ELECTROENCEPHALOGRAPHIC AND PSYCHOPHYSIOLOGICAL DIFFERENTIATION OF 
BEHAVIORAL MEDICINE PATIENTS: An Empirical Investigation

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INTRODUCTION

To our knowledge, diagnostic clinical electroencephalography (EEG) has not been used in the systematic study of patients referred for behavioral medicine treatment. For this reason, we decided to undertake a comprehensive empirical EEG study of a large number of patients evaluated for treatment at the Behavioral Medicine Clinic and Stress Disorders Research Laboratory. This report will present a series of conceptually related empirical findings which suggest that EEG may be an important investigative and clinical tool in this population. While major EEG abnormality was rare, minor paroxysmal EEG dysrhythmias were seen in abundance and they showed robust relationships to (1) type of clinical presentation, (2) psychophysiological response to laboratory induced stress and (3) amount and frequency of endorsed "physical" symptoms over and above the primary complaint.

METHOD

Patients evaluated and treated at the Behavioral Medicine Clinic and Stress Disorders Research Laboratory are predominately adults without psychiatric complaint who present with a wide range of stress related symptoms. All patients have received a medical screen appropriate to their presenting problem. All patients are required to undergo a series of formal evaluations which must include a laboratory based assessment of their psychophysiological responsivity to induced stress. This assessment is termed a "Psychophysiological Profile" and involves continuous monitoring of (1) Peripheral Skin Temperature, (2) Skin Conductance, (3) Frontalis EMG, (4) Heart Rate, and (5) Systolic and Diastolic Blood Pressure during pre-stress baseline, induced stress, and post-stress recovery periods. The pre-stress resting baseline phase is subdivided into an eye's open and an eye's closed relaxation period. Stress is cognitively induced with mental arithmetic tasks given under pressure. From continual digital readouts of
peripheral skin temperature, skin conductance, and heart rate an examiner
notes, for each period, high and low values as well as estimates of median
values. Systolic and diastolic blood pressure recordings are automated and
printed out at three minute intervals throughout the entire procedure and
EMG levels are recorded on a moving strip chart for later inspection. For
the purpose of this study, stress response scores on any given physiological
variable were defined as the change or difference between the eye's open
pre-stress baseline condition and the induced stress period. The
psychophysiological profile was obtained and scored without knowledge of
the EEG results. Due to technological difficulties, skin conductance
measures could not be used during a large portion of the study and this
variable will not be reported upon.

Clinical EEGs were secured on 62 Behavioral Medicine referrals aged 15
to 75 and they were interpreted without knowledge of the results of the
psychophysiological profile or the results of the symptom endorsement
measures used. All patients were known to be Behavioral Medicine referrals
at the time of EEG study and in some of the cases the nature of the
presenting problem was known as well. Those EEGs which failed to contain a
sleep tracing (N=10) were omitted from the study sample as were two EEGs
with questionable findings and one tracing which was uninterpretable due to
excessive medication (Diazepam) effect. The inter-interpretor reliability
and test-retest reliability of our EEG methods are adequate and have been
The incidence of clinical EEG findings is based on a sample of 49 patients
with complete wake and sleep tracings. Data analyses involving
psychophysiological measures were stopped prior to entry of the most recent
nine patients and includes a total pool of 41 cases. Due to sporadic
measurement errors, sample sizes for the different EEG-psychophysiological
analyses range from 36 to 40.
CLINICAL EEG FINDINGS

Table 1A summarizes the EEG findings among 49 nonpsychiatric behavioral medicine patients aged 15 to 75. Despite previous empirical reports suggesting the screening value of EEG in the detection of covert physical disease in psychiatric patients, EEG abnormality of neurological or medical importance was rare (2%) among our patients. Surprisingly, we did find an exceptionally high (42.9%) incidence of various minor paroxysmal EEG dysrhythmias\(^1\) which published lines of evidence, including some depth electrode studies, suggest may be subcortical in origin.

