mood. The usefulness of ECT in the syndrome was recorded by Enoch & Trethowan (1979), and it is of interest that its effectiveness in a mentally handicapped subject was similar to that in normal subjects.

This case demonstrates once more that it is possible to find presentations of even the more uncommon psychiatric syndromes in mentally handicapped people, with features little modified by their lower intellectual functioning. It also underlines the fact that just as the symptoms can be of a relatively conventional nature, so also can be the treatment: ECT was as useful in this case as it has been in people of normal intelligence.

References

ANDERSON, E. W. (1964) *Psychiatry*, London: Balliere, Tindall.

BURD, L. & KERBESHIAN, J. (1985) Tourette syndrome, atypical pervasive developmental disorder and Ganser syndrome in a 15-year-old visually impaired, mentally retarded boy. *Canadian Journal of Psychiatry*, **30**, 74–76.

ENOCH, M. D. & TRETHOWAN, W. M. (1979) Cotard's syndrome. In *Uncommon Psychiatric Syndromes*. Bristol: John Wright.

REID, A. H. (1984) Gilles de la Tourette syndrome in mental handicap. *Journal of Mental Deficiency Research*, **28**, 81–83.

SEGLAS, J. (1897) *La Delire de Negation*, Vol. 1. Paris: Masson.

Anthony Kearns, MB, BCh, MRCPsych, *Clinical Director, St Mary's, Drumcar, Dunleer, Co Louth, Ireland (formerly Senior Registrar, Harperbury Hospital, Radlett Herts, and Royal Free Hospital, Hampstead).*

(*Accepted 4 November, 1985*)

British Journal of Psychology (1987), **150**, 114–117

Senile Dementia and Parietal Lobe Dysfunction

Two samples of patients suffering from senile dementia were administered the Kew tests for parietal lobe function and aphasia. No evidence emerged of any clinical heterogeneity; parietal lobe test performance was found to be more a function of degree of impairment rather than of age.

Interest in the clinical differentiation of patients with senile dementia has a long history. Several earlier writers had suggested a distinction between 'simple' or benign senile dementia, and a more clinically impaired or symptomatic type (cf. Bleuler, 1928, Schenk, 1955). As growing evidence emerged of similar underlying neuropathology in presenile Alzheimer's disease and senile dementias, however, a clinical unity has been drawn (cf. Rossor, 1982). Nevertheless, interest has been maintained in possible heterogeneity in both clinical manifestations, aetiology and prognosis within the primary degenerative dementias of Alzheimer type, especially with regard to differences in age and focal cortical signs (cf. Seltzer, 1984).

A much quoted study was reported by McDonald (1969) who distinguished between type A and type B senile dementia patients, on the basis of signs of parietal lobe dysfunction. Type A referred to patients making more than one error on a six-item 'parietal' scale, whom McDonald found to be younger and yet to have a significantly enhanced mortality. In a later paper, Hare (1978) claimed that using McDonald's measures, patients who showed only memory impairment and disorientation but no parietal signs had quite favourable outcomes, unlike the clear dementias who had poor outcomes (and parietal signs). Roth (1980) has argued, however, that "focal features . . . after the age of 70 can rarely be given the same status as the parietal lobe phenomena found . . . in the fifth and sixth decades".

https://doi.org/10.1192/bjp.150.1.114 Published online by Cambridge University Press

While not employing McDonald's measures, other studies have examined different clinical features in senile dementia in relation to subsequent mortality, and two studies have demonstrated a relationship between 'temporoparietal' signs and enhanced mortality (Kaszniak *et al*, 1978; Naguib & Levy, 1982). At the same time such parietal lobe features as drawing ability and such temporal lobe features as nominal aphasia have been shown to correlate strongly with measures of severity of cognitive impairment within a population of senile dementia patients, supporting Roth's comment that focal features do not represent unique clinical phenomena in these cases, and thus may not be useful bases for a meaningful source of clinical heterogeneity (Skelton-Robinson & Jones, 1984; Moore & Wyke, 1984).

In neither McDonald's nor Hare's papers are there signs to exclude deliberately cases whose clinical features may have suggested a vascular aetiology to the dementia. In attempts to replicate McDonald's findings it would seem necessary (as he recognised at the time) to try and exclude possible cases of vascular (multi-infarct) dementia, and indeed to minimise the effects of physical health and of long-term institutionalisation. The present report describes data from two studies employing McDonald's Kew test, on patients selected to avoid the above complicating factors.

