Quantitative electroencephalography in Alzheimer’s disease: comparison with a control group, population norms and mental status

Verner Knott, PhD; Erich Mohr, PhD; Colleen Mahoney, BA; Vadim Ilivitsky, MD

Objective: Given that quantitative electroencephalography (EEG) has repeatedly shown excessive slow wave activity in dementia of the Alzheimer type (DAT) that increases with disease progression, we assessed the clinical utility of this tool by comparing various approaches used to assess slowing. Design: Cross-sectional study comparing quantitative EEG data from patients with DAT with normative data from an elderly control group and from EEG norms derived from a large population. Participants: 35 subjects diagnosed with probable DAT and 30 elderly controls. Outcome measure: EEG recorded from 21 scalp sites of each patient and elderly control during vigilance-controlled, eyes-closed, resting conditions was spectrally analyzed to yield measures of absolute and relative power in delta, theta, alpha and beta bands and indices of mean alpha band and total band frequency. Results: Group comparisons of raw or age-regressed z-score population normative values yielded different profiles with respect to direction of frequency band changes, regional topography and clinical rating correlations, but both procedures evidenced overall patterns of EEG slowing in DAT. However, both methodologies yielded only modest (75%) classification rates. Conclusion: Quantitative EEG remains a valuable research tool but, as yet, an unproven diagnostic tool, for DAT.

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Medical subject headings: alpha rhythm; Alzheimer disease; beta rhythm; delta rhythm; dementia; discriminant analysis; electrodiagnosis; electroencephalography; theta rhythm

Introduction

Despite the marked brain pathology evident post mortem, with the exception of cortical biopsy, in vivo biological markers are as of yet unable to provide a definite diagnosis of dementia of the Alzheimer type (DAT). Clinical assessment and diagnosis of DAT, achieving an accuracy of approximately 85%, is typically reached by a procedure of exclusion after laboratory, psychometric and structural or functional imaging tests, including noninvasive tests of neuroelectrophysiology. Neuroelectric methodologies, in the form of routine conventional or quantitative electroencephalography (EEG), have contributed significantly to the identification and characterization of the central pathophysiology underlying DAT. In numerous laboratories, including our own, spectral analysis of background rhythms during resting states has repeatedly demonstrated electric slowing which, although not pathognomonic of DAT, has, more or less, been found to correlate with the degree of clinically and psychometrically assessed cognitive impairment in patients with dementia.1,2

Of the multitude of spectral features, amplitude–power band activity, topography and peak frequency have been found to be DAT stage dependent.3,4 Early stages of the illness have been associated with increased theta activity, whereas later, more severe stages of the illness have evidenced a marked increase in delta activity that is often accompanied by activity decreases in alpha and beta frequencies and reductions in peak alpha frequency. Topographically, excessive slow wave activity has been reported to be relatively widespread across scalp recordings, but delta and theta increments have occasionally been found to be more prominent in left temporal regions7 and alpha and beta have tended to be located more anteriorly in patients with DAT than in controls.5,9

The noninvasiveness and nondemanding nature of quantitative EEG and its independence from educational and motivational factors make it an attractive and objective evaluative CNS tool in the diagnostic work-up of patients with probable DAT — patients who often lack the resources for a more advanced behavioural assessment of cognitive functions. In assessing the clinical usefulness of quantitative EEG, a panel of experts, jointly appointed by the American Clinical Neurophysiology Society (ACNS, formerly the American EEG Society) and the American Academy of Neurology, concluded that this technique may be useful in evaluating certain patients with dementia for whom clinical evaluation, neuroimaging and routine EEG studies are inconclusive.10 Laboratories attempting to routinely use this neuroelectric approach as a diagnostic aid must first acquire normative data that will allow the accurate description of the EEG features that differentiate DAT from healthy aging and are proportional to the functional impairment. Individual laboratories can acquire control data by systematically recording age-matched group samples (n ≥ 20), or they can use one of several commercially available EEG data banks derived from the testing of large (n > 100) samples.

This study attempts to advance the clinical usefulness of quantitative EEG by (a) comparing spectrally analyzed EEG power and frequency data of a group of patients with DAT with 2 sources of normative data: a normal age-matched control group and published adult age-regressed EEG population (n > 200) norms (“neurometrics”)11–14 and (b) examining the relation between distinctive EEG features derived from these 2 comparative procedures and global functional impairment, as assessed by the Mini-Mental State Examination (MMSE).15 As part of the former objective, the study addressed whether it is possible to accurately classify DAT patients and controls by discriminant analysis of power and frequency data.

