may have on survival. In order to investigate this possibility, the data were reanalysed using Cox's proportional-hazards model. This procedure allows one to assess the relative influence of predictor variables on survival data, and the results are summarised in Table 2. Predictor variables age, sex, CAPE total score, Kew parietal score and Kew aphasia score were built into the model. The dependent variable was taken as survival time from study entry (0–>48 months). None of the variables in isolation were significantly related to survival time from study entry, and taken together the variables accounted for only 32% of the observed variance, just achieving statistical significance ($P = 0.048$).

Table 1
Characteristics of the total demented sample ($n = 29$) (means (s.d.s.))

	Survivors ($n = 17$)	Deceased ($n = 12$)
Age: years	76.4 (5.4)	78.3 (5.9)
Sex	7M, 10F	1M, 11F*
Hachinski Index	4.3 (2.6)	5.1 (3.0)
Years in full-time education	9.8 (1.8)	9.1 (0.9)
CAPE information/ orientation score	5.3 (2.8)	3.1 (1.8)***
CAPE physical disability score	4.2 (2.3)	5.1 (1.8)
CAPE total score	1.2 (4.2)	−2.0 (2.2)***
NART IQ	101.2 (8.6)	99.2 (5.4)
MHVS score	9.8 (4.0)	10.7 (4.4)
Kew parietal score	5.2 (0.8)	4.4 (1.6)
Kew aphasia score	3.9 (1.5)	2.6 (1.6)**

*$P < 0.05$ (Fisher's exact test), **$P = 0.04$, ***$P = 0.02$ (ANOVA).

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Table 2
Cox's proportional-hazards model

Variable	d.f.	χ^2	P	D (R^2)
Age	1	1.33	0.2488	0.0525
Sex	1	2.55	0.1104	0.0960
Kew parietal score	1	1.14	0.2865	0.0452
Kew aphasia score	1	0.02	0.8802	0.0009
Cape total score	1	2.23	0.1351	0.0851
Model	5	11.16	0.0483	0.3174

More detailed survival analyses (e.g. comparing relative risk factors within Cox's model during the 48 months) were considered inappropriate given the relatively small data set. Focusing on the deceased subgroup only, the correlation between Kew parietal score and months of survival ($r = 0.47$) was statistically insignificant.

Discussion

In this four-year follow-up study we have failed to demonstrate the predictive powers of parietal signs in a sample of the demented elderly. The only measures which differentiated between the survivors and deceased were sex, an aphasia measure and degree of global cognitive impairment. Our observation of proportionally more women dying is at variance with the findings of Naguib & Levy (1982) and of Colgan (1985), who found a relative excess of men in their deceased sample. We have no obvious explanation for this difference, particularly as the sexes in our study failed to differ significantly on any of the measures which may have been related to life expectancy (e.g. age or severity). That our aphasia measure discriminated between survivors and deceased was also an unexpected finding. It is possible that this was a chance result, particularly as the *P* value was just significant at 0.04, or it may be that the aphasia measure was merely representing an index of severity of the dementing process. Evidence in favour of this latter explanation is provided by examining the initial correlations between measures: Kew aphasia scores correlated significantly with Cape information/orientation scores (rho = 0.49, $P < 0.01$).

It is interesting to note, however, with regard to the cognitive measures, that our data are very similar to those provided by Colgan (1985), who found that the only measure to differ significantly between survivors and deceased was score on a general mental test. As in the present study, scores on parietal or parietotemporal tests failed to differentiate deceased from survivors. Similarly, in a five-year prospective study, Whitehead & Hunt (1982) found that death was related to poor intellectual functioning. These authors also found that the Kew parietal scale failed to distinguish between deceased and survivors and stated that: "McDonald's (1969) result appears discordant, since he was able to demonstrate differential outcome according to performance on the Parietal Scale; but even his results could be explicable in terms of general deficits, since this task is very simple and impaired performance is unlikely except in the context of considerable dysfunction".

Apart from the initial study by McDonald (1969), the best support for the view that neuropsychological parietal signs are predictive of a sinister prognosis was suppled by Naguib & Levy (1982), who did find a significant difference between survivors and deceased on a test of parietotemporal dysfunction. However, it is important to note that this was not a specific and isolated finding – the authors also found significant differences between the two groups on mental test score and on a digit-copying test. Our interpretation of available evidence leads us to the conclusion that the degree of general cognitive impairment may have some predictive power in senile dementia, but we agree with Roth (1980), who argued that "focal features . . . after the age of 70 can rarely be given the same status as the parietal lobe phenomenon found . . . in the fifth and sixth decades". Where differences on parietal tests have been observed between survivors and deceased, in our opinion the most parsimonious interpretation is that these occur in the context of, and reflect, general severity of cognitive deterioration, and as the dementing process proceeds, patients may well accumulate cortical deficits (Grady *et al*, 1988; Gilleard *et al*, 1987). This argument does not deny the possible predictive importance of structural changes which may occur in the parietal lobe, and which may be assessed by CT, as presented by Naguib & Levy (1982). However, it is important to note that Colgan (1985), in his CT study, did not report specific parietal lobe differences; rather he found that parietal, occipital and left thalamic CT measures all predictively differentiated survivors from deceased, again suggesting a more global impairment among those demented elderly who have a sinister prognosis. Recent research does suggest that specific pathology in the parietal region may occur in some cases of dementia, and that this may be related to severity. For example, Hunter *et al* (1989) reported that cerebral blood flow in the left parietal region correlated almost perfectly (rho = 0.95) with the CAMDEX CAMCOG total score. Further evidence describing the relationship between parietal lobe pathology and severity of dementia has been provided by Johnson *et al* (1988) and Tamminga *et al* (1987).

We feel that the conclusion made by Gilleard *et al* (1987) remains justified, namely that the burden

of proof remains for investigators to show that clinical signs of parietal lobe impairment do indeed contribute unique clinical information concerning the course and nature of senile dementia, rather than merely reflecting the severity of deterioration.

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*Ronan O'Carroll, PhD, *Research Clinical Psychologist, MRC Brain Metabolism Unit, Royal Edinburgh Hospital, Morningside Park, Edinburgh EH10 5HF*; Janice Whittick, MPhil, *Senior Clinical Psychologist, Department of Psychology, Bangour Village Hospital, Dechmont, West Lothian EH52 6LW*; Elizabeth Baikie, MPhil, *Principal Clinical Psychologist, Royal Victoria Hospital, 13 Craigleith Road, Edinburgh EH14 2DN*

*Correspondence