Method

Subjects

The first group of patients were seen and tested by E. S. using the Kew Cognitive Map. They were 20 female patients aged 65 or over attending psychogeriatric day hospitals or recently admitted to a psychogeriatric unit. All had a clinical diagnosis of senile dementia, no psychiatric history before the age of 65 years and none of the usual indices of possible multi-infarct dementia (i.e. no history of stroke, no focal neurological signs, no history of hypertension, no peripheral atherosclerosis). Laboratory investigations had not revealed any evidence of associated physical disorders. Mean age of this sample was 80.1 ± 4.6 years (range 70–87 years).

The second group of patients were seen and tested by R.O'C. using the parietal and aphasia scales of the Kew Map. Eighteen patients were selected on the basis of being in good health, attending a psychogeriatric day hospital while living at home, having a clinical diagnosis of senile dementia, no previous psychiatric history, age over 65 years, and a Hachinski index of 4 or less (Hachinski *et al*, 1975). Mean age of this sample was 78.3 ± 5.5 years (range 66–86 years).

All the patients had at least a 6-month history of mental deterioration and forgetfulness, all could repeat at least three digits forward, and all were capable of some degree of self-care. It is very difficult to date accurately the onset of dementia; however, no subjects with obvious presenile dementia were included in the analysis.

Measures

Both groups were tested on the 'aphasia' and 'parietal' subscales of the Kew Cognitive Map (McDonald, 1969). The Kew Aphasia test requires the subject to (1) name a wristwatch, (2) name the wrist strap or band, (3) name the buckle or clasp, and to state what the following appliances are used for: (4) refrigerator, (5) thermometer, (6) barometer. The Kew Parietal test was not described in detail by McDonald (1969). The version used in the present study required the subject to (1) show left hand on request, (2) touch left ear with right hand, (3) identify an object (key or coin) by touch alone, (4) correctly identify finger or hand when touched, (5) demonstrate normal 2 point discrimination, (6) construct a square with matches.

In both groups, apart from the Kew parietal and aphasia scales, the patients also completed other psychological tests, including a mental status test. The first group completed a 10-item extension of the Kew 'memory scale', the second the 12-item Information/Orientation scale of the C.A.P.E. (Pattie & Gilleard, 1979). Given the equivalence of most mental status test measures, the differences in these two tests seemed unlikely to be of significance (cf. Gurland, 1980).

Results

Table I presents the cross-tabulation of the aphasia and parietal scales. The rank order correlation between the two scales is $+0.70$, $P<0.001$, indicating a significant association between 'temporal lobe' and 'parietal lobe' signs. However, while patients who do badly on the aphasia test may still make no errors on the parietal test, the reverse is not the case, suggesting either that the appearance of 'parietal' signs is a later, more severe index of deterioration or that the test items in the aphasia scale are simply more difficult to perform.

When the sample is split according to McDonald's 'A' and 'B' types (A = those scoring more than 1 error on the parietal scale, B = those scoring 1 or less), and their mean

TABLE I

Cross-tabulation of Kew Aphasia and Parietal Scale Scores

Kew Parietal Scale	*0–1*	*2–3*	*4–5*	*6*	*Total*
Kew Aphasia Scale					
0–1	2	1	3	1	7
2–3	0	2	9	2	13
4–5	0	1	2	11	14
6	0	0	0	4	4
Total	2	4	14	18	38

https://doi.org/10.1192/bjp.150.1.114 Published online by Cambridge University Press

ages compared, no differences emerged. Group A X age = 79.0 years, $n = 13$; group B X age = 79.3 years, $n = 25$. In the first group of patients the correlation between age and parietal score was $\varrho = -0.077$ and in the second group $\varrho = 0.085$, both non-significant. Rank order correlations between the two samples' mental status test scores and aphasia scores were: $\varrho = +0.63$ ($n = 18$) and $+0.59$ ($n = 20$); between mental status test scores and parietal scores: $\varrho = +0.59$ ($n = 18$) and $+0.16$ ($n = 20$). The latter correlation is not significant: all others are significant at $P < 0.01$.

