DEMENTIA: A CLINICAL AND EEG STUDY OF 274 PATIENTS OVER THE AGE OF 60

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INTRODUCTION

THE purpose of the investigation was the assessment of dementia from a clinical and EEG viewpoint in patients aged 60 and over, resident in a mental hospital containing a high proportion of elderly long-stay persons. All patients of suitable age were examined irrespective of diagnosis, provided that this was technically possible and, if alive, they were re-examined three years later, in the hope that a longitudinal study would give additional information as suggested by Roseman *et al.* (1952) and Sheridan *et al.* (1955).

The clinical assessment of dementia is of necessity somewhat vague and subjective. Because of this, correlation with EEG findings was attempted in two surveys on aged patients in dissimilar hospitals. The first, already reported (Turton, 1958), concerned a unit taking primarily short-term patients of good prognosis. The findings obtained did not lend any support to the view that the EEG was of value in differentiating between mild dementia and depression with agitation or retardation.

In the investigation described herewith, it was hoped that a group in which dementia was prominent in a considerable proportion of cases, might yield further information about the nature and extent of EEG changes in old age. Luce and Rothschild (1951, 1953) felt there was a direct correlation between EEG abnormality and the degree of mental impairment in elderly patients. McAdam and McClatchey (1952) found very few abnormal records in patients over 60, who did not show evidence of clinical deterioration and, later, McAdam and Robinson (1956, 1958) using rating scales showed a trend of conformity between clinical and EEG assessments.

These findings were essentially confirmed by Mundy-Castle *et al.* (1954) and Silverman *et al.* (1955). More recently, Obrist and Henry (1958) found that 88 per cent. of a group of aged psychiatric patients with a functional disorder had normal tracings, whereas 79 per cent. of patients suffering from "brain syndrome" showed diffuse slow waves on the EEG.

However, not all workers have found so satisfactory a correlation between EEG findings and the clinical estimate. Liberson and Seguin (1945) stressed that a third of definitely organic patients have a normal or a borderline normal record. Strauss and Greenstein (1948), examining patients with cerebrovascular disease found that 67 out of 95 had records with no slow activity. Even severe C.N.S. lesions were associated with a normal record in 24 patients.

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Weiner and Schuster (1956) found that only a half of their patients with minimal dementia had abnormal records.

Furthermore any abnormal EEG findings have to be assessed and compared with the known changes that occur in elderly persons (Obrist, 1951; Silverman *et al.*, 1955; Maggs and Turton, 1956). It was for this reason that it was considered desirable to investigate as large a sample as possible.

METHOD

(a) *Physical Examination*

Each patient was examined physically, particular attention being paid to the nervous and cardiovascular systems. Illnesses occurring between the two EEG examinations were noted and any change in physical signs.

(b) *Assessment of Dementia*

Dementia was assessed on clinical grounds, from the patients' general behaviour and performance and simple tests of information and orientation; in addition, each patient was tested for his digit span and his performance with the Babcock Sentence (Zangwill, 1943).

Dementia was graded according to the following scale:

(*i*) *Absent:* No evidence of dementia clinically or with tests.

(*ii*) *Minimal* (+)*:* No evidence of dementia clinically. Diminished digit span and/or impaired performance with Babcock Sentence.

(*iii*) *Moderate* (++)*:* Organic dementia would be suspected on clinical grounds. There was definite impairment of recent memory, difficulty in solving simple sums, often some disorientation, and impaired performance on the tests. There was no deterioration in habits or character.

(*iv*) *Severe* (+++)*:* Obvious dementia clinically. In addition to a more severe impairment of memory, information and orientation, impaired digit span and meaningless attempts at the Babcock Sentence, there was some deterioration in habits—viz., unkempt appearance, urinary incontinence, crude feeding and often some disinhibition in behaviour.

(*v*) *Gross* (++++)*:* These patients were mostly in bed and doubly incontinent. There was gross memory impairment and disorientation. Much of their speech was meaningless.

