SHORT REPORT

EEG findings in dementia with Lewy bodies and Alzheimer's disease

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Abstract

***Objectives*—To evaluate the role of the EEG in the diagnosis of dementia with Lewy bodies (DLB).**

***Methods*—Standard EEG recordings from 14 patients with DLB confirmed at postmortem were examined and were compared with the records from 11 patients with Alzheimer's disease confirmed at postmortem**

***Results*—Seventeen of the total of 19 records from the patients with DLB were abnormal. Thirteen showed loss of alpha activity as the dominant rhythm and half had slow wave transient activity in the temporal lobe areas. This slow wave transient activity correlated with a clinical history of loss of consciousness. The patients with Alzheimer's disease were less likely to show transient slow waves and tended to have less marked slowing of dominant rhythm.**

***Conclusions*—The greater slowing of the EEG in DLB than in Alzheimer's disease may be related to a greater loss of choline acetyltransferase found in DLB. Temporal slow wave transients may be a useful diagnostic feature in DLB and may help to explain the transient disturbance of consciousness which is characteristic of the disorder.**

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Dementia with Lewy bodies (DLB) has recently been described as the second most common pathological type of dementia, accounting for up to 20% of elderly hospitalised patients coming to postmortem.[1 2] Many patients with DLB, however, are probably often erroneously misdiagnosed as having senile dementia of Alzheimer type (SDAT) or toxic delirium.[3] This difficulty in clinical diagnosis may now be partly overcome given the growing consensus about clinical diagnostic criteria[4]; however, their prospective utility and the role of investigative methods in enhancing accuracy of clinical diagnosis have yet to be evaluated.

The EEG is often carried out as part of the clinical assessment of patients with dementia as a non-invasive, widely available procedure which is generally well tolerated, even by patients with cognitive impairment. The EEG findings, which may support a diagnosis of Alzheimer's disease are slowing of alpha activity and an increase in slow frequency activity.[5] These findings may also differentiate dementing disorders from metabolic or toxic confusional states. Findings of EEG previously reported in a few patients with DLB[6–11] suggested that in addition to generalised slowing, sharp or triphasic waves may be characteristic features useful in clinical diagnosis.

There are pragmatic reasons to predict focal EEG abnormalities of the temporal lobe in DLB. The predilection sites for neuropathological change in DLB are in subcortical and temporal lobe structures and in addition, the hallucinations and loss of consciousness which are seen in some cases are often reminiscent of temporal lobe dysfunction, as seen, for example, in temporal lobe epilepsy.

The purpose of this study was to describe the characteristic EEG findings in a group of cases of DLB confirmed by postmortem, specifically looking for evidence of focal abnormalities in the temporal lobes, or for triphasic or sharp waves, and to compare these records with those from cases of SDAT confirmed by postmortem.

Subjects and methods

Fourteen cases of DLB confirmed by postmortem were selected on the basis of the availability of EEG records and 11 patients with SDAT were similarly available for comparison. The patients, who were aged between 64 and 89 at the time of EEG, were all from Newcastle hospitals and had died between 1984 and 1991. Neuropathological diagnosis was made according to previously published methods.[12] All of the patients had been comprehensively assessed in a specialist psychogeriatric unit where EEGs are carried out on all patients as part of a routine dementia assessment. Standard EEG techniques were used.

The EEG records were examined blind to diagnosis on two occasions by three raters. The raters knew that the patients had dementia but had no other clinical details. The EEGs were assessed on the following criteria: dominant frequency, presence of other frequencies, left/right asymmetry, mean amplitude, and presence of focal abnormalities including spikes, sharp waves, triphasic waves or transient slow wave activity.

Case notes were examined using a specially designed standardised schedule to record specific clinical features. The duration of illness was identified as being the time between first onset of symptoms, as reported by the care giver and the date of the EEG, given in months. The timing of the EEG was also expressed as a percentage of the total period from onset to death. It was noted whether the patients either experienced episodic loss of consciousness, or hallucinations. A global rating scale of severity at the time of the EEGs was made using the clinical dementia rating (CDR) of Hughes *et al*[13] (a score of 0 indicates no dementia, 0.5 indicates possible dementia, 1 indicates mild dementia, 2 indicates moderate dementia, and 3 indicates severe dementia).