Discussion

The findings do not support McDonald's qualitative distinction between a younger parietal lobe-impairment group and an older benign parietal lobe-intact group of patients diagnosed as senile dementia. Rather the study suggests a general relationship between existing deterioration in mental status and the accumulation of 'cortical' deficits. (The insignificant correlation between mental status test score and parietal score in Group 1 is an exception.) Since 'parietal' signs seem to occur later than 'temporal lobe' signs, it might be expected that their presence will augur an increased probability of dying in the near future. Of course this assumed 'sequence' of deterioration may reflect the different level of difficulty of the items in the 'parietal' and 'aphasia' tests rather than the true progression of dementia. However, recent evidence (Colgan, 1985) seems to confirm that level of mental deterioration reflected in impaired mental status performance is of equal if not greater prognostic significance than 'parieto-temporal' impairment scores in predicting mortality within a dementing population.

The age differences that were observed in McDonald's study may be explicable when those factors leading elderly patients to be admitted into long-term care are considered. Thus Green & Timbury (1979) have reported that their day hospital patients who were married and subsequently institutionalised were behaviourally more deteriorated than similar patients living alone. Since older patients, especially older women, are more likely to be living alone, this may lead to their being less deteriorated on admission — findings which have indeed been noted from behaviour ratings (Gilleard, 1978).

It is possible that, as Roth (1980) suggests, in presenile dementia such cortical localising signs may be significantly more frequent and distinctive compared with senile dementia patients. Nevertheless, empirical studies such as those reported by Pearce & Pearce (1979) have not reported any greater frequency of 'dysphasia, dyspraxia or agnosia' between pre-senile and senile groups. Similar conclusions to those indicated here of a global progression in the development of cognitive deficits in dementia have been reached by Reisberg and his team (Reisberg, 1984). The weight of empirical evidence does not at present provide much support for any clear-cut clinical heterogeneity in the primary (non-vascular) dementias.

Finally, it may be argued that the failure to replicate McDonald's findings reflects the inadequacies of the Kew Cognitive Map, rather than the unimportance of parietal lobe dysfunction in differentiating subtypes of senile dementia (witness the evidence from CT regional density studies, such as those reported by Colgan, 1985). Whilst this may be so, the burden of proof remains for investigators to show that clinical signs of parietal lobe impairment do indeed contribute unique information concerning the course and nature of senile dementia, rather than merely reflecting the severity of deterioration.

References

BLEULER, E. (1928) *Textbook of Psychiatry*, 2nd ed. London: Longmans.

COLGAN, J. (1985) Regional density and survival in senile dementia. *British Journal of Psychiatry*, **147**, 63–66.

GILLEARD, C. J. (1978) An investigation into the nature of behavioural change in the dementias of old age. Unpublished Ph.D. thesis, University of Leeds.

GREEN, J. G. & TIMBURY, G. C. (1979) A geriatric psychiatry day hospital service; a five year review. *Age and Ageing*, **8**, 49–53.

GURLAND, B. J. (1980) The assessment of the mental health status of older adults. In *Handbook of Mental Health and Aging*, (eds J. E. Birren & R. B. Sloane). Englewood Cliffs, New Jersey: Prentice-Hall.

HACHINSKI, V. C., ILIFF, L. D., ZILHKA, E., du BOULAY, G. H., MARSHALL, J., ROSS RUSSELL, R. W. & SYMON, L. (1975) Cerebral blood flow in dementia. *Archives of Neurology*, **32**, 632–637.

HARE, M. (1978) Clinical checklist for the diagnosis of dementia. *British Medical Journal*, **ii**, 266–267.

KASZNIAK, A. W., FOX, J., GANDELL, D. L. & RAMSAY, R. G. (1978) Predictors of mortality in presenile and senile dementia. *Annals of Neurology*, **3**, 246–252.

MCDONALD, C. (1969) Clinical heterogeneity in senile dementia. *British Journal of Psychiatry*, **115**, 267–271.

MOORE, V. & WYKE, M. A. (1984) Drawing disability in patients with senile dementia. *Psychological Medicine*, **14**, 97–105.

NAGUIB, M. & LEVY, R. (1982) Prediction of outcome in senile dementia; a computed tomography study. *British Journal of Psychiatry*, **140**, 263–267.

PATTIE, A. H. & GILLEARD, C. J. (1979) *Manual for the Clifton Assessment Procedures for the Elderly (CAPE)* Sevenoaks: Hodder & Stoughton Educational.