In Table 1B behavioral medicine patients are contrasted with a previously published large control population in terms of the relative incidence of minor paroxysmal EEG patterns of the exact same type as found in our patients. We wished to ensure that the comparison involved equivalent age ranges for both groups. Since 93% of our patients are age 20 and above, we contrasted the number of paroxysmal EEGs for patients and literature controls within the 20 to 60 year age range. As is shown in Table 1B, behavioral medicine patients have a significantly elevated incidence of these EEG dysrhythmias as contrasted with controls. Both the literature controls and the patient population involve identical EEG recording methodology and interpretive criteria.

Furthermore, the patient population was dichotomized post-hock on clinical grounds into a Group "A" consisting of patients with evidence of a strong psychophysiological presentation and a Group "B" in which a psychophysiological component was not felt to be present. Group "A" consisted primarily of vascular and tension headache cases with a small number of other presenting problems including labile hypertension,

\(^1\)Minor paroxysmal EEG dysrhythmias include (1) asymptomatic focal spiking, (2) Small Sharp Spikes, (3) Diffuse Paroxysmal Slowing, (4) Paroxysmal Runs of Slow, (5) Six per second Spike & Wave, (6) Fourteen and six per second Positive Spikes, and (7) B-Mitten Patterns.
TABLE 1A. EEG FINDINGS AMONG NONPSYCHIATRIC BEHAVIORAL MEDICINE REFERRALS AGE 15 TO 75 WITH ADEQUATE EEGs (N=49).

<table>
<thead>
<tr>
<th></th>
<th>N</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>NORMAL EEG</td>
<td>27</td>
<td>55.1</td>
</tr>
<tr>
<td>FOCAL SLOWING</td>
<td>1</td>
<td>2.0</td>
</tr>
<tr>
<td>MINOR PAROXYSMAL DYSRHYTHMIA</td>
<td>21</td>
<td>42.9</td>
</tr>
</tbody>
</table>

TABLE 1B. PAROXYSMAL EEGs IN BEHAVIORAL MEDICINE PATIENTS AGE 20 TO 60 (N=43) VERSUS LITERATURE CONTROLS* AGE 20 TO 60 (N=556).

<table>
<thead>
<tr>
<th></th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>BEHAVIORAL MEDICINE</td>
<td>41.8</td>
</tr>
<tr>
<td>CONTROLS</td>
<td>10.9</td>
</tr>
</tbody>
</table>

\[\chi^2=33.262, df=1, p<.00001\]

TABLE 1C. PAROXYSMAL EEGs IN BEHAVIORAL MEDICINE PATIENTS AS A FUNCTION OF TYPE OF CLINICAL PRESENTATION (See Text).

<table>
<thead>
<tr>
<th></th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>STRONG PSYCHOPHYSIOLOGICAL COMPONENT</td>
<td>73.7</td>
</tr>
<tr>
<td>WEAK OR ABSENT PSYCHOPHYSIOLOGICAL COMPONENT</td>
<td>25.9</td>
</tr>
</tbody>
</table>

paroxysmal abdominal pain, and tachycardia. Group "B" contained patients with chronic pain related to post injury tissue damage as well as cases of phantom limb pain, phobia, weight control, smoking cessation, dysphagia, blepharospasm, dyskinesias, insomnia, and sexual dysfunction. As can be seen in Table 1C, fully 73.7% of those patients with a presumed psychophysiological component had paroxysmal EEG dysrhythmia as contrasted with only a 25.9% incidence among patients felt not to have a strong psychophysiological presentation.

PERIPHERAL SKIN TEMPERATURE

For the 40 behavioral medicine patients with complete data, peripheral skin temperature response to induced stress ranged from -3.5°F to +10°F as measured from pre-stress baseline levels. In Figure 1 the distribution of stress induced skin temperature change for paroxysmal EEG and normal EEG patients is shown. Those with normal EEGs show little change with the majority showing values during stress that are within ±1°F of their baseline pre-stress levels. In contrast, stress induced skin temperature change among paroxysmal EEG patients shows a markedly wide variability containing some of the most extreme change scores encountered. Originally we defined any temperature change from baseline which was greater than ±1°F as a positive response while all changes that fell within ±1°F of baseline were considered as insignificant fluctuation. Of patients with paroxysmal EEGs 82.4% have stress induced peripheral skin temperature change greater than 1°F whereas only 30.4% of normal EEG patients do so. In Table 2A the association between paroxysmal EEG and increased skin temperature change is shown to be statistically significant.