Finally, clinical symptoms were examined for correlations with pathological diagnosis and EEG findings. The data were analysed using the statistical package for the social sciences (SPSS). When more than one variable was compared, correlation coefficients and *t* values are given.

Results

The DLB group contained seven men and seven women, the SDAT group five men and six women. Individual case details are given in the table. Three (21%) of the patients with DLB had been diagnosed as having Parkinson's disease before developing dementia (Parkinson's disease+DLB). The two groups were similar in age distribution (SDAT mean 73.4 (SD 10.21) years, DLB mean 78.1 (SD 7.24) years).

The DLB group had a slightly shorter duration of illness at the time of EEG (DLB mean 20.1 (SD 16.9) months), SDAT mean 31.4 (SD 15.6) months (p=0.100). When the timing of the EEG was expressed as the time of onset to EEG divided by time of onset to death, the two groups were similar (DLB mean (SD 25.2) 59.3%, SDAT mean (SD 22.9) 52.9%, Mann Whitney U = 64 (NS)). The distribution of CDR scores was similar at the time of EEG suggesting an equivalent degree of impairment between the two groups. No patient had a history suggestive of clinical seizures.

The EEG records of these 25 patients were examined and are referred to by the case num-

Electroencephalographic and clinical features of patients with dementia with Lewy bodies and senile dementia of Alzheimer type

Case No	*Age at EEG (y)*	*Time from onset of clinical symptoms to death (months)*	*Clinical dementia rating*	*Hallucinations*	*Episodic loss of consciousness*	*Time from onset of clinical symptoms to date of EEG recording (months)*	*Time of onset to EEG as % onset to death*	*Dominant activity*	*Other frequencies*	*TLSWT*
SDAT Patients:										
1	80	99	1	No	No	60	61	Alpha	Beta Theta	No
2	76	108	0.5	No	No	36	33	Alpha	Theta Beta	No
3[1]	64	96	2	No	Yes	47	49	Delta	Theta	Yes L/R
4	76	19	2	Auditory	No	16	84	Alpha	Theta (minimal)	No
5	77	50	1	No	No	36	72	Alpha	Theta Beta Delta	No
6	80	54	1	No	No	39	72	Alpha/ Theta	Nil	No
7	65	21	3	No	No	15	71	Delta	Theta Alpha	Yes
8	92	36	2	Yes, visual	No	4	11	Alpha	Theta Delta	No
9	76	63	2	No	No	30	48	Theta	Delta Alpha	No
10	54	128	1	No	No	29	23	Alpha	Theta Delta	No
11	67	57	2	No	No	33	58	Delta	Theta Beta	No
DLB Patients:										
12	85	107	2	No	No	17	16	Alpha	Theta Delta	No
13	82	32	2	Visual	Yes	20	63	Alpha	Beta	Yes L
14 Record 1	84	31	2	Visual	Yes	24	77	Theta	Delta Beta	No
Record 2								Theta	Delta	No
15[2]	84	6	1	No	Yes	5	83	polyrhythmic	Alpha Theta Delta	Yes L
16	72	15	0.5	No	No	4	27	Theta	Beta	No
17 Record 1	64	41	1	Visual	Yes	24	59	Alpha	Delta Theta beta	Yes L
Record 2								Alpha	Beta Theta	Yes L/R*
Record 3								Polyrhythmic	Delta Theta	No
18[3]	84	2	2	Visual and Auditory	No	1	50	Delta	Theta	No
19	68	20	2	Visual and Auditory	No	11	55	Alpha	Theta Delta	Yes L/R
20[4]	89	22	1	Visual	No	16	73	Theta	Delta	No
21	79	27	0.5	No	Yes	11	41	Theta	Nil	Yes L/R
22 Record 1	71	32	1	Visual	Yes	20	63	Delta	Theta	Yes L/R
Record 2[5]								Delta	Theta	Yes L
23[6]	77	24	2	Visual/olfactory/ gustatory	Yes	23	96	Delta	Theta	No
24 Record 1	79	70	2	No	No	69	99	Alpha	Theta Delta	No
Record 2								Theta	Alpha	Yes L
25	76	120	0.5	No	No	36	30	Alpha Theta	Delta	No

