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Clinical Heterogeneity in Senile Dementia

By CARRICK McDONALD

Roth and Morrisey (1952), in isolating senile dementia (senile psychosis) as a distinct nosological entity, used as their definition "an illness of insidious onset pursuing a uniformly progressive course with steadily augmenting mental decay". In 1955 Roth defined senile psychosis as "a condition with a history of gradual and continuously progressive failure in the common activities of everyday life and a clinical picture dominated by failure of memory and intellect and disorganization of a personality, where these were not attributable to specific causes such as infection, neoplasm, chronic intoxication or cerebral vascular disease known to have produced cerebral infarction". Kay (1962) defines dementia as a "gross deterioration of intellectual capacity and of memory, together with severe and persistent disorientation for time and place". He diagnosed senile dementia "when there was no evidence of focal brain disease". Roth *et al.* (1966) describe senile dementia as "the extreme form of intellectual decay of old age".

It is obvious that, although dementia is fundamental to the concept of the extremely common dementing diseases, authorities are undecided whether to define it by social criteria (failure of activities of everyday life) or in global impressionistic terms (mental decay), or in terms of sub-functions (intellectual capacity and memory), or in terms of a supposed aetiology (intellectual decay of old age).

Similar difficulties are encountered in attempts at validation of the concept of senile dementia in pathological terms. Although Corsellis (1962) concluded that there was quite a good correlation between clinical diagnosis and neuro-pathological changes, 29 per cent. of his group of senile dements showed either slight or no cerebral atrophy. In 1966 Roth *et al.* correlated scores of dementia, psychological test performance, and age against a mean plaque count estimated in 37 senile demented patients. Although the mean plaque count correlated significantly with a dementia score and with psychological test scores, no significant correlation was found between the mean plaque count and the patient's age.

In addition there have been tentative clinical descriptions of a sub-group to be excluded from senile dementia. Kay (1962) said "it is important to note that disorientation of minor degrees . . . mild memory impairment . . . bodily decrepitude . . . were not regarded as evidence of senile or arteriosclerotic dementia." Kral (1962) described two types of memory dysfunction, one with a slow progression and the other with a quick or malignant progression, based on a qualitative distinction of types of amnesia. Patients who could remember an event without remembering the details of it were regarded as suffering from benign memory dysfunction. Patients with inability to recall events of the recent past "whereby not relatively unimportant data and parts of an experience but the experience as such cannot be recalled", were called malignant memory dysfunctions. Kral pointed out that incidence of death within a four-year observation period was higher in the malignant dysfunction than in the benign dysfunction group. If these groups have a different prognosis it is important to examine whether any clinical features can be used to differentiate them.

This paper examines a group of patients currently diagnosed as senile dementia and attempts to isolate, using clinical criteria, two groups with differing natural histories.

One problem was to show that different patterns of behaviour or function could be discerned and that these different patterns carried different prognoses. Two immediate difficulties were encountered. Since the prognoses of the two hypothesized conditions might differ, comparisons of function between

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cases could only be made when the stage of the illness was controlled for as far as possible, i.e. it was necessary to ensure that cases at the extreme end of the natural course of one condition were not compared with cases who had run, for example, only through 25 per cent. of the course of the other condition.

The second difficulty in dealing with cerebral function is that of the diaschitic effect described by von Monakow in 1914 and discussed by Brain (1965). Von Monakow stated that if a cerebral function is undermined it will have far-reaching effects on other functions. Therefore if functions A and B are deficient the reason could be that A has undermined B, or B has undermined A, or A and B are being undermined by an extraneous factor, or finally that A and B are being undermined independently by two factors. Therefore failure in a function is difficult to interpret. However, if functions A and B are tested and one function is intact the effect of diaschisis can be ignored. That is, in order to establish different patterns of function reliance must be placed on successful performance.