Since we realized that a ±1°F change cut off point could be challenged as arbitrary, we reanalyzed the data by dividing the total distribution of absolute skin temperature change at the median. The analysis shown in
## TABLE 2A. PERIPHERAL SKIN TEMPERATURE CHANGE (Baseline To Stress) AS A FUNCTION OF EEG CATEGORY (N=40).

<table>
<thead>
<tr>
<th></th>
<th>&gt;1°F CHANGE</th>
<th>&lt;1°F CHANGE</th>
<th>CHANGE &gt;MEDIAN</th>
<th>CHANGE &lt;MEDIAN</th>
</tr>
</thead>
<tbody>
<tr>
<td>PAROXYSMAL EEG</td>
<td>14</td>
<td>3</td>
<td>13</td>
<td>4</td>
</tr>
<tr>
<td>NORMAL EEG</td>
<td>7</td>
<td>16</td>
<td>7</td>
<td>16</td>
</tr>
</tbody>
</table>

\[ X^2 = 10.56, \ df = 1, \ p < .005 \]

\[ X^2 = 8.29, \ df = 1, \ p < .005 \]

## TABLE 2B. BASELINE PERIPHERAL SKIN TEMPERATURE (Pre-Stress) AS A FUNCTION OF EEG CATEGORY.

<table>
<thead>
<tr>
<th></th>
<th>LOWER THIRD (&lt;81°F)</th>
<th>MIDDLE THIRD (81.3°F-88.2°F)</th>
<th>UPPER THIRD (89°F-95.2°F)</th>
</tr>
</thead>
<tbody>
<tr>
<td>PAROXYSMAL EEG</td>
<td>7</td>
<td>8</td>
<td>1</td>
</tr>
<tr>
<td>NORMAL EEG</td>
<td>6</td>
<td>5</td>
<td>12</td>
</tr>
</tbody>
</table>

\[ X^2 = 9.1, \ df = 2, \ p < .02 \]
Table 2A indicates that paroxysmal EEGs are significantly associated with temperature increases above the median. Figure 2 shows the distribution of absolute skin temperature change for paroxysmal and normal EEG groups. The mean stress induced absolute peripheral skin temperature change is significantly higher for patients with positive EEG findings. Furthermore, the point-biserial correlation between EEG and absolute temperature change is robust (r = .52, p < .001). It is not likely that the EEG-peripheral skin temperature change relationships are epiphenomena of personality style variables or "negative affectivity". A Pearson correlation between raw scores on the Eysenck personality inventory neuroticism scale and absolute stress induced temperature change is only slight (r = .04, p = ns).

Typically, stress should produce a peripheral vasoconstriction and a decrease in peripheral skin temperature. Peripheral vasodilation and skin temperature rise are viewed as "paradoxical" responses to a stress event. We find that 68.8% of our paroxysmal EEG patients but only 21.7% of our normal EEG patients respond to stress with temperature elevations of greater than 1°F.

For reasons which are obscure, patients with paroxysmal EEGs also seem to have resting baseline skin temperatures which are significantly lower than those values found with normal EEG subjects. In Table 2B the total distribution of pre-stress baseline peripheral skin temperatures is divided into thirds. As can be seen, there is a statistically significant tendency for lower baseline temperatures to be found among patients with paroxysmal EEGs.

FRONTALIS EMG

In our population (N=38) Frontalis EMG response to cognitive stress ranged from a 17.2 microvolt increase over baseline levels to "paradoxical"
EMG decreases as large as 9.8 microvolts. The distribution of EMG change from baseline to stress for both paroxysmal EEG and normal EEG subjects is shown in Figure 3. The distribution for normal EEG patients is clearly skewed in the direction of increased EMG levels with stress whereas the distribution for paroxysmal EEG patients is skewed toward decreased microvoltage with stress. The difference between the distributions is statistically significant. In Table 3A the two EEG groups are contrasted in terms of the presence or absence of a "paradoxical" EMG decrease with stress. Fully 70.6% of paroxysmal EEG patients but only 33.3% of the normal EEG patients reduced EMG levels during the stress period. The association between atypical paradoxical drops in EMG level under stress and paroxysmal EEG dysrhythmia is statistically significant.