EEG in Alzheimer’s disease

témoin âgé au repos, les yeux fermés et dans des conditions de vigilance contrôlée pour mesurer la puissance absolue et relative des bandes delta, thêta, alpha et bêta et produire des indices pour la bande alpha moyenne et la fréquence totale des bandes. Résultats : Les comparaisons entre les groupes de valeurs normatives de la population d’écarts réduits bruts ou après régression selon l’âge ont donné des profils différents en ce qui concerne l’orientation des changements de bandes de fréquences, la topographie régionale et les corrélations d’évaluation clinique, mais les deux interventions ont démontré des tendances globales au ralentissement de l’EEG dans les cas de DTA. Les deux méthodologies n’ont toutefois donné que des taux de classification modestes (75 %). Conclusion : L’EEG quantitative demeure un outil de recherche utile mais elle n’a pas encore fait ses preuves comme outil de diagnostic pour la DTA.
Methods

Subjects

Study participants included 35 (18 men) subjects diagnosed with probable DAT, according to criteria outlined in McKhann et al., and 30 (18 men) age-matched controls. The patients or their caregivers gave informed consent before participating in the study. A complete medical history was recorded and laboratory (including an EKG, EEG and blood and urine analyses), neurological (including computed tomographic scans to rule out other possible causes of dementia) and psychiatric screens, as well as clinical assessments, were performed before study entry. Subjects were excluded if they had a history of a psychiatric disorder, alcohol or drug abuse, head trauma, CNS disease (other than dementia in patients), severe physical illness or hypertension. All subjects had been free from CNS medications for at least 14 days before being tested.

Recording

EEG was recorded in a sound-attenuated chamber immediately adjacent to the control room housing the computer, video monitors, amplifiers and recorders. During recording sessions, subjects sat, semi-reclined with eyes closed, legs elevated and neck and arms supported for a 10-min EEG acquisition period. To keep vigilance state at a constant level, subjects were verbally alerted at 2-min intervals and any time there were signs of behavioural drowsiness.

Tin electrodes were positioned with an electrode cap on 21 scalp sites (Fp1, Fp2, Fpz, F3, F4, Fz, F7, F8, C3, C4, Cz, T3, T4, T5, T6, P3, P4, Pz, O1, O2, Oz), according to the 10/20 international system, and were referenced to electronically linked earlobes. Another electrode placed between Fpz and Fz served as ground. Electro-oculographic activity was recorded from an electrode positioned on the right external canthus, referenced to the mid-frontal site. All electrode impedances were kept below 5 kΩ. To reduce the frequency and extent of electro-oculographic and noncerebral artifacts, patients were informed before each recording of the possible contaminating effects of eye movements, muscular contractions and gross motor movements on EEG activity. Electrical signals were recorded with a Cadwell Spectrum 32 system (Cadwell Laboratories, Kennewick, Wash.) with amplifier bandpass settings of 0.5–70.0 Hz. Continuous analog-to-digital samples (200 Hz) for each of the recording amplifiers were stored on optical disk for later off-line analysis.

Statistical analysis

During off-line analysis, digitized data points from 48 artifact-free epochs (i.e., clear of contamination from muscle activity, lateral or vertical electro-oculographic deviations, body movement and perspiration) of 2.5-s duration were subjected to a Fast Fourier Transform (FFT) analysis for calculations, at each of the 21 scalp sites, of average (i.e., of the 48 epochs), absolute (µV2) and relative (%) power in the 4 standard EEG frequency bands: delta (1.5–3.5 Hz), theta (3.5–7.5 Hz), alpha (7.5–12.5 Hz) and beta (12.5–25.0 Hz). For descriptive purposes only, the raw EEG absolute and relative power values were topographically mapped and colour-coded using rectangular interpolation from the 4 nearest electrodes.

The derived raw EEG variables were log transformed, as recommended for neurometrics, and expressed as age-regressed z scores (proportional to probabilities) representing, in standard deviation units, the degree of departure from population norms derived from 278 (age range 17–82 years) medically and psychiatrically screened normal adults (made commercially available by Cadwell Laboratories). Multiple independent replications of the neurometric EEG norms have shown them to be independent of culture and socioeconomic status.