(*vi*) *Not Tested:* These patients were all schizophrenics, many of them mute, and it was not possible to grade them according to the above scale because of their lack of co-operation in testing, or because of doubt as to whether deterioration in behaviour derived as much from psychosis as dementia.

In addition to assessing the degree of dementia, an attempt was made to classify the patients with respect to the type of dementia from which they suffered, viz., Presenile, Senile, Arteriosclerotic, G.P.I., and Dementia following head injury, carbon monoxide poisoning, alcoholism and epilepsy of long standing.

Arteriosclerotic dementia was diagnosed on the grounds of the history and associated physical signs, viz., history of minor or major strokes, and abnormal

C.N.S. signs associated with these, arteriosclerotic Parkinsonism, hypertension (>190/110) and hypertensive changes in the optic fundi. Cases without these signs and beginning after the age of 65 were classified as senile.

(c) *EEG and Criteria of EEG Abnormality*

The majority of the records were taken on a 6-channel portable machine, although a few were recorded on a conventional Ediswan 8-channel electroencephalograph. The portable machine was used so as not to upset easily agitated senile patients by removing them to strange surroundings. The recording was thus effected in the patient's ward or in close proximity to it. Even so a considerable number of the patients were too disturbed for a satisfactory recording on either the first or second occasion and this inevitably introduced some bias into the sample. Only those records which were of a satisfactory length and comprised a reasonable number of electrode patterns were accepted. The records were all reported by one of us (E.C.T.) according to a fixed pre-arranged plan, and without any clinical information whatsoever about the patient.

Apart from the records classified as normal the following five groups were recognized:

(*i*) *Borderline Normal.* These almost all showed a definite excess of fast activity with little or no alpha rhythm and usually some increase in the theta component.

(*ii*) *Generalized Slow Wave Changes of Moderate Degree.* These records showed a definite increase in the slower rhythms persistently in all or most areas of the cortex. Focal or localized changes were in some cases present but were not outstanding.

(*iii*) *Generalized Slow Wave Changes of Severe Degree.* The changes were similar to those noted above but they were more marked and always consisted of a delta dominant record, usually with waves of low frequency and high amplitude.

(*iv*) *Focal Slow Wave Changes.* These records showed predominantly a delta focus although there might be associated diffuse changes as well.

(*v*) *Epileptic Changes.* The records showed changes of the kind which would normally be considered of a characteristic epileptic pattern whether localized or generalized.

No patients were included who were on a high dosage of barbiturates or other drugs and, in fact, the majority were on no drugs at all although a few received nocturnal sedation. No patients had been treated with E.C.T. within three months of the recording. The subjects were awake and not hungry.

Results

The total number of cases studied was 274, of whom 110 were male and 164 female. Thirty-one (28·4 per cent.) of the males died and 43 (23·2 per cent.) of the females. The overall death rate for males and females for the three-year period of study was 27·4 per cent. There was no significant difference in the death rate as between the sexes or in the age groups 60–69 and 70–79. The death rate was slightly higher in the 80–89 age group in both sexes, and both patients aged 90+ at the beginning of the study died. Neither the presence of EEG abnormality, nor the type of EEG abnormality if present, had any

relation to the death rate, and the actual causes of death were in the great majority of cases quite unrelated to EEG abnormality.

The death rate did vary, however, according to the clinical diagnosis:

	Number of Cases	Deaths	Death Rate Over 3 Years (Per cent.)
Total	274	74	27·4
Arteriosclerotic dementia	15	5	33
Senile dementia	22	12	54
Senile paranoid psychosis	21	8	38
Schizophrenia	125	24	19
Affective psychosis	59	11	19
Epilepsy	12	6	50