Table 3B considers three levels of EMG response—(1) a middle range of only slight change from baseline, (2) a decrease of greater than two microvolts and (3) an increase of greater than two microvolts. Again, paroxysmal EEG patients seem to display significantly decreased EMG tension at the time of experimentally induced stress. However, the point-biserial correlation between EEG group and EMG change is only marginal ($r=.27, p<.10$). As was found with peripheral skin temperature change with stress, there is no correlation between raw scores on the Eysenck personality inventory neuroticism scale and stress induced EMG change ($r=.08, p=ns$).

**HEART RATE**

The heart rate response to stress is complex. When the distribution of absolute heart rate change produced by stress was dichotomized by a median split, a relationship between EEG cateogry and dichotomized heart rate change was not found ($X^2=0.5079, df=1, p=ns$). However, when heart rate change from baseline to stress period was grouped in 5 beat/minute intervals (i.e., 0-4, 5-9, and 10 and above) there was a robust EEG effect
FIGURE 3

PAROXYSMAL DYSRHYTHMIA
N=17

NON-PAROXYSMAL EEG
N=21
t=2.16, df=36, p < .05

PERCENT OF SUBJECTS

FRONTALIS EMG CHANGE (microvolts) BASELINE TO STRESS

-10uV to -8uV  -6uV to -4uV  -2uV to 0.0uV  +2uV to +4uV  +6uV
-8.1uV to -6.1uV -4.1uV to -2.1uV -0.1uV to +1.9uV +3.9uV to +5.9uV +7.9uV
### TABLE 3A. FRONTALIS EMG CHANGE (Baseline To Stress) AS A FUNCTION OF EEG CATEGORY (N=38).

<table>
<thead>
<tr>
<th>Paroxysmal EEG</th>
<th>Paradoxical EMG Decrease</th>
<th>No Change OR EMG Increase</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>12</td>
<td>5</td>
</tr>
<tr>
<td>Normal EEG</td>
<td>7</td>
<td>14</td>
</tr>
</tbody>
</table>

\[ X^2 = 5.22, \text{df}=1, \ p<.025 \]

### TABLE 3B. THREE LEVELS OF FRONTALIS EMG CHANGE (Baseline To Stress) AS A FUNCTION OF EEG CATEGORY.

<table>
<thead>
<tr>
<th></th>
<th>&gt;2uV Increase</th>
<th>0±2uV</th>
<th>&gt;2uV Decrease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Paroxysmal EEG</td>
<td>2</td>
<td>4</td>
<td>11</td>
</tr>
<tr>
<td>Normal EEG</td>
<td>7</td>
<td>9</td>
<td>5</td>
</tr>
</tbody>
</table>

\[ X^2 = 6.61, \text{df}=1, \ p<.05 \]
which is shown in Table 4A and in Figure 4. Only one normal EEG patient was at the upper level of 10 or more beats/minute increase whereas nearly half (46.7%) of the paroxysmal EEG patients displayed such large heart rate increases. The association between paroxysmal EEG and increased heart rate response to stress is significant. Inspection of Figure 4 suggests that paroxysmal EEG patients are split between being either clearly high heart rate responders or clear non-responders with only 6.7% falling into the middle range. In contrast, normal EEG patients do not show such a clear U-shaped distribution of stress induced heart rate response but instead show a linear distribution which is markedly skewed toward the direction of minimal heart rate response. Statistically, the mean heart rate increase from baseline to stress was significantly higher for the paroxysmal EEG group as contrasted with the normal EEG patient group. In addition, a point-biserial correlation between EEG groups and absolute heart rate increase with stress was substantial (r = .45, p < .01). As expected, raw scores on the Eysenck Personality Inventory Neuroticism scale correlated poorly with stress induced heart rate change (r = .18, p = ns).

Since the above 2x3 analysis (Table 4A) was derived from an inspection of graphed data (Figure 4), it may have been opportunistic. Thus an additional 2x3 contingency was formed with heart rate stress response data now trichotomized into lower quartile, middle two quartiles, and upper quartile. This is shown in Table 4B and a significant association between increased heart rate stress response and paroxysmal EEG continues to be seen.