For the primary objective of comparing controls and patients, raw and z-score values for each power and frequency index were subjected to separate group (2 levels) × site (21 levels) split-plot analyses of variance (ANOVA), with both between (group) and within (site) subject factors. Because spectral ratios have been shown to be a sensitive discriminator of DAT, the ratio of absolute alpha power to theta power was also subjected to the same ANOVA procedure. Greenhouse–Geisser’s conservative degrees of freedom were used, where appropriate, to compute probability values so as to reduce type I errors associated with multiple tests and inflated degrees of freedom with repeated-measures ANOVAs. Follow-up tests of interactions were carried out by Student Neuman–Keuls tests, which controlled for experiment-wise error.

In addition to employing ANOVAs to examine raw and z-score group differences for power and fre-
quency, the “presence” of statistically abnormal patient “group” z-score deviations from normative values was examined by the procedure of Prichep and John,\textsuperscript{29} which involved multiplying the group z score by the square root of its sample size.

The secondary objective of relating EEG values of patients with disease severity was carried out by correlational statistics using Pearson’s product-moment test. To reduce the number of tests, MMSE scores were correlated only with the raw and z-transformed power and frequency values that were significantly different from controls and population norms. Again, to further reduce the number of tests, only the average values from electrodes significantly differentiating patient data and control data were used in the correlational analysis.

Results

Raw EEG

The mean age (and standard deviation [SD]) of the patients diagnosed with DAT was 72.2 (SD 7.9) years and of the controls was 73.7 (SD 5.8) years. The mean MMSE score for patients with DAT was 19.4 (SD 8.2) and for the control group was 73.7 (SD 5.8) years. The mean age (and standard deviation [SD]) of the patients diagnosed with DAT was 72.2 (SD 7.9) years and of the controls was 73.7 (SD 5.8) years.

Absolute and relative average power maps for the patients and control group are shown in Fig. 1 and Fig. 2, respectively. Absolute delta analysis yielded a significant group (\(F = 5.68, p < 0.02\)) and group \(\times\) site interaction (\(F = 3.39, p < 0.007\)), with follow-up tests showing greater power for patients at parietal (P3, P4), occipital (O1, O2) mid-temporal (T3–T4), posterior-temporal (T5, T6) and at F8, Pz and Oz sites (\(p < 0.05\)). Patients also exhibited greater theta power across all scalp sites (\(F = 23.19, p < 0.001\)). No significant differences were observed with absolute alpha and beta, but alpha/theta ratios across all sites were significantly smaller in patients than controls (\(F = 8.19, p < 0.006\)).

Relative delta power did not differentiate the 2 groups, but group differences were observed with relative theta (\(F = 25.43, p < 0.007\)) and alpha (\(F = 7.81, p < 0.007\)), theta being increased and alpha being decreased compared with controls. Relative beta analysis exhibited significant group (\(F = 5.27, p < 0.03\)) and group \(\times\) site (\(F = 3.10, p < 0.008\)) effects, with patients showing reduced beta compared with controls at parietal (P3, Pz, P4), mid-temporal (T3, T4), posterior-temporal (T5, T6) and at F4, C4 and F8 sites (\(p < 0.05\)).

Follow-up of significant group (\(F = 6.95, p < 0.02\)) and group \(\times\) site (\(F = 3.69, p < 0.002\)) effects showed mean total frequency (Fig. 3) of patients to be lower than that of controls at parietal (P3, Pz, P4), mid-temporal (T3, T4), posterior-temporal (T5, T6), occipital (O1, Oz, O2) and C4 and F8 sites (\(p < 0.05\)). Similar group (\(F = 6.21, p < 0.02\)) and group \(\times\) site (\(F = 3.13, p < 0.02\)) effects were observed, with mean alpha frequencies (Fig. 3), with patients showing a slower frequency than controls at parietal (P3, Pz, P4), mid-temporal (T3, T4) posterior-temporal (T5, T6), frontal (F7, F8) and C4 sites (\(p < 0.05\)).

z-Score EEG

Abnormalities

Group-averaged topographic map displays of z scores for absolute and relative power values are shown in Fig. 4 and Fig. 5, respectively. Determination of the presence of a statistically significant group z score was carried out by the procedure employed by Prichep and John,\textsuperscript{29} where estimation of the probability level of a group-averaged z score of size \(n\) is derived by multiplying the mean z value by the square root of \(n\). Thus, in Fig. 4 and Fig. 5, mean z scores of ±0.44 and ±0.47 correspond to a \(p < 0.01\) for patients and controls, respectively.