TABLE I

	Number of Cases	Normal EEG	Abnormal EEG
"Normals" (Maggs and Turton, 1956)	82	38 (46%)	44 (54%)
All cases this series	274	133 (48%)	141 (52%)
No dementia	93 (30%)	57 (61%)	36 (39%)
Dementia not tested	77 (28%)	36 (46%)	41 (54%)
Dementia+	16	5 (25%)	11 (75%)
Dementia++	34	20 (60%)	14 (40%)
Dementia+++	32	12 (37%)	20 (63%)
Dementia++++	21	2 (7%)	19 (93%)
All dementia	103	39 (30%)	64 (70%)
All dementia>+	87	34 (39%)	53 (61%)
Presenile dementia	4	2	2
Arteriosclerotic dementia	15	3 (20%)	12 (80%)
Senile dementia	22	3 (14%)	19 (86%)
Schizophrenia	125	70 (56%)	55 (44%)
Affective psychosis	59	32 (54%)	27 (46%)
Senile paranoid psychosis	21	9 (43%)	12 (57%)
G.P.I.	5	4	1
Mental defect	5	4	1
Epilepsy	12	3 (25%)	9 (75%)

The General EEG Picture

The following observations may be made from an examination of Table I.

(*i*) The number of abnormal EEGs (52 per cent.) in this series taken as a whole is almost exactly the same as that found by Maggs and Turton (54 per cent.) in their series of normals over the age of 60.

(*ii*) If we compare the proportion of EEG abnormality (70 per cent.) in all those showing dementia with those showing no dementia on testing (30 per cent. abnormal EEGs), we find that the likelihood of an abnormal EEG is greater if dementia is present.

($\chi^2=10\cdot73$, which is significant at 0·01 level.)

Even so, we may still observe the number of abnormal EEGs among those graded as having no dementia, and the 39 normal records among

the 103 patients who exhibited dementia in some degree. The Table shows that normal records were still found among those graded as severely or grossly demented.

(*iii*) If, however, we compare the incidence of EEG abnormality among those having a definite organic dementia (presenile, senile, arteriosclerotic and G.P.I.) with those having no dementia, then the relationship between dementia and EEG abnormality is a good deal closer.

($\chi^2=15\cdot26$, which is significant at the 0·001 level.)

It may be that the relationship between EEG abnormality and organic dementia would have been a little closer had our clinical estimate of dementia been more accurate—i.e. that in spite of excluding 77 patients as being unsuitable for testing, we may still have graded some deteriorated schizophrenics as being demented when they were not. A comparison of the figures for definite organic dementia with those for all patients classified as demented lends some support to this view.

($\chi^2=4\cdot9$, which is significant at the 0·05 level.)

(*iv*) The figures for senile paranoid psychosis and for those in whom it was impossible to test for dementia were substantially the same as for a group of normals.

(*v*) The number of cases of presenile dementia is too small for analysis, although 2 of the 4 had normal EEGs; however, we did not do barbiturate and stimulation studies, such as those reported by Letemendia and Pampiglione (1958) or Liddell (1958).

(*vi*) The figures for schizophrenia and affective psychosis are similar, and show a slightly higher proportion of normal EEGs than the "normals".

EEG Changes After 3 Years

Although all 200 of the 274 patients who survived the three-year period had repeat physical examination and assessment of dementia, it was only possible to repeat the EEG on 190.

Of these 190, there was no EEG change in 162.

Twelve normal records showed moderate or severe diffuse slow-wave change on the second examination and one borderline record and one showing focal changes, showed moderate diffuse slow activity. One showing moderate slow activity showed a delta dominant record after three years and one normal record revealed epileptic changes. Only 16 of the 190 records, then, changed for the worse.

Twelve records showed improvement after three years (Borderline→normal, 2; Moderate slow→normal, 7; Moderate slow→borderline, 1; Epileptic→borderline or normal, 2).

Changes in Degree of Dementia and Change in EEG

In 9 patients dementia was judged to have increased over the three-year period; in 7 of these, the EEG remained unchanged, and in 2, there was a commensurate change in the EEG.

C.N.S. Signs and the EEG

Abnormal C.N.S. signs were present in 41 patients; 28 of these had abnormal EEGs and 13 were normal in this respect. In 13 patients there were

changes in C.N.S. signs between the two examinations, but only 4 of these showed a change in the EEG.

Further EEG Results

There was very little difference in the EEG pattern between the sexes or between the age-groups 60–69, 70–79, and 80–89.