TLSWT = Temporal lobe slow wave transients.
*Increased in number from record 1.
Sharp waves and other EEG features:
[1]Sharp waves on left + right.
[2]Left posterior sharp wave.
[3]Slow waves anteriorly.
[4]Delta dominant anteriorly.
[5]Suggestion of sharp + slow wave complex.
[6]Sharp wave on left side.

bers in the table. The eleven cases of Alzheimer's disease had a single EEG recorded for each patient. A total of 19 records from 14 patients in the DLB group were examined, because three patients had two records each and one patient had three records. All but patients 2 and 4 (both SDAT) were clearly abnormal with alpha activity decreased outside the normal frequency. Preservation of alpha activity was more common in SDAT (55%) than in DLB (36%) (p=0.45). One (9%) patient with SDAT and seven (50%) patients with DLB had no remaining alpha activity. Six (55%) patients with SDAT and 11 (79%) with DLB had delta components. Overall, this suggests that patients with DLB show a greater tendency towards slowing of both dominant and non-dominant rhythms.

TEMPORAL LOBE SLOW WAVE TRANSIENTS

Temporal lobe slow wave transients were defined as episodes of delta or theta often associated with sharply contoured components but not strictly fulfilling the criteria for spikes (monophasic waves of duration <80 ms) or sharp waves (duration 80–200 ms). They had a duration <1 second and occurred in the temporal lobe areas only.

Temporal lobe transients were seen much more often in patients with DLB (seven out of 14 (50%)) whereas only two (18%) of the patients with SDAT showed this abnormality (p=0.11). Nine of the 19 DLB records showed this abnormality.

There was no significant correlation between temporal slow wave transients and either symmetry (p=0.57) or alpha dominance (p=0.19).

CORRELATION BETWEEN CLINICAL FEATURES AND TEMPORAL LOBE SLOW WAVE TRANSIENTS

Temporal lobe slow wave transients were highly correlated with reported episodes of loss of consciousness, both in the total group (p=0.01) and within the DLB group (p=0.05) whereas hallucinations (p=0.5), duration of illness (p=0.59), and global severity as measured by CDR (p=0.51) were not.

Discussion

Electroencephalographic findings in DLB have previously been described only for a few cases. A comprehensive review of the literature found only 13 descriptions of EEGs in DLB confirmed by postmortem.[6–9 11 14]

In this study 19 records from 14 patients with DLB confirmed by postmortem were examined. Seventeen of these records were abnormal. The key findings were a loss of alpha activity as the dominant rhythm in 13 of the records, presence of temporal lobe slow wave transients in 50% of cases, and a strong correlation between the presence of these temporal lobe slow wave transients and a clinical history of loss of consciousness.

Previous reports have commented on this slowing of dominant rhythm[5 7 11 14] and one author has described a case in which triphasic waves were present.[9] Periodic synchronous discharges have been described[8] as have the presence of sharp waves.[6 7]

No evidence of triphasic waves was found in the records examined, suggesting that this is not a frequent finding in DLB. Only two of the cases described here (5 and 19) had frontal slowing. No cases showed periodic synchronous discharges. Two patients with DLB in this study did had definite sharp waves, in addition to which patient 18 had a single sharp wave, which was not included in the analysis as it was unlikely to have been regarded as relevant in clinical practice.

Episodic loss of consciousness in now considered a supportive feature of DLB in the consensus criteria. The nature of these episodes of loss of consciousness is not clear. Proposed mechanisms[14] include gait and balance disorder, autonomic instability including postural hypotension, or an epileptic phenomenon. The finding in this paper of a relation between temporal lobe slow wave activity and a clinical history of loss of consciousness is indicative of a primary cerebral dysfunction.

This study is largely a descriptive account of the major EEG findings in DLB. The next step in this work should involve prospective serial EEG examinations from clinically diagnosed patients with DLB followed up to postmortem, with a larger group of patients with SDAT for comparison. An alternative and complimentary approach would be to investigate the role of quantitative EEG analysis in DLB.

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