With respect to the absolute power z scores, both the control group and, to a lesser extent, the patient group exhibited significantly reduced delta power when compared with population norms. This reduction was evident at all sites except in the patients where delta reductions did not reach significance at several temporal (T3, T4, T5, T6) sites. Compared with the control group, which showed significantly reduced theta at all sites when compared with population norms, patients showed significantly greater theta at all but 1 (Oz) occipital site. Absolute theta z scores for patients were larger at the left mid-temporal (T3) site than at the right mid-temporal site (T4). Generally, z scores showed both patients and controls to have less alpha power than population norms, reaching significance at all but the T4 and T6 sites in controls and at left posterior-central scalp sites in patients. Absolute z-score power values also showed reduced beta in both groups compared with population norms, with beta reductions reaching significance at all but T3 and F7 sites in patients and T4 and F8 sites in controls.

With respect to relative power z scores, both patients...
and controls tended to show reduced delta power compared with population norms, reaching significance in both groups at anterior-central sites. Relative theta was greater only for the patient group compared with the population norms (the exception being elevated theta in controls at Fpz), reaching significance at all sites and showing temporal asymmetry, with theta being greater in the left (T3) than in the right (T4) mid-temporal site. Compared with population norms, relative alpha power z scores were significantly reduced only in the

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**Fig. 1:** Group-averaged delta ($\delta$), theta ($\theta$), alpha ($\alpha$) and beta ($\beta$) topographic colour-coded maps of absolute power ($\mu$V$^2$) for patients with dementia of the Alzheimer type (DAT) and elderly controls.

**Fig. 2:** Group-averaged delta ($\delta$), theta ($\theta$), alpha ($\alpha$) and beta ($\beta$) topographic colour-coded maps of relative power (%) for patients with DAT and elderly controls.
patient group at all but Fp1 and Fpz sites. Only the patient group exhibited major reductions in relative beta power $z$ scores compared with norms, with reduced power being evident at all but Oz and several right-hemisphere (F8, T4, O2) sites.

Mean total band frequency $z$ scores for patients, but not for controls, were significantly reduced at all but Fp1, Fp2, Fz, F7, F8 and T4 sites compared with population norms. Similar effects were seen with mean alpha frequency for patients, but not controls, with significant $z$-score reductions at all but the T4 site. Also, the mean alpha frequency $z$ score at the left mid-temporal (T3) site (sample mean $-1.0$) was markedly lower than at the right mid-temporal (T4) site (sample mean $-0.4$).

**ANOVAs**

ANOVA comparisons of patient and control $z$ scores revealed significant differences for delta and theta absolute power, but not absolute alpha or beta. Patients exhibited greater negative delta $z$ scores ($F = 6.63, p < 0.02$) and greater positive theta $z$ scores ($F = 23.78, p < 0.001$) across all sites. Significant group differences were also found for alpha/theta $z$-score ratios, with patients exhibiting greater negative values than controls ($F = 8.04, p < 0.006$).

ANOVA comparisons of relative power showed group differences with theta, alpha and beta $z$-score values, with patients showing greater positive theta ($F = 27.24, p < 0.001$) and greater negative alpha ($F = 8.44, p < 0.006$) and beta ($F = 6.80, p < 0.02$) $z$ scores than controls. Follow-up of a significant group $\times$ site interaction ($F = 3.20, p < 0.008$) showed beta $z$ scores to be significantly more negative ($p < 0.05$) at all but occipital and Cz sites.

Mean total ($F = 9.20, p < 0.004$) and alpha band ($F = 5.79, p < 0.004$) frequency $z$ scores were significantly more negative for patients than controls at all sites (Fig. 3).

**EEG–MMSE correlations**

Table 1 shows the results of the correlational analysis relating the EEG band indices that significantly differentiated patients and controls with MMSE scores of patients with DAT. All of the relevant band indicies were significantly correlated with patient MMSE scores; delta and theta were negatively related to mental status scores, and alpha, beta and total frequency were positively related to MMSE scores. The raw, but not the $z$ score, absolute alpha/theta ratio also correlated positively with MMSE scores.

**Discriminant analysis**

Because too many predictors in a multisite EEG database can yield 100% correct classification by chance, only the band indices that (by ANOVAs) differentiated patients and controls were used. For the discriminant analysis of raw scores, the indices selected included absolute delta, theta, alpha/theta ratios and relative delta, theta, alpha and beta, as well as total and alpha mean frequency. For the discriminant analysis of $z$ scores, the indices selected were absolute delta and...
theta, relative theta, alpha and beta, as well as total and mean alpha frequency. To further reduce the number of predictors, values within each of these bands were averaged across the electrode sites that significantly differentiated patients and controls. Separate stepwise discriminant analyses were carried out for the raw and z-score EEG measures.