PEARCE, J. M. S. & PEARCE, I. (1979) Nosology of Alzheimer's disease. In *Alzheimer's Disease: Early Recognition of Potentially Reversible Deficits*, (eds A. I. M. Glen & L. J. Whalley. Edinburgh: Churchill Livingstone.

REISBERG, B. (1984) Commentary on "Is Alzheimer's disease inherited?" by Folstein and Powell, *Integrative Psychiatry*, **2**, 171–173.

ROSSER, M. N. (1982) Dementia. *Lancet*, **i**, 1200–1203.

https://doi.org/10.1192/bjp.150.1.114 Published online by Cambridge University Press

ROTH, M. (1980) The diagnosis of dementia in late and middle life. In *The Epidemiology of Dementia*, (eds J. A. Mortimer & L. M. Schuman). Oxford: Oxford University Press.

SCHENK, V. W. D. (1955) *Folia Psychiatrica et Neurologica Neerland*, cited by Corsellis, J. A. N. & Meyer, A. (1959) Aspects of neuropathology in relation to mental disease. In *Recent Progress in Psychiatry*, (eds G. W. T. H. Fleming & A. Walk. Vol. 3, London: Churchill.

SELTNER, B. (1984) Commentary on 'Is Alzheimer's disease inherited?" by Folstein and Powell *Integrative Psychiatry*, **2**, 170–171.

SKELTON-ROBINSON, M. & JONES, S. (1984) Nominal dysphasia and the severity of senile dementia. *British Journal of Psychiatry*, **145**, 168–171.

C. J. Gilleard, PhD, *Formerly Lecturer in Clinical Psychology, University of Edinburgh, currently Senior Lecturer in Child Psychology, Middle East Technical University, Ankara, Turkey*; E. Spain, MPhil, *Senior Clinical Psychologist, Department of Clinical Psychology, 255 Crail Street, Glasgow*; *R. E. O'Carroll, PhD, *Clinical Psychologist, Department of Clinical Psychology.*

*Correspondence: *Andrew Duncan Clinic, Royal Edinburgh Hospital, Morningside Park, Edinburgh EH10 5HF.*

(*Accepted 17 October 1986*)

British Journal of Psychiatry (1987), **150**, 117–121

Brain Imaging in a Case of Capgras' Syndrome

A patient developed Capgras' syndrome as part of an interictal psychosis of epilepsy; magnetic resonance imaging revealed bilateral subcortical lesions in occipitotemporal and frontal regions. These findings have implications for the postulated association between Capgras' syndrome and neuropsychological deficits, in particular prosopagnosia.

The delusional misidentification of closely familiar persons as being alien imposters, despite the acknowledgement of an extreme resemblance, is known as Capgras' syndrome. This phenomena has attracted a variety of explanations. Capgras & Carette (1924) offered a psychoanalytical interpretation of the second case described, and such explanations still have their adherents (Berson, 1983). However, in their original description, Capgras & Reboul-Lachaux (1923) noted a prodrome of derealisation, and suggested that it was this compelling feeling of strangeness which determined the emergence of the delusion—a view which has found support in similar observations by Christoloudou (1977) and by Todd *et al* (1981). Others have remarked on the frequency of organic brain disease, and have proposed that underlying neuropsychological deficits, such as prosopagnosia (Hayman & Abrams, 1977) or sensory inattention (Stuss & Benson, 1984) are important in its evolution. These concepts are appealing, but remain speculative. The new brain imaging techniques, however, offer an opportunity to test such hypotheses in a more direct way. A case of Capgras' syndrome is described, in association with unusual cerebral lesions whose relevance to observed neuropsychological impairments and to the syndrome itself are of interest.

Case report

Jane is a 19-year-old, left-handed girl, the youngest of five daughters of caring, professional parents. There is no family history of psychiatric disorder or epilepsy. She was born by difficult forceps delivery, 2 weeks post-mature, and it was thought she had suffered perinatal intraventricular haemorrhage, although lumbar punctures were repeatedly negative. After being nursed in an incubator for 8 days, she recovered well, and milestones were within normal limits. At the age of 5 years, she developed complex partial epilepsy: her seizures comprised absence attacks, lasting several minutes, during which she would be unresponsive, clench her fingers, and pluck at her clothes. She often experienced a brief prodrome of flashing coloured lights.

https://doi.org/10.1192/bjp.150.1.114 Published online by Cambridge University Press