The most common abnormality was a moderate degree of diffuse slow-wave change, and this was frequently present in patients with no dementia and among those who could not be tested. A severe degree of diffuse slow-wave change does, however, appear to be associated with dementia, although it may be objected that the number of cases showing such changes is small (9 out of 21 cases of gross dementia).

We could not associate any particular type of EEG abnormality with any of the various diagnostic groups. Again, when abnormality was present, a moderate degree of diffuse slow-wave change was the most common. Delta-dominant records and focal abnormalities were found only in those cases of dementia classified as arteriosclerotic or senile.

The number of borderline-normal EEGs was rather fewer than in previous series of this kind. The proportion was slightly higher in females and in the age-group 60–79. The finding had no relation to dementia or to the clinical diagnosis.

Discussion

The results in this investigation are similar to those obtained by one of us (Turton, 1958) investigating predominantly a quite different type of clinical material in a dissimilar hospital. Furthermore, despite the large numbers of persons investigated over a three-year period the actual value of the serial recordings proved to be slight, in contradistinction to the findings of Sheridan *et al.* (1955) who stated that it was exceptional for serial recordings taken at three-monthly intervals on one individual with a chronic brain syndrome of either arteriosclerotic or senile origin to remain unchanged.

Between severe organic deterioration and EEG changes the correlation was good, but until this degree was reached, the EEG was of no value in assessing the degree of mental involvement. This work therefore does not support the value of the EEG as a diagnostic weapon in the assessment of degree of dementia as some authors such as McAdam and Robinson (1956, 1958) have claimed. Furthermore, as a normal record is not infrequently found in a patient with obvious clinical impairment and an organic mental syndrome and an abnormal record in persons with no clinical signs or symptoms, the EEG may be positively misleading. Our results in this respect differ from those of Pampiglione and Post (1958), who concluded that patients over 60 with psychiatric disorders free from obvious clinical signs and symptoms of cerebral organic disease only rarely have abnormal EEGs, that a normal EEG is rare in the presence of definite clinical evidence of organic disease, and that a definitely normal EEG rules out important brain changes. Our results are disappointing, as an objective method of assessing the degree of dementia would be of value not only in "mixed" cases where there is a suspicion of dementia underlying depression but also where, by reason of psychosis, clinical examination is unsatisfactory.

It appears that the electrical response of the human brain varies widely from individual to individual under similar circumstances of stress. Until the

nature of the compensating mechanism is more clearly defined, the reasons for the large individual variations encountered cannot be truly assessed.

Summary and Conclusions

1. Two hundred and seventy-four patients between the ages of 60 and 79 were examined over a three-year period. This consisted of physical examination, assessment of dementia and EEG; it was only possible to perform repeat EEG examination on 190 out of the 274 patients, and repeat clinical examination on 200.

2. The criteria for dementia and for EEG abnormality are defined.

3. Although EEG abnormality is more likely in the presence of dementia where dementia is judged definitely to be absent, there are still a considerable number of abnormal EEGs even in the latter group and of normal EEGs where there is no clinical doubt about dementia. This statement holds good even in groups diagnosed as suffering from senile, arteriosclerotic and presenile dementia, and G.P.I., although EEG abnormality is more likely in these cases.

4. There is little correlation between clinical estimate of the degree of dementia and EEG abnormality, except where dementia is gross and obvious.

5. The results indicate that the EEG is of little help to the clinician when he cannot assess underlying dementia because of co-existent psychosis.

6. The proportion of EEG abnormality in patients suffering from schizophrenia, senile paranoid psychosis and affective psychosis was similar to that of a group of normals of the same age.

7. There was no correlation between change in degree of dementia and EEG change, but abnormalities on clinical examination of the C.N.S. were usually reflected in the EEG.

8. There was no EEG abnormality which could be said to be characteristic of any particular type of dementia in this series. The most common abnormality was a moderate excess of diffuse slow activity.

9. Of the 190 EEGs which were repeated after three years, 162 showed no significant change, 15 showed an increase in the amount of slow activity present and 12 returned to normal.

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