Material and Method

All female patients in Kew chronic geriatric mental hospital who were alive on a census date and who had been given a diagnosis of senile dementia on admission (N = 158) were tested on the Wechsler adult intelligence scale (WAIS). If they were able to make a score on any of the sub-tests of the WAIS the patients were admitted to the study. Patients were given six clinical tests of memory, six tests of parietal lobe function, and six tests for aphasia. In addition the patients attempted the Weigl-Goldstein-Scheerer colour form sorting test (Goldstein and Scheerer 1941). In this test the patient was required to sort discs into groups. The patient could use colour or shape, and if no progress was being made a sorting by shape was demonstrated, and the patient could then score by dividing the remaining discs by shape. Attempts at patterning the discs were regarded as failures. Performance on these tests was recorded on the Kew cognitive map devised for this purpose of screening patients clinically (Appendix 1).

In this way 51 subjects were obtained, with a mean age of 80·3 years. It is obvious that, as intended, the sample was heavily weighted in the direction of preserved abilities, as compared with the initial hospital census population.

The problem now resolved into two questions. Which test, if any, divided the population into two? Did this test show any prospect of meaningful clinical reference?

Table I

No. of Subjects	Error Score						
	0	1	2	3	4	5	6
Memory scale ..	0	0	0	2	8	8	33
Parietal scale ..	19	11	1	4	1	10	5
Weigl scale ..	1	19	5	26	–	–	–
Abstract scale ..	0	7	14	30	–	–	–
Aphasia scale ..	2	12	8	9	4	8	8

Results

Table I shows the error score in the various sub-scales. Memory showed heavy damage with minimal distribution across the scale, as was expected since all cases had been diagnosed as senile dementia clinically. Both the parietal error scores and Weigl error scores split the patients satisfactorily. This effect in the Weigl scores is exaggerated by the fact that there were only four possible scores as opposed to seven in the parietal scale.

Table II shows Weigl performance compared with parietal performance. Patients required to perform extremely badly on the Weigl scale before they began to show serious error scores on the parietal scale, i.e. parietal success may accompany serious Weigl deterioration and would, on this ground, seem the better test to divide patients if success rather than failure is the criterion of choice.

Table II

Weigl Error Score	N	Mean Parietal Error Score
0	1	0
1	19	0·36
2	5	0·4
3	26	3·85

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On consideration of the distribution of parietal error scores (Table I) it seemed reasonable to separate the subjects into those who scored no errors or one error (low error scores) as compared to those who scored more than one error (high error scores). This made sense clinically in that physical frailty often invalidated one of these tests, e.g. an arthritic patient might fail totally in attempting to make a square with matches.

The mean age of the patients with low error scores on these tests was 81·25 years, and the mean age of patients with high error scores was 79·0 years. If low error scores merely preceded high error scores in the natural history of senile dementia then low error scorers should have been younger as a group instead of older. Applying the Students' "t" test to these ages gave a difference significant at the 10 per cent. level of confidence, which was promising since this sample was a skewed one as mentioned above.

In order to study the natural histories of "low error scorers" and "high level scorers" a sample unskewed in the direction of preserved abilities was necessary. Therefore consecutive female geriatric admissions were screened clinically for the signs of dementia. If there was a history of strokes or epileptic seizures or if there were demonstrable focal neurological signs the patients were excluded as being suspect arteriosclerotic dementias. If cognitive disturbance presented, with fluctuating disorientation and restlessness, then an acute or subacute confusional state was diagnosed and the patient was retested two months after admission. If at that time confusion was no longer present clinically and the patient still presented a clinical dementia then she was included in the cohort. The patients in the cohort were required to perform the tests on the parietal scale. If they scored 0 or one error they were assigned to Group B. If they scored more than one error they were assigned to Group A.

In a cohort of 162 consecutive admissions to Kew mental hospital, 29 warranted a diagnosis of confusion-free senile dementia on admission. This group was subdivided into 12 of group A (high error) and 17 of group B (low error) whose mean ages were 79·6 and 83·4 years respectively.

The hypothesis that these groups came from the same population was tested using the Mann-Whitney "U" test (Siegel, 1956). Using a one-tailed test a probability value of 0·01 was obtained, supporting the hypothesis that the two groups came from different populations. At six months after admission 50 per cent. of group A (high error) were dead but only 6 per cent. of group B were dead, i.e. the older group on admission had the better six-month prognosis.