SYSTOLIC BLOOD PRESSURE

When the distribution of systolic blood pressure pre-stress baseline to stress period absolute change scores was dichotomized by a median
FIGURE 4

**PAROXYSMAL DYSRHYTHMIA**
N=15

**NON-PAROXYSMAL EEG**
N=21

t=2.09, df=34, p < .05

PERCENT OF SUBJECTS

HEART RATE CHANGE (beats per minute) BASELINE TO STRESS

0-4 BPM

5-9 BPM

10+ BPM

FIGURE 5

**PAROXYSMAL DYSRHYTHMIA**
N=16

**NON-PAROXYSMAL EEG**
N=22

PERCENT OF SUBJECTS

SYSTOLIC BLOOD PRESSURE ABSOLUTE CHANGE BASELINE TO STRESS

0-4 mm Hg

5-9 mm Hg

10+ mm Hg
TABLE 4A. HEART RATE ABSOLUTE CHANGE (Baseline To Stress) AS A FUNCTION OF EEG CATEGORY (N=36).

<table>
<thead>
<tr>
<th>HEART RATE CHANGE IN BEATS/MINUTE</th>
<th>0-4 BPM</th>
<th>5-9 BPM</th>
<th>10+ BPM</th>
</tr>
</thead>
<tbody>
<tr>
<td>PAROXYSMAL EEG</td>
<td>7</td>
<td>1</td>
<td>7</td>
</tr>
<tr>
<td>NORMAL EEG</td>
<td>13</td>
<td>7</td>
<td>1</td>
</tr>
</tbody>
</table>

\[ X^2 = 10.08, \text{df}=2, \ p<.01 \]

TABLE 4B. HEART RATE CHANGE IN QUARTILES (Baseline To Stress) AS A FUNCTION OF EEG CATEGORY.

<table>
<thead>
<tr>
<th>LOWER QUARTILE</th>
<th>MID-RANGE 26-74%</th>
<th>UPPER QUARTILE</th>
</tr>
</thead>
<tbody>
<tr>
<td>PAROXYSMAL EEG</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>NORMAL EEG</td>
<td>5</td>
<td>14</td>
</tr>
</tbody>
</table>

\[ X^2 = 7.66, \text{df}=2, \ p<.05 \]

TABLE 5. SYSTOLIC BLOOD PRESSURE CHANGE (Absolute Change Baseline To Stress) AS A FUNCTION OF EEG CATEGORY

<table>
<thead>
<tr>
<th>LOWER QUARTILE</th>
<th>MID-RANGE 26-74%</th>
<th>UPPER QUARTILE</th>
</tr>
</thead>
<tbody>
<tr>
<td>PAROXYSMAL EEG</td>
<td>0</td>
<td>13</td>
</tr>
<tr>
<td>NORMAL EEG</td>
<td>9</td>
<td>6</td>
</tr>
</tbody>
</table>

\[ X^2 = 12.55, \text{df}=2, \ p<.005 \]
split, a statistically significant relationship between EEG and blood pressure change could not be demonstrated. However, as was done previously with the heart rate data, the systolic blood pressure changes were graphed using 5mmHg intervals and the results are shown in Figure 5. This time patients with normal EEGs are seen to have a U-shaped distribution with the group sharply divided between non-responders and highly responsive patients and with only a minority (9.1%) in the middle range. Conversely, paroxysmal EEG patients are seen to have stress induced blood pressure change clearly skewed toward high responsivity. Not shown, a 2x3 contingency analysis of the Figure 5 data showed a significant positive relationship between paroxysmal EEG and increased systolic blood pressure response to laboratory induced stress ( $X^2=7.51$, df=2, p<.025 ). However, wishing to avoid opportunistic analyses derived from graphed data using arbitrary intervals, we statistically trichotomized the distribution of blood pressure change scores into lower quartile, middle two quartiles, and upper quartile sections. The results of contrasting this trichotomy against EEG category is given in Table 5. As can be seen, paroxysmal EEG is significantly related to increased blood pressure response to stress. Unlike the variables reported on above, a significant point-biserial correlation between EEG category and systolic blood pressure was not found ( r=.13, p=ns ). Systolic blood pressure is the only variable for which pre-stress to stress change scores correlated with the Neuroticism scale of the Eysenck Personality Inventory ( r=.32, p<.05 ) which suggests that at least some contribution from personality variables may be occurring.