Table 2 displays the results of the 2 separate discriminant function analyses and jackknife replication classifications. The overall classification rate using raw and z-score EEG values was 75.4%. Similar rates were found with subsequent jackknife classification procedures. Absolute and relative theta, as well as mean total frequency contributed to the raw EEG classification (achieved in 3 steps), whereas relative theta alone contributed to the z-score EEG classification (achieved in 1 step).

Discussion

Most previously conducted quantitative EEG studies employing power spectrum analysis in DAT have described a pathologically slowed spectrum which, in comparison with the spectrum of a normal-aged control group, has been characterized by a shift in power from high to low frequencies. The methodological novelty of this study centred on the comparison of spectrally derived data of patients with DAT with 2 control databases. Although both comparative procedures generally confirmed a profile of neuroelectric slowing in DAT, they generated somewhat qualitatively distinct appearances of this profile in terms of: the direction of electrical frequency changes, the specific brain regions separating patient and controls, the correlations between EEG band indices and clinical ratings and the ability of each to classify individual spectrums as patient or control profiles.

In contrast to the patient versus control group comparisons, which revealed increased absolute but not relative delta power in DAT, patient EEGs compared with age-regressed EEG norms via z scores evidenced reduced absolute delta power at all but mid- and
posterior-temporal regions, as well as reduced relative delta power at anterior scalp regions. Similarly, $z$ scores of elderly controls reflected delta reductions which were of significantly greater negative magnitude than those seen in patients. Although the lowered delta in patients is somewhat surprising, and may reflect differences in “vigilance control” procedures used in this study and in neurometrics, it is not unexpected in healthy aged controls; reduced delta in elderly adults in good health has been reported previously, and it is generally accepted that normal aging is associated with desynchronization of the EEG (i.e., decreased slowing and increased fast activity). Given that negative delta power $z$ scores acquired under vigilance-controlled conditions may characterize the normal aging process, the progressive decline of these negative $z$-score DAT values toward zero or positive may be a valid indicator of disease progression for those laboratories employing a neurometric comparison under vigilance-controlled conditions, as delta power increments in DAT are typically observed only with advanced stages of the disease.

Of the EEG parameters differentiating the patient and elderly control groups, $z$-scored relative beta and total and alpha mean frequency showed widespread changes in DAT across all recording regions, whereas the same raw-scored parameters exhibited regional effects, separating the groups on the basis of significant differences primarily in non-anterior sites (i.e., parietal, temporal and occipital areas). Widespread, diffuse cortical spreading of EEG indices differentiating patients and controls may well reflect a previously reported “anteriorization” process paralleling neuronal and synaptic loss in temporoparietal regions in early stages and in frontal regions at later stages. Differences in topography among the methods may also reflect the differential sensitivity of the parameters to different pathological processes, or to different manifestations of the same process in different cortical areas. That each band index may be tapping a distinct behavioural function is supported by the finding that EEG alpha activity has been associated with attentional processes, whereas beta activity has been indicated to be a useful measure of appropriate cognitive and emotional processes.

Also of particular topographic interest with age-regressed $z$ scores is the hemispheric asymmetry observed with abnormal group values in DAT. The

![Fig. 5: Group topographic maps for relative delta ($\delta$), theta ($\theta$), alpha ($\alpha$) and beta ($\beta$) $z$-score power in patients with DAT and elderly controls. (See Fig. 4 caption for an explanation of the map values.) Significant $z$ scores ($p < 0.01$) are shaded in gray.](image)
patient group exhibited significant abnormal slowing of mean alpha and total band frequency, as well as reduced relative beta at the left mid-temporal (T3) site, but not at the right mid-temporal (T4) site. Left temporal slowing has been reported in a number of studies and, together with these observations, they overlap with positron emission tomography (PET) findings of left temporal and parietal hypometabolic activity and parallel findings of better correlations of glucose metabolism and MMSE scores with left than with right parietal values. As these neuroelectric asymmetries were evident only with age-regressed z scores, this population norm comparative procedure, apparently more sensitive to hemispheric differences, may be a better choice when attempting to relate specific hemispherically sensitive cognitive deficits with particular brain regions.