When the diagnoses were corrected retrospectively at two months, adding those cases who had shown confusion on admission, the parietal tests divided the resulting 57 senile dementias into 31 in group A and 26 in group B. The mean age of group A now became 76·4 years and that in group B, 83 years, thereby increasing the age difference between the groups. At six months' follow-up, 26 per cent. of group A were dead and only 4 per cent. of group B

Discussion

Thus cases currently labelled as senile dements can be satisfactorily divided into two groups using their performance on a series of tests, in particular those of parietal lobe function. The groups so created are meaningful in that they have different natural histories. The group with preserved parietal lobe function is significantly older and has a much better six-month prognosis than the group with deteriorated parietal lobe function. Since they are older as a group it is unlikely that the cases with a higher six-month life expectancy are showing an earlier stage of the condition. It is simpler to postulate the existence of two conditions currently inclusively labelled senile dementia, the first (group A) or "true" senile dementia attacking a younger age group and carrying a bad six-month prognosis, and the second (group B) manifesting itself in an older age group and carrying a very much better six-month prognosis.

Group B in this study may relate to Kral's (1962) "benign memory dysfunction" group. His method of assessing the quality of the memory upset leads to serious difficulties, however. If a patient claims to remember the Second World War but cannot remember that her

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principal enemies were the Germans and the Japanese, has she remembered the central event and forgotten "relatively unimportant" parts of the experience? If so she would qualify as a benign memory dysfunction without adequately demonstrating that she did, as she claimed, remember the central event. It is more likely that with subtler psychometric methods a quantitatively smaller memory disorder would be delineated in group B of this study, and this might correlate with Kral's observations.

Group B is composed of 80-year-olds and 90-year-olds and its symptoms are relatively slowly progressive. It is tempting, therefore, to equate it with a concept of cerebral senescence as opposed to the mild memory disorder of normal old age. However, the possibility of differing pathology with age must also be considered. It is possible that the younger age group has cerebral tissue which reacts more vigorously to the pathological process of senile dementia, in the same way that carcinoma shows a diminishing malignancy with age.

It is obvious that the demonstration of heterogeneity within the current group of senile dements, using common clinical criteria, opens the way to further research into genetic, dietary, and social aetiological factors. It is hoped to present a thorough and subtler psychometric study of the census group, as suggested above. It is also hoped to follow groups A and B to post-mortem, to ensure that the patchy dementia group, i.e. those with preserved parietal lobe function, do not suffer from subclinical cerebral arteriosclerosis to a significantly greater extent than do patients with a global dementia.

Summary

1. Following a study of the literature on senile dementia the author examined the possibility of dividing a group of such cases into two meaningful groups.

2. Those female senile dements in a psychogeriatric hospital who retained some cognitive ability were subjected to a series of common clinical tests plus the Weigl-Goldstein-Scheerer colour form sorting test. The results were noted, and six tests of parietal lobe function seemed to divide the group into two most satisfactorily, group B having relatively intact parietal function.

3. Using these tests 57 consecutive female admissions suffering from senile dementia were divided into the two groups, A and B. Group A had a significantly lower mean age than group B, and at six-months' follow-up 26 per cent. of them but only 4 per cent. of the older group were dead.

4. The findings are discussed.

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Appendix I: Kew Cognitive Map

Age at testing.....*Name* .....
Age on admission.....*Notes* .....
Mark a cross at the appropriate place to indicate failure.

Memory	
What year are we in?	.....
What month of the year is it?	.....
Who were we fighting in the 2nd World War? Germans	.....
Japanese	.....
What year were you born?	.....
What is the capital city of Australia	.....
Error score	.....

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Aphasia
- Name a wrist watch .....
- Name the wrist strap or band .....
- Name the buckle or clasp .....
- What is a refrigerator for? .....
- What is a thermometer for? .....
- What is a barometer for? .....

Error score .....

Parietal
- Show me your left hand .....
- Touch your left ear with your right hand .....
- Coin in right hand named as a 20*c.* 2*s.* or 1*d.* .....
- No tactile sensory inattention present .....
- Normal 2-point discrimination .....
- Construct a square with matches .....

Error score .....

Abstraction
- Subtract 27 from 65 (presented visually) .....
- Too many cooks spoil the broth .....
- A rolling stone gathers no moss .....

Error score .....

Weigl
- Able to sort by colour .....
- Able to sort by shape .....
- Able to sort when given an example .....

Error score .....

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