Diastolic blood pressure responses to stress were not significantly related to EEG findings.
CONSISTENCY OF RESPONSE ACROSS SYSTEMS

The previous analyses have shown that minor subcortical paroxysmal EEG dysrhythmia is strongly related to psychophysiological response to laboratory induced stress. However, do paroxysmal EEG patients tend to respond consistently across all four of the physiological measures studied (Peripheral Skin Temperature, Frontalis EMG, Heart Rate, Systolic Blood Pressure) or do different patients tend to be responsive on different measures? In order to answer this question, we selected all patients who had complete data on all four of the physiological measures (N=34). For both EEG groups, patients were ranked according to whether or not they had positive responses on 4, 3, 2, 1, or none of the physiological measures studied. The results of this procedure are shown in Figure 6 where it can be seen that two thirds (66.7%) of the paroxysmal EEG patients, but only 10.5% of the normal EEG patients are responsive on 3 or 4 of the 4 measures employed. In Table 6 a statistical analysis of these data is given and it indicates a significant tendency for patients who have paroxysmal EEG patterns to be responsive across multiple physiological systems when exposed to induced stress in a laboratory situation.

PHYSICAL SYMPTOM ENDORSEMENT

In a separate study conducted by one of the authors (E.O.M.), 14 patients with paroxysmal EEGs and 21 normal EEG patients completed the Pennebaker Inventory Of Limbic Languidness ("PILL")\(^1\) - a self report scale consisting of 54 common physical symptoms which are ranked on a 5-point scale ranging from "never or almost never" to "more than once every week". The PILL was developed as an alternative to the better known

\(^1\)Pennebaker, J.W. The Psychology Of Physical Symptoms, New York Springer-Verlag, 1982
FIGURE 6

<table>
<thead>
<tr>
<th>Number of Four Psychophysiological Measures with &quot;Reactive&quot; Response</th>
<th>Paroxysmal EEG (N=15)</th>
<th>Non-Paroxysmal EEG (N=19)</th>
</tr>
</thead>
<tbody>
<tr>
<td>100% (4/4)</td>
<td>Dark gray</td>
<td>Light gray</td>
</tr>
<tr>
<td>75% (3/4)</td>
<td>Dark gray</td>
<td>Light gray</td>
</tr>
<tr>
<td>50% (2/4)</td>
<td>Dark gray</td>
<td>Light gray</td>
</tr>
<tr>
<td>25% (1/4)</td>
<td>Dark gray</td>
<td>Light gray</td>
</tr>
<tr>
<td>0% (0/4)</td>
<td>Dark gray</td>
<td>Light gray</td>
</tr>
</tbody>
</table>
TABLE 6

TABLE 6. NUMBER OF PSYCHOPHYSIOLOGICAL VARIABLES (Temperature, EMG, Heart Rate, Blood Pressure) WITH POSITIVE RESPONSE AS A FUNCTION OF EEG CATEGORY.

<table>
<thead>
<tr>
<th></th>
<th>0-1</th>
<th>2</th>
<th>3-4</th>
</tr>
</thead>
<tbody>
<tr>
<td>PAROXYSMAL EEG</td>
<td>3</td>
<td>2</td>
<td>10</td>
</tr>
<tr>
<td>NORMAL EEG</td>
<td>12</td>
<td>5</td>
<td>2</td>
</tr>
</tbody>
</table>

\[ X^2 = 11.68, \text{ df}=2, p<.005 \]
inventories that are lengthy, use a limited time frame, and tend to focus on rare or pathological symptoms. The scale can be used in a clinical context to assess individual patterns or as a global measure of symptom reporting. It is stable over time with a test-retest reliability of $r=.79$. According to the author, the scale assesses a trait tendency to report physical symptoms.

In this sub-study, the EEG was secured and interpreted without knowledge of the patients score on the PILL.