EEG–MMSE correlations were similar when using raw and age-regressed z scores and, in general, support the contention, as observed in other studies, that indices of EEG slowing (i.e., increased slow wave power, decreased fast wave power and slowed frequency) correlate positively with ratings of cognitive decline and, as such, are useful physiologic markers of disease progression. Slowing was also reflected in spectral ratios, with patients exhibiting reduced raw and z-score absolute alpha/theta power ratios, but only raw-score ratios were correlated, positively, with clinical ratings. Raw spectral ratios may then be proposed as an alternative to the z-score ratio measure in the study of dementia because, in addition to correcting for baseline power variation, spectral ratios capture both the increases in low-frequency and decreases in high-frequency power seen in DAT and have, in some studies, offered advantages over the standard power parameters in detecting and discriminating DAT and other pathological, aged patient groups.

Table 1: Pearson correlations between EEG indices (that significantly differentiated DAT from controls) and DAT scores on the Mini-Mental State Examination

<table>
<thead>
<tr>
<th>Spectral feature, band</th>
<th>Raw EEG measure, r</th>
<th>z-score EEG measure, r</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Absolute power</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Delta</td>
<td>-0.42</td>
<td>-0.42</td>
</tr>
<tr>
<td>Theta</td>
<td>-0.61</td>
<td>-0.41</td>
</tr>
<tr>
<td>Alpha/theta</td>
<td>0.49</td>
<td>NS</td>
</tr>
<tr>
<td><strong>Relative power</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Theta</td>
<td>-0.61</td>
<td>-0.58</td>
</tr>
<tr>
<td>Alpha</td>
<td>0.50</td>
<td>0.51</td>
</tr>
<tr>
<td>Beta</td>
<td>0.48</td>
<td>0.48</td>
</tr>
<tr>
<td><strong>Mean frequency</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>0.60</td>
<td>0.59</td>
</tr>
</tbody>
</table>

Note: DAT = dementia of the Alzheimer type; NS = not significant. n = 35, r = 0.33, p < 0.05 (2-tailed).

Table 2: Classification of patients with DAT and controls on the basis of discriminant analyses of raw and z-score EEG indices

<table>
<thead>
<tr>
<th>Data analyzed, actual group</th>
<th>Classification as</th>
<th>% correct</th>
<th>Jackknife classification as</th>
<th>% correct</th>
</tr>
</thead>
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<tr>
<td></td>
<td>Patient</td>
<td>Control</td>
<td>Patient</td>
<td>Control</td>
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<tr>
<td><strong>Raw EEG</strong></td>
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<td></td>
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<td></td>
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<tr>
<td>Patients</td>
<td>35</td>
<td>25</td>
<td>10</td>
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<tr>
<td>Controls</td>
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<td>6</td>
<td>24</td>
<td>80.0</td>
</tr>
<tr>
<td>Mean %</td>
<td></td>
<td></td>
<td></td>
<td>75.4</td>
</tr>
<tr>
<td><strong>z-score EEG</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Patients</td>
<td>35</td>
<td>24</td>
<td>11</td>
<td>68.6</td>
</tr>
<tr>
<td>Controls</td>
<td>30</td>
<td>5</td>
<td>25</td>
<td>83.3</td>
</tr>
<tr>
<td>Mean %</td>
<td></td>
<td></td>
<td></td>
<td>75.4</td>
</tr>
</tbody>
</table>
until these current classification figures are improved, reliably reproduced and validated prospectively, both methods underscore the use of quantitative EEG as a research tool, but not as a “diagnostic tool” or “diagnostic marker” for DAT.

References


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**Jock Cleghorn Prize**

This prize, which will consist of a suitably engraved plaque and a cheque for $500, will be awarded by the CCNP for the best poster presentation by a research trainee (graduate student or clinical resident) at the Annual Meeting of the CCNP. Candidates wishing to have their poster presentation considered should send a covering letter and a copy of their submitted abstract to Dr. Andrew J. Greenshaw at the address below. Those already applying for travel bursaries will automatically be considered for the Jock Cleghorn Prize. All others can contact Dr. Greenshaw.

The poster presentations will be judged at the Annual Meeting by a committee consisting of at least 3 members of the Awards Committee (or substitute judges to be chosen by the Council from the CCNP membership if Awards Committee members are unable to attend the Annual Meeting). Topics on either basic or clinical aspects of neuropsychopharmacology will be considered. The poster should represent research in which the graduate student or resident is the primary investigator, and(s)he should be the first author of the submitted abstract. The winner of the award will be announced in the first Newsletter following the Annual Meeting.

Research trainees should send a copy of the abstract they are submitting to the 2001 meeting of the CCNP along with a covering letter stating that they wish to have their presentation considered for the prize to:

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*The deadline for submissions is March 31, 2001.*