The distribution of scores on this instrument for both paroxysmal EEG and normal EEG groups is shown in Figure 7. There are no significant age differences between EEG groups. The results indicated that the mean PILL score was significantly higher for the paroxysmal EEG group than for the normal EEG group. The point-biserial correlation between EEG category and PILL scores was substantial ($r=.46$, $p < .01$). Furthermore, when the total distribution of PILL scores was split at the median, 85.7% of the paroxysmal EEG patients had scores above the median and only 23.8% of the normal EEG group scored in that range (Fisher's Exact Test, $p < .002$).

The increased physical symptom reporting of paroxysmal EEG patients is conceptually consistent with their heightened autonomic lability and also replicates some early literature reports suggesting increased autonomic symptomatology in people with these EEG signals.

**DISCUSSION**

The analyses presented above support a number of empirical conclusions. The most general finding is that minor paroxysmal EEG dysrhythmias are seen significantly more frequently among adult behavioral medicine patients than among age equated normal controls. However of far greater importance is the observation that paroxysmal EEG dysrhythmia may be uniquely related to an
FIGURE 7

- PAROXYSMAL EEG
  - N=14
- NORMAL EEG
  - N=21
  - t=3.21, df=33, p < .01

PERCENT OF SUBJECTS

LOW SYMPTOM ENDORSEMENT

"PILL" SCORES AS A FUNCTION OF EEG CATEGORY

HIGH SYMPTOM ENDORSEMENT
important subset of behavioral medicine patients with common clinical and psychophysiological characteristics.

Under laboratory conditions, paroxysmal EEG dysrhythmia is strongly related to elevated and/or "paradoxical" physiological response to stress across a spectrum of physiological response systems. Furthermore, patients with strong psychophysiological clinical presentations (i.e. Vascular Migraine, Labile Hypertension etc.) have an exceptionally high incidence of these EEG dysrhythmias (73.7%) as contrasted with other behavioral medicine patients (i.e. Post Injury Chronic Pain etc.) felt to be without a significant psychophysiological component to their complaint (25.9%) or age equated normal controls (10.9%). In addition, patients with these minor paroxysmal EEG dysrhythmias endorse significantly more somatic symptoms over and above their presenting complaint than do normal EEG patients. Taken together, these findings suggest the hypothesis that such minor dysrhythmias may be associated with a heightened vulnerability to labile physiological disruption under stress and as such an increased vulnerability to certain stress related disorders. If supported by additional research, this would have important heuristic implications in that such EEG signals may emerge as viable CNS biological markers for identifying individuals with inherent autonomic lability. This could also have potential explanatory value in understanding that subpopulation of patients in general who repeatedly seek consultation for a variety of vague autonomic somatic complaints for which no firm medical diagnosis of illness can be established.

The EEG patterns in question are minor dysrhythmias and most of them are considered to be controversial. In many laboratories they are either interpreted as "normal variants" or not mentioned at all. Certainly they are not associated with major neurological disease or dysfunction. These EEG patterns are more frequently seen among psychiatric populations where
they have been weakly associated with emotional lability, impulsiveness, acting out and to some extent aggressive behavior. Studies of these EEG patterns among psychiatric patients have often produced conflicting empirical results. However the clinical correlates of these EEG events may be obscured by the complexity of the psychiatric symptom overlay. If the effects of these minor dysrhythmias are subtle, as they might be, then they may be masked and hence not be discernable against a background of major psychiatric disturbance. Our behavioral medicine patients are essentially non-psychiatric and perhaps this is why we are able to find such clear cut correlates between these EEG signals and psychophysiological and clinical variables. This would suggest that the meanings of these EEG dysrhythmias may best be researched within normal populations (which might be difficult due to their low incidence among normals) or in behavioral medicine populations with low psychiatric expressivity.

The types of minor EEG dysrhythmias encountered in this study are presumed to be subcortical in origin. With some of the discrete patterns there have been published depth electrode studies supporting this contention. This would be compatible with the lack of obvious cognitive deficit related to these EEG signals as well as their prior association with emotional variables and the current data linking them to autonomic lability.

Finally and importantly it should be stressed that the results of this study are incompatible with the notion that these EEG patterns are normal or normal variants and as such have no relationship to behavior or function. Were such EEG signals in fact normal and hence without consequence, then the results reported in this investigation would